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RESPONSUM

om

hvorvidt det vil være i strid med menneske- og miljøretlige regler at etablere 5G-systemet i Danmark

ENDELIG UDGAVE

Rachel Santini, leder af forskernetværket "Dansk Institut for Folkesundhed", Rådet for Helbredssikker Telekommunikation, EHS-foreningen og Oplysningsforbundet May Day har bedt mig udarbejde et responsum om ovennævnte problemstilling.

Responsummet baseres på retsreglerne i Den Europæiske Menneskerettighedskonvention, FN's børnekonvention, EU's habitatdirektiv, fuglebeskyttelsesdirektiv og forsigtighedsprincip, samt Bern- og Bonn-konventionerne om beskyttelse af dyr og planter.

Besvarelsen er opdelt i et pkt. 1, som vedrører faktum (om 5G-systemet samt forskning i skadevirkninger ved radiofrekvent elektromagnetisk stråling), og et pkt. 2, der sammenholder faktum med ovennævnte retsregler. Pkt. 3 indeholder en overordnet konklusion.

Indholdsfortegnelse

1. Faktum.

1.1. Hvad er 5G?

"5G" er en samlebetegnelse for den næste (femte) generation af kommunikationssystem til mobiltelefoner og internetbrug. Det har ikke nogen lovbaseret definition og er tilsyneladende hverken fuldstændigt fastlagt eller standardiseret.

Den tilgængelige information om det påtænkte system kommer primært til udtryk via de tiltænkte opgaver og formål, som 5G skal varetage, jf. f.eks. Europa-Kommissionens "Working document" af 14. september 2016, pkt. 3, og teleindustriens 5G-manifest[1] af 7. juli 2016.

5G-systemet er bl.a. beskrevet således i den videnskabelige litteratur, jf. Neufeld og Kuster (2018), p. 705:

"THE FIFTH generation of wireless communication technology (5G) promises to facilitate transmission at data rates up to a factor of 100 times higher than 4G. For that purpose, higher frequencies (including millimeter-wave bands), broadband modulation schemes, and thus faster signals with steeper rise and fall times will be employed, potentially in combination with pulsed operation for time domain multiple access. 5G is designed as a ubiquitous communication system spanning applications such as high-bandwidth mobile data and telephony, real-time machine-to-machine communication (e.g., autonomous mobility), and the Internet of Things (IoT)."

Det er bl.a. ikke endnu fastlagt, hvilke frekvensbånd 5G-systemet ville benytte, og det fremgår af følgende tabel, at de frekvenser, der bl.a. overvejes, er de samme, som hidtil har været udnyttet til tidligere generationer[2]:

Table 1: The Main Frequency Bands for 5G Standards Taken up Globally

Frequency Band	Frequency Range	Countries/Regions	Comments
Low Band	<1 GHz (UHF) usually 600/700 MHz	EU, USA, India	Current favourite as longer range, so less costly infrastructure and more familiar technology
Mid Band	3-5 GHz (above UHF)	EU, Korea, Rep., China, India with USA at 2 GHz; China and Japan in 2020	More spectrum available, with compromise on range and performance
High Band	20-100 GHz	EU, USA, Korea, Rep.; in 2020 - China, Japan, India	Short range (10-150m), high speed, low latency

Source: Bertenyi, 2017; authors.

Det hedder i et svar af 1. april 2019 fra Energi-, Forsynings- og Klimaministeren til folketingets udvalg om samme om systemets påtænkte implementering i Danmark bl.a.:

"De grænseværdier, der bliver anvendt i Danmark, har baggrund i anbefalinger fra EU, der er baseret på værdier fastlagt af den Internationale Kommission for Ikke-ioniserende Stråling

1 http://ec.europa.eu/newsroom/dae/document.cfm?action=display&doc_id=16579

2 Tabellen er anvendt i Europa-Parlamentets rapport af april 2019 om "5G Deployment – State of play in Europe, USA and Asia", s. 10.

(ICNIRP).

Teleselskaberne skal sikre overholdelse af grænseværdierne, som er 2 W/kg, hvor folk opholder sig og færdes. Disse grænseværdier er teknologineutrale og det betyder, at grænseværdierne gælder, uanset hvilken teknologi der er tale om fx 2G, 3G, 4G eller 5G.

Teleselskaberne har oplyst, at de forventer, at antallet af antennepositioner vil blive forøget med 15-25 % frem mod 2025 som følge af udrulningen af 5G. Det er teleselskabernes forventning, at den samlede elektromagnetiske eksponering (stråling) vil blive øget med 10-20 % i forhold til i dag.

Teleselskaberne forventer, at eksponeringen fra mobilnettene fortsat vil ligge langt under grænseværdierne, også når 5G-nettene er fuldt udbyggede.

5G-nettet vil være baseret på højere frekvenser end de øvrige teknologier, og mobilsignalerne vil derfor række kortere. Det betyder, at der vil være behov for at lave et mere fintmasket net med flere basestationer (small cells). Disse basestationer vil sende med en lavere effekt end fx de antenner, som sender på 2G, 3G og 4G. Strålingen vil derfor også være tilsvarende mindre."[3]

1.2. Forskningen.

I hvert fald siden 1966 har der været videnskabelig forskning, som dokumenterede helbredsmæssige skadevirkninger ved elektromagnetiske felter.[4]

Det er denne forskning, som skal sammenholdes med det påtænkte 5G-systems kendte karakteristika, jf. pkt. 1.1. ovenfor, og de "grænseværdier" (maksima for udledning af en bestemt form for miljøpåvirkning), der p.t. anvendes i EU og Danmark, jf. pkt. 2.1. nedenfor.

Det har ikke været praktisk muligt at gennemgå det fulde tilgængelige videnskabelige materiale, som underbygger de ovenfor beskrevne skadevirkninger på menneskers og dyrs helbred ved eksponering for radiofrekvent elektromagnetisk stråling, da dette materiale tæller flere tusinder artikler.

Det gennemgåede materiale er dels fremfundet af undertegnede selv, dels fremsendt af bestiller, herunder efter anmodning fra undertegnede.

1.2.1. Fokus i nærværende responsum.

Opmærksomheden er centreret omkring de resultater, der positivt dokumenterer enten egentlige skadevirkninger eller risici herfor på mennesker, dyr og planter.

I det omfang sådanne dokumenterbare forskningsresultater foreligger, er disse i sagens natur af langt større betydning end undersøgelser, der ikke har været i stand til at identificere en skadevirkning eller risiko herfor, al den stund sidstnævnte gruppering ikke i sig selv udelukker, at der rent faktisk er skadevirkninger eller risici.

Er det én gang lødigt videnskabeligt bevist, at der er enten skadevirkning eller risiko for skade, er det ikke relevant, om der ti andre gange er gennemført lødige forsøg, som ikke kunne påvise en sådan skadevirkning eller risiko. Det er blot op til videnskaben at afklare, hvorfor de ti øvrige lødige forsøg ikke påviste det, der nu foreligger videnskabeligt bevist, for på denne

3 Se i øvrigt pkt. 2.1 nedenfor om de i Danmark anvendte grænseværdier.

4 Jf. Pall 2018 p. 9, der henviser til Marha K. 1966, artiklen: *"Biological effects of high-frequency electromagnetic fields (translation)"*.

måde bedre at kunne forstå, hvorfor og hvordan skaderne opstår eller kan opstå.[5]

Dette kan muligvis illustreres med undersøgelse af bestanden af svaner: Konstateres det ved én undersøgelse, at der findes sorte svaner, er det ikke relevant, at der ved ti andre undersøgelser ikke er fundet nogen svaner, der var sorte. Det er nu engang påvist, at den sorte svane eksisterer, og det kan muligvis være nyttigt at afklare, hvorfor de ti andre undersøgelser ikke konstaterede det samme.[6]

Det ville være misvisende, om man forsøgte sig med en "statistisk gennemsnitsbetragtning", og på den måde f.eks. konkluderede, at der kun er 1/11 sandsynlighed for, at den sorte svane rent faktisk findes, fordi dette kun er bevist ved én undersøgelse, mens ti andre ikke fandt noget bevis herfor.

1.2.2. Mennesker: Helbredsmæssige skader og risici.

1.2.2.1. DNA-skader.

I 2015 foretoges en videnskabelig gennemgang af de dengang mere end 100 tilgængelige peer-reviewed studier, som vedrørte undersøgelsen af såkaldt "oxidative effekter" af lavintensitets-radiofrekvent stråling (herefter forkortet: RFR).

Undersøgelsen (Yakymenko et al 2015[7]) viste bl.a., at det var plausibelt, at EHS-lignende[8] tilstande i hvert fald til dels forårsages af eksponering over for lav-intensitets RFR (p. 195), og at eksponeringen kunne medføre kræft (p. 196), begge fremkaldt af "oxidativt stress". Det konstateredes således, at 93% af undersøgelser viste at strålingen medførte dannelsen af reaktive oxidative forbindelser og oxidativt stress i alle undersøgte levende organismer fra celler, planter, insekter, forsøgsdyr til mennesker (sædceller), jf. ibid. p. 186.

Yakymenko et al 2015, p. 186:

"All above mentioned studies dealt with the effects of low-intensity RFR. This means that the intensity of radiation was far below observable thermal effects in biological tissues, and far below safety limits of the International Commissions on Non-Ionizing Radiation Protection (ICNIRP) (ICNIRP, 1998)."[9]

Ibid., p. 187:

"Low-intensity RFR is referred to as radiation with intensities which do not induce significant thermal effects in biological tissues. Accordingly, any intensity of RFR under the ICNIRP limits can be referred to as low-intensity. In this paper we will analyze only the effects of low-intensity RFR."

Ibid., p. 196 (konklusion):

"...a broad biological potential of ROS and other free radicals, including both their mutagenic effects and their signaling regulatory potential, makes RFR a potentially hazardous factor for

5 Se i samme retning Philips et al (2009), *"Electromagnetic fields and DNA damage."*, offentliggjort i det videnskabelige tidsskrift "Pathophysiology" nr. 16 (2009), pp. 79–88, pp. 84 – 85. P. 85 anføres: *"Each study to investigate RFR-induced DNA damage must be evaluated on its own merits, and then studies that both show effects and do not show effects must be carefully evaluated to define the relationship of experimental variables to experimental outcomes and to assess the value of experimental methodologies to detect and measure these outcomes (see Section 2)."*

6 Eksemplet er bl.a. anvendt af videnskabsfilosoffen Karl Popper.

7 Offentliggjort i det videnskabelige tidsskrift "Electromagnetic Biology and Medicine", nr. 35 2016, pp. 186 – 202.

8 EHS står for Elektro Hyper Sensitivitet, og er en fysisk lidelse, hvorefter personen får en række symptomer af ophold i nærheden af udstyr, der afgiver elektromagnetisk stråling. Kaldes ofte "el-overfølsomhed".

9 Disse grænseværdier omtales nærmere i pkt. 2.1 nedenfor.

human health. We suggest minimizing the intensity and time of RFR exposures, and taking a precautionary approach towards wireless technologies in everyday human life."

Det amerikansk baserede, forskerdrevne "BioInitiative 2012 - A Rationale for Biologically-based Exposure Standard for Low-Intensity Electromagnetic Radiation" har d. 15. november 2017 offentliggjort en gennemgang af 200 da foreliggende undersøgelser af radiofrekvent elektromagnetisk strålings påvirkning af frie radikaler (*"free radicals"*), som fremkalder såkaldt "oxidativt stress", jf. omtalen af Yakymenko (2015) ovenfor.

Gennemgangen viste, at der i 180 af de 200 undersøgelser (90 %) var konstateret statistisk signifikante effekter, medens der i de sidste 20 (10 %) ikke var reporteret nogen statistisk signifikant effekt.

Martin Pall 2018[10] konstaterede maj 2018, at der på det tidspunkt eksisterede minimum 21 videnskabelige undersøgelser (siden 1971), som dokumenterede DNA-skader ved radiofrekvent elektromagnetisk stråling, og at disse førte til kromosomskader og andre mutationer.

Endvidere konstaterede han, at der var minimum 19 studier (siden 1981), som dokumenterede, at denne stråling fremkaldte frie radikaler og oxidativt stress[11].

REFLEX-studiet (2004) blev gennemført af 12 forskningsinstitutioner på vegne af EU med et budget på 3 mio. euro. Blandt resultaterne var, at der ved en stråleabsorptionsrate (herefter forkortet "SAR") på 1,3 W/kg (dvs. under de af ICNIRP anbefalede grænseværdier på 2,0 W/kg for krop og hoved, jf. pkt. 2.1. nedenfor) skete en betydelig forøgelse af DNA-skader (p. 109):

"RF-EMF exposure at a SAR of 1.0 W/kg and below had no effect on Comet formation in HL-60 cells (expressed as Olive Tail Moment OTM) as compared to control and sham-exposed cells. On the other hand RF-EMF at SAR of 1.3 W/kg and above caused a significant increase in DNA strand breaks. The maximum of this effect was observed at SAR 1.3 W/kg (OTM = 2.20 ± 0.16) and 1.6 W/kg (2.24 ± 0.10)."

Endvidere var denne strålingsstyrke den, som producerede den største effekt på DNA (p. 119, gentaget p. 222):

"...were applied following RF-field exposure of HL-60 cells at that exposure condition with the most significant effect on DNA integrity (1800 MHz, continuous wave, 1.3 W/kg, 24h)."

Ibid., p. 223, afsnit 5.2.1., laboratoriedeltager 2, konklusion 9:

"Within the investigated SAR energy ranges RF-EMF under the in-vitro conditions used are genotoxic in HL-60 cells without affecting cell-cycle distribution cell proliferation or cell progression."

Ibid., konklusion 10:

"The partial-body SAR for any 10-gram tissue like for example the head as exposed region to

10 PhD, prof.emeritus i biokemi og Basic Medical Sciences, Martin L. Pall - *"5G: Great risk for EU, U.S. and International Health! Compelling Evidence for Eight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures and the Mechanism that Causes Them"*, pp. 6 – 8.

11 Ibid., pp. 11 – 12.

mobile phone electromagnetic fields should not exceed 2 W/kg according to the Radio-Radiation Protection Guidelines. Notably, our findings on genotoxic effects of RF-fields in HL-60 cells have been shown for SAR levels below these acceptable partial-body SAR levels."

Ibid., p. 223, afsnit 5.2.2., laboratoriedeltager 3:

"Our results imply a genotoxic action of RF-EMFs below proposed radiation safety levels."

Det blev dog tillige samlet set konkluderet (p. 226), at eftersom der "alene" var tale om laboriatorieforsøg, var REFLEX-studiet ikke i sig selv nok til at konkludere, at de (fortsat) gældende grænseværdier medførte fare for menneskers helbred, men at forsøget gjorde en sådan konklusion mere nærliggende. Endvidere konkluderedes, at:

"Furthermore, there exists no justification anymore to claim, that we are not aware of any pathophysiological mechanisms which could be the basis for the development of functional disturbances and any kind of chronic diseases in animal and man."

Udover REFLEX-studiet har der ifølge i øvrigt tilgængelige oplysninger været udført mere end 40 studier, som viser DNA-skader ved eksponering for radiofrekvent elektromagnetisk stråling[12].

Disse omfatter bl.a.:

Burlaka et al (2013), *"Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation."*[13], p. 223:

"In conclusion,the exposure of developing quail[14] *embryos in ovo to extremely low intensity RF-EMR of GSM 900MHz during at least one hundred and fifty-eight hours discontinuously leads to the significantly increased rates of superoxide and nitrogen oxide generation in embryo cells. This was accompanied by a significantly increased level of lipid peroxidation,a depression of key antioxidant enzymes activity,and significantly,2–3-fold,increased level of oxidative damage of DNA in embryo cells."*

Blank og Goodman (2011), *"DNA is a fractal antenna in electromagnetic fields."*[15], p. 411:

"Since DNA can interact with EMF over a wide range of frequencies, and does not appear to be limited to an optimal frequency, it has the functional properties of a fractal antenna.
…
From the above analysis of the effect of EMF on the stress response, DNA strand breaks and cancer epidemiology, the fractal property of DNA is apparent in the ELF and RF ranges.
…
Electron transfer is a plausible mechanism for EMF interactions with DNA at higher frequencies where higher energies are involved. The damage due to DNA strand breaks that occur at

12 Dokumentationsliste vedhæftes responsummet som **bilag 1**.

13 Offentliggjort i det videnskabelige tidsskrift "International Journal of Radiation Biology" vol. 87, no. 4, 2011, pp. 409-15.

14 Det er almindeligt at anvende dyr til at evaluere helbredsrisici for mennesker, og at anvende sådanne undersøgelser som basis for retningslinjer og grænseværdier, jf. f.eks. Engels et al (2014), *"Anthropogenic electromagnetic noise disrupts magnetic compass orientation in a migratory bird"* (Nature 2014, vol. 509), p. 354:*"...animal studies are commonly used to evaluate human health risks and have contributed to guidelines for human exposures..."* Det foreligger oplyst, at Miljøstyrelsen i skriftligt svar af 20. februar 2019 har oplyst tilsvarende og henvist til European Chemicals Agency med linket https://echa.europa.eu/da/information-on-chemicals/biocidal-active-substances

15 Offentliggjort i det videnskabelige tidsskrift "Experimental Oncology", vol. 35, no. 3, pp. 219 – 225.

higher frequencies, including ionising radiation, is generally attributed to oxidation, another chemical name for electron transfer. Because of the greater energy at higher frequencies, reactive oxygen species, such as peroxides, contribute to the DNA damage. However, DNA strand breaks occur over a wide range of frequencies, and do not demonstrate frequency optima related to molecular reaction kinetics." (understreget her)

Det hedder videre om de p.t. anvendte grænseværdier for menneskers eksponering over for radiofrekvent elektromagnetisk stråling (jf. pkt. 2.1 nedenfor) p. 413:

"...The existing 100 mT ELF exposure limit set by ICNIRP (International Commission for Non-Ionizing Radiation Protection) is many times higher than the 0.4 mT where a doubling of childhood leukemia risk is widely acknowledged. It has also been pointed out that the specific absorption rate (SAR), the widely used thermal standard for EMF safety, does not relate at all to the biological thresholds of the stress response in the ELF and RF ranges, and that the threshold for the same biological process differs by many orders of magnitude in the two ranges (Blank and Goodman 2004)."

Philips et al (2009), *"Electromagnetic fields and DNA damage."*[16], p. 85:

"RFR exposure does indeed appear to affect DNA damage and repair, and the total body of available data contains clues as to conditions producing effects and methodologies to detect them.

…

The lack of a causal or proven mechanism(s) to explain RFR-induced effects on DNA damage and repair does not decrease the credibility of studies in the scientific literature that report effects of RFR exposure, because there are several plausible mechanisms of action that can account for the observed effects. The relationship between cigarette smoking and lung cancer was accepted long before a mechanism was established. ..."

Panagopoulos (2019), *"Comparing DNA damage induced by mobile telephony and other types of* man-made electromagnetic fields*"*[17], p. 53 (resumé):

"The number of studies showing adverse effects on living organisms induced by different types of man-made Electromagnetic Fields (EMFs) has increased tremendously. Hundreds of peer reviewed published studies show a variety of effects, the most important being DNA damage which is linked to cancer, neurodegenerative diseases, reproductive declines etc. Those studies that are far more effective in showing effects employ real-life Mobile Telephony (MT) exposures emitted by commercially available mobile phones...." (understreget her)

Undersøgelsen konstaterer videre, at andre egenskaber end udelukkende signalstyrken er væsentlige årsager til skadevirkningerne, jf. ibid.:

"...The crucial parameter for the intense bioactivity seems to be the extreme variability of the polarized MT signals, mainly due to the large unpredictable intensity changes."

Tilsvarende fra konklusionen, ibid. p. 60:

"The importance of exposure variability shown in the present study implies the need to define EMF-exposures not only by frequency components and average intensity values, but by reporting maximum and minimum intensity as well, frequency variations, pulsing or

16 Offentliggjort i det videnskabelige tidsskrift "Pathophysiology" nr. 16 (2009), pp. 79–88.

17 Offentliggjort i det videnskabelige tidsskrift *"Mutation Research-Reviews in Mutation Research"* nr. 781, 2019, pp. 53–62.

continuous wave, modulation, and - of course - polarization."

Ibid., p. 59 – 60 (konklusion):

"It comes that variability in the EMF exposure is an extremely important factor in order for the specific type of polarized EMF to be able to induce biological/health effects.
…
The extreme and unpredictable variability of the real-life MT signals that apparently seems to be the reason for the corresponding intense bioactivity, does not concern only the 2nd generation (GSM) MT signals tested in our experiments and in the present review, but all existing types of digital MT signals (2nd, 3rd, 4th generation), and all types of modern digital microwave telecommunication signals/EMFs (DECT phones, Wi-Fi routers, etc.), since they all operate under the same principles combining RF carrier signals with ELF pulsing and modulation of similar frequency bands, emitting variable information each moment which in turn makes the emission variable in intensity, frequency, waveform etc. In fact, with every new generation of telecommunication devices (e.g. 3rd, 4th, 5th generation mobile phones or base antennas) the amount of information transmitted each moment (speech, text, images, video, internet, etc.) is increased, resulting in higher variability and complexity of the signals with the living cells/ organisms even more unable to adapt. The result of the recent study that found a real 3rd generation (UMTS) MT EMF to be more bioactive than real 2nd generation (GSM) MT EMF emitted by the same device [36] is in line with this fact." (understreget her)

Studiet D'Silva et al (2017)[18], omtalt som reference [36] i ovennævnte undersøgelse, rummer følgende beskrivelse af sine resultater og konklusion, jf. det tilhørende resumé:

*"**Results:** In our study, the exposure of developing chick embryos to 2G and 3G cell phone radiations caused structural changes in liver in the form of dilated sinusoidal spaces with haemorrhage, increased vacuolations in cytoplasm, increased nuclear diameter and karyorrhexis and significantly increased DNA damage.*
***Conclusion:** The chronic exposure of chick embryo liver to RFR emitted from 2G and 3G cell phone resulted in various structural changes and DNA damage. The changes were more pronounced in 3G experimental group. Based on these findings it is necessary to create awareness among public about the possible ill effects of RFR exposure from cell phone."*

Om den anvendte metode er bl.a. oplyst følgende, ibid. p. 6:

"A popular brand cell phone hand set and a service provider were used for network connection for both 2G and 3G exposure. For exposure activation, the cell phone was rung from another cell phone for duration of three minutes each, every half an hour, with the first exposure given at 12th hour of incubation (4.30 am-4.30 pm). The total exposure for a 12 hour period was 75 minutes followed by 12 hour of exposure-free period. This was repeated daily up to 12th day of incubation."

Studiet refererede endvidere, at:

"Non-thermal stress is more deleterious than thermal stress and is known to cause oxidative stress [5], production of free radicals [6], structural changes in plasma membrane [7], changes in ionic transport [8] and also increased DNA damage [9]."

1.2.2.1.1. Delkonklusion.

Der foreligger klar videnskabelig dokumentation for, at radiofrekvent elektromagnetisk stråling, også under de i Danmark anvendte grænseværdier, jf. pkt. 2.1 nedenfor, forårsager DNA-skader på både mennesker og dyr.

18 Offentliggjort i det videnskabelige tidsskrift *"Journal of Clinical and Diagnostic Research"*, 2017 Jul, Vol-11(7), pp. 5 – 9.

Panagopoulos (2019) dokumenterer, at det ikke alene er strålestyrken, der har betydning for den forventelige skadevirkning. Derudover dokumenteres, at fundene pga. teknologifællesskabet mellem generationerne også vil være gældende for 5G.

1.2.2.2. Kræft.

1.2.2.2.1. Klassificering.

IARC (International Agency for Research on Cancer) er WHOs agentur for kræftforskning.

Agenturet har i 2011 klassificeret elektromagnetisk stråling som "muligvis kræftfremkaldende for mennesker".[19]

En senere videnskabelig undersøgelse offentliggjort november 2018 konkluderede, at der i henhold til IARCs kriterier er grundlag for at klassificere elektromagnetisk stråling som "kræftfremkaldende for mennesker"[20], hvilken var begrundet således, med henvisning til gennemgang af en række undersøgelser foretaget forud herfor:

Miller et al november 2018[21], p. 674:

"...Analysis of a subset of cases (58% of all cases) based on operator-recorded information showed significant brain cancer risks for children with a significant trend of increase in risk with increasing years of use. Based on children's memory of both ipsilateral and contralateral use there were significant increased risk of brain cancer along with a marginal increase of risk with an increasing number of calls..."

Ibid., p. 675:

"Carlberg and Hardell (2013) also reported that persons diagnosed with a glioblastoma multiforme (GBM) exposed to RFR[22] emanating from WTDs[23] had a significantly shorter survival period than those without such exposures."

Ibid., p. 676:

"Coureau et al. (2014) reported on a French national study of mobile phone use and brain tumors (glioma and meningioma) between 2004 and 2006.
…
There was a marginal increase in risk with increasing hours of use (p_{trend}=0.07). A small number of urban users showed a significant 8-fold increased risk for brain tumors excluding temporal or frontal lobes (OR[24] 8.2. 1.37–49.07). The authors commented: 'Finally, we observed increased OR for urban use for gliomas, a result inconsistent with the hypothesis of a higher RF power output during calls in rural areas, documented by some Swedish study. However, our results are consistent with a recent international study showing no difference between rural and urban exposition in most countries except in Sweden, and a Hardell study when considering gliomas separately.'"

Ibid., p. 676:

"Momoli et al. (2017) undertook a re-analysis of the Canadian data from the 13-country case-control Interphone Study (2001–2004).

19 Jf. IARC monograph vol. 102 (2013), p. 419, pkt. 6.3.
20 En såkaldt "klasse 1-klassifikation" i IARC's system.
21 Offentliggjort i det videnskabelige tidsskrift "Environmental Research", 2018 nr. 167, pp. 673 – 683.
22 Radiofrequency radiation, jf. ibid. p. 673.
23 Wireless Transmitter Devices, jf. ibid. p. 673.
24 Odds ratio, jf. ibid. p. 674.

…
For glioma, when comparing those in the highest quartile of use (> 558 lifetime hours) to those who were not regular users, the odds ratio was 2.0 (95% confidence interval: 1.2, 3.4). After adjustment for selection and recall biases, the odds ratio was 2.2 (95% limits: 1.3, 4.1), thus allaying concerns that bias could explain the positive findings in the Interphone study."

Ibid., p. 676:

"Akhavan-Sigari et al. (2014) reported that patients with glioblastoma multiforme who had used cellphones≤3 h per day had better survival than those with cellphone use of ≥3 h per day.
…
This study shows that genetic changes, compatible with carcinogenic effects, result from higher exposure to RFR."[25]

Ibid., p. 676:

"Carlberg and Hardell (2015) performed a pooled analysis from 1997 to 2003 and 2007–2009 of the risk of meningioma from cell and cordless phone use. In total, 1625 meningioma cases and 3530 controls were analyzed. Overall no association with use of mobile or cordless phones was found. However, they reported an increased risk among heavy users of both mobile and cordless phones from various wireless phone types (wireless combines all phone types) (Table 11). The risk increased significantly per 100 h of use from four wireless phones categories."

Ibid., p. 677:

"Hardell et al. (2013a) pooled acoustic neuroma results from casecontrol studies conducted in 1997–2003 and 2007–2009, including 316 participating cases and 3530 controls. ... There is some evidence of a dose-response relationship is evident with mobile and cordless phones associated with ORs of 4.5 and 6.5 respectively for 20 or more years of use. There were similar results per cumulative hours of use (Table 12)."

Ibid., p. 677:

"Moon et al. (2014), in a matched case-control study from Korea examining 119 cases of vestibular schwannoma and 238 controls attending for routine examinations in the same institution found no difference between cases and controls in the duration, time of use or cumulative use of mobile phones. However, in a case-case analysis they found that vestibular Schwannoma tumor volume was greater in those with higher use compared to lower use of mobile phones and in those with regular compared to non-regular use (Table 13)."

Ibid., p. 678:

"Zada et al. (2012) examined data from three major U.S. cancer registries: Los Angeles County, California Cancer Registry, and the National Cancer Institute's Surveillance, Epidemiology and End Result for 12 U.S. states (SEER 12) from 1992. The APC for GBM (grade IV glioma) and Glioma was reported by brain region. Table 17 shows APC changes by cancer registry for GBM and for glioma located in three anatomical regions of the brain, showing significant increases compatible with increasing use of mobile phones.
Consistent with the study above, Cardis et al. (2011) reported that the combined percentage of the total radiation absorbed by the frontal lobe (19%), the temporal lobe (50%) and the cerebellum (18%) was 81% at 900 MHz and was 86% at 1800 MHz (frontal lobe 14%, temporal lobe 50%, cerebellum 13%)."

Ibid., p. 679:

25 Patienterne i studiet (63 voksne, heraf 38 M 25 K) blev opereret for kræftsvulster år 2008 – 2011, jf. Akhavan-Sigari et al 2014, p. 117.

"7. Case series

West et al. (2013) reported multiple[26] primary breast cancers in young women who had regularly placed a cellphone in their bras (Table 20). Tumors were reported to have occurred subcutaneously directly under the antennas of the phones. Subsequently, a number of other such cases have come to light with unusually located breast tumors relative to reported cell phone storage in the bra.
Peleg (2012) discussed a cancer cluster among young workers at an Israeli Antenna Range Facility. It was believed that significant RFR exposures took place as a result of workplace conditions. Five of about 30 workers were diagnosed with cancer. This was regarded as significantly greater than the expectation. Peleg et al. (2018) extended this analysis to 47 patients with cancer previously exposed to whole-body prolonged RFR, mainly from communication equipment and radar. They found that the percentage frequency of haemo-lymphatic (HL) cancers in the case series was very high, at 40% with only 23% expected for the series age and gender profile, 95% confidence interval: 26–56%, $p < 0.01$; 19 out of the 47 patients had HL cancers.
Stein et al. (2011) studied 56 cancers among 49 military personnel (47 male, 7 females) exposed to intense prolonged RFR between 1992 and 2011. Based on exposure information reconstructed from reported histories, it was assumed that significant RFR exposures took place as a result of workplace conditions. The average duration of exposure was 13 years; the average age at diagnoses was 43. There appeared to be an excess of both haemolymphatic and testicular cancers."

Ibid., p. 680 (diskussion af resultater):

"Nevertheless[27], recent case-control studies from Sweden and France corroborate findings of earlier studies in providing support for making a causal connection between cell phone use and brain cancer, as well as acoustic neuroma, also called Vestibular Schwannoma. Hardell and Carlberg (2013) concluded that the Bradford Hill criteria for causality have now been fulfilled. It is notable that three recent meta-analyses all confirm significant increased risk of glioma after 10 or more years of use of cell phones (Bortkiewicz et al., 2017; Prasad et al., 2017; Yang et al., 2017). The Aydin et al. (2011) data that relied on billing records along with children's recall of their uses of phones approaches and in some instances met conventional tests of statistical significance and indicated that four years or more of heavy cell phone radiation causes glioma in children. This finding is consistent with that of Hardell and Carlberg (2015) who showed that those who began using cell phones and/or cordless phones regularly as children had between 4 and 8-fold increased risk of glioma as adults."

Ibid., p. 680 (diskussion af resultater):

"Potential cancer sites and other outcomes for consideration in new studies include... Other sites than brain and acoustic neuroma could potentially increase in incidence when untested whole-body exposure occurs, this may be the case with several evolving technologies....other possible sources of exposures that have not been evaluated include areas close to cellular base station antennas, the yet-to-be introduced 5 G communication systems, and rapidly evolving occupational exposure and novel systems for Wi-Fi (Peleg, 2009)."

Ibid., p. 681 (diskussion af resultater):

"There are indications particularly from the Ramazzini animal studies that other environmental exposures might make people more susceptible to a combination of exposures (Falcioni et al., 2018). This combinatorial issue been noted in studies of occupational exposure to chemicals, metals and electromagnetic fields (Navas-Acien et al., 2002). Separately, no effects were

26 Der var tale om 4 patienter i alderen 21, 21, 33 og 39, jf. ibid. tabel 20.

27 Citatet er i artiklen efterstillet en gennemgang af sædvanlige forbehold i forbindelse med anvendelse af de forskellige, gængse forskningsmetoder, som ligeledes har været anvendt i de i artiklen omhandlede studier.

observed but when combined with EMF strong results were found. In the Ramazzini studies finding a synergistic interaction between RFR and ionizing radiation, RFR served as a promoter while in the NTP animal studies RFR served as a direct carcinogen and genotoxic agent (National Toxicology Program, a, b, 2018.).”

Ibid., p. 681 (diskussion af resultater):

”Individual hypersensitivity to electric and radiofrequency fields (EHS) is a relatively newly reported phenomenon in the west, although cases of radiation sickness have been found in the former Soviet literature from the 1960s and 1970s. Case studies and individual reports together identify a population which would benefit from RFR exposure reduction (Davis et al., 2017). Because of serious methodological difficulties in operationalizing the concept and a lack of investment in research, definitive epidemiological studies of EHS have not yet been conducted.”

Ibid., p. 681 (diskussion af resultater):

”However, non-cancer outcomes such as sperm damage, hearing loss and loss of visual acuity are likely to be more commonly linked to mobile phone use.”

Ibid., p. 681 (konklusioner):

”The Epidemiological studies reported since the 2011 IARC Working Group meeting are adequate to consider RFR as a probable human carcinogen (Group 2 A). However, they must be supplemented with the recently reported animal data as performed at the Ramazzini Institute and the US National Toxicology Program as well as by mechanistic studies. <u>These experimental findings together with the epidemiology reviewed here are sufficient in our opinion, to upgrade the IARC categorization of RFR to Group 1, carcinogenic to humans.</u>
…
In light of the evolving science concerning mobile phone and screen time exposures and the longer-term risk of cancer established by both epidemiological and toxicological studies, current evidence is strong enough to go from precaution concerning possible risk to prevention of known risks.
…
The precautionary principle should be applied now and suitable warning messages provided to adults and critically to children and their parents.
…
experimental evaluations and modeling are essential before distributing newer systems (e.g. 5 G) for which no safety data have been obtained. The absence of systematic testing of such technologies should not be confused with proof of safety.
…
In the meantime, the evidence amassed thus far from epidemiology strengthens the case for instituting the precautionary principle with respect to exposures to RFR, especially to young children and men and women that wish to reproduce. … Where studies have been carried out on human sperm quantity and quality there are increasing indications of serious human health impacts. To ignore those findings and subject humans to unevaluated novel RFR frequencies places current and future generations at risk.” (understreget her)

<u>1.2.2.2.2. Øvrigt.</u>

En lang række øvrige videnskabelige studier bekræfter, at udsættelse for radiofrekvent elektromagnetisk stråling (også under de p.t. i Danmark anvendte grænseværdier, jf. pkt. 2.1 nedenfor) kan være kræftfremkaldende. En række af dem er:

<u>Blank og Goodman (2011)</u>, *”DNA is a fractal antenna in electromagnetic fields.”*[28], p. 411:

28 Offentliggjort i det videnskabelige tidsskrift ”Experimental Oncology”, vol. 35, no. 3, pp. 219 – 225.

"Regarding the connection between EMF and the incidence of cancer, the different EMF energy levels in the non-ionising and ionising ranges all involve interaction with and activation of DNA and induction of protein synthesis. The ability of EMF to cause DNA strand breaks and damage to proteins in the non-ionising range is similar to the destructive effects on DNA of the much more energetic X-rays and gamma rays in the ionising ranges, where it is generally acknowledged that the cancers are due to DNA damage. The recent epidemiology studies in the non-ionising range link EMF-caused changes in DNA with cancer. Additional support comes from the study showing that the absence of DNA repair genes is associated with a greater incidence of leukemia from exposure to low frequency EMF (Yang et al. 2008)." (understreget her)

Lerchl et al (2015), *"Tumor promotion by exposure to radiofrequency electromagnetic fields below exposure limits for humans"*, p. 585 (resumé):

"...Since many of the tumor-promoting effects in our study were seen at low to moderate exposure levels (0.04 and 0.4 W/kg SAR), thus well below exposure limits for the users of mobile phones, further studies are warranted to investigate the underlying mechanisms. Our findings may help to understand the repeatedly reported increased incidences of brain tumors in heavy users of mobile phones."

Yakymenko et al 2015, jf. pkt. 1.2.1. ovenfor, viste bl.a., at lav-intensitets RFR kunne medføre kræft (p. 196).

Prasad et al (2017), *"Mobile phone use and risk of brain tumours: a systematic review of association between study quality, source of funding, and research outcomes."*[29], p. 808 (konklusion):

"In our review of the literature and meta-analysis of case–control studies, we found evidence linking mobile phone use and risk of brain tumours especially in long-term users (>10 years). We also found a significantly positive correlation between study quality and outcome in the form of risk of brain tumour associated with use of mobile phones. Higher quality studies show a statistically significant association between mobile phone use and risk of brain tumour. Even the source of funding was found to affect the quality of results produced by the studies."

Der er tale om et systematisk, videnskabeligt review af den foreliggende forskning, som altså fandt klar basis for at kæde brugen af mobiltelefoner sammen med kræftsvulster i hjernen.

Endvidere fandt det pågældende review, at de studier, der havde den bedste videnskabelige kvalitet, var de samme, som dem, der fandt den pågældende sammenhæng, og at finansieringskilden også havde spillet en rolle i studiernes kvalitet.

Det amerikanske National Institute of Health udgav november 2018 rapporten *"TOXICOLOGY AND CARCINOGENESIS STUDIES IN Hsd:SPRAGUE DAWLEY SD RATS EXPOSED TO WHOLE-BODY RADIO FREQUENCY RADIATION AT A FREQUENCY (900 MHz) AND MODULATIONS (GSM AND CDMA) USED BY CELL PHONES"*, hvoraf fremgår p. 125 - 126 (konklusioner):

"GSM-Modulated RFR

29 Offentliggjort i det videnskabelige tidsskrift "Neurological Sciences", 2017, vol. 38, pp. 797 – 810.

Under the conditions of this 2-year whole-body exposure study, there was clear evidence of carcinogenic activity[30] of GSM-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla were also related to RFR exposure. The incidences of benign or malignant granular cell tumors of the brain, adenoma or carcinoma (combined) of the prostate gland, adenoma of the pars distalis of the pituitary gland, and pancreatic islet cell adenoma or carcinoma (com-bined) may have been related to RFR exposure. There was equivocal evidence of carcinogenic activity of GSM-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats based on the incidences of schwannomas of the heart.*

…

CDMA-Modulated RFR

Under the conditions of this 2-year whole-body exposure study, there was clear evidence of carcinogenic activity of CDMA-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain were also related to RFR exposure. The incidences of adenoma of the pars distalis of the pituitary gland and adenoma or carcinoma (combined) of the liver may have been related to RFR exposure. There was equivocal evidence of carcinogenic activity of CDMA-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley SD rats based on the incidences of malignant schwannoma of the heart, malignant glioma of the brain, and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla. Increases in nonneoplastic lesions of the heart, brain, and prostate gland in male rats, and of the brain in female rats occurred with exposures to CDMA-modulated RFR at 900 Mhz." (understreget her)

Om den umiddelbare overførbarhed af disse resultater på mennesker hedder det, ibid. p. 125:

"The malignant schwannomas of the heart observed in male rats in the current studies and the malignant gliomas observed in the brain of male rats, arise from the same cell type as the acoustic neuromas (vestibular schwannomas) observed in humans, though in a different location. This lends credence to the possible association of these tumors with cellular phone use. The cellular origin of malignant gliomas in the rat brain is unclear, but they do arise from glial cells (support cells in the brain), as do human glioblastomas, so it is possible that such an association exists for these tumors as well. However, the interpretation of these findings with respect to specific risks to humans from cellular telephone use is beyond the scope of the current studies. Further efforts to characterize the molecular basis by which RFR elicits its effects in rats, and a more complete assessment of the exposure conditions in the current studies in relation to exposures to humans from cellular telephone technologies should provide con-text to aid understanding of the implications of the current findings to human health." (understreget her)

Falcioni et al. (2018), *"Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission"*[31], p. 496 (resumé):

"The RI[32] findings on far field exposure[33] to RFR are consistent with and reinforce the results of

30 Asterisken refererer til undersøgelsens p. 16, hvor de forskellige bevisstandarder er nærmere defineret. "Clear evidence" er defineret som: *"...studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy."*

31 Offentliggjort i det videnskabelige tidsskrift "Environmental Research", 2018, vol. 165, pp. 496 – 503.

32 RI står for "Ramazzini Institute", som var det forskningsinstitut, hvor undersøgelsen blev foretaget.

33 Far field exposure indebærer i dette tilfælde, at undersøgelsen genskabte forholdene for bestråling med en 1.8 GHz mobilmast, jf. ibid. p. 497, pkt. 2.1.

the NTP study[34] on near field exposure, as both reported an increase in the incidence of tumors of the brain and heart in RFR-exposed Sprague-Dawley rats. These tumors are of the same histotype of those observed in some epidemiological studies on cell phone users. These experimental studies provide sufficient evidence to call for the reevaluation of IARC conclusions regarding the carcinogenic potential of RFR in humans." (understreget her)

Uddybende er konstateret, jf. ibid. p. 501:

"...The statistically significant increase in the incidence of heart Schwannomas observed in male rats in the late part of their life, both in the RI and NTP studies, are consistent with the epidemiological findings, where the highest increase in risk of vestibular Schwannoma among humans exposed to RFR was observed in men over 50 years of age with the highest cumulative exposure (Hardell et al., 2013, 2003)."

Forsøget blev gennemført således, at de eksponerede dyr blev udsat for en stråleabsorption på estimeret 0,001 til 0,3 W/kg (jf. p. 499), hvilket er betydeligt lavere end den anvendte grænseværdi i Danmark på 2 W/kg, jf. pkt. 2.1 nedenfor.

De statistisk signifikante resultater fremkom i den gruppe af rotter, som var blevet udsat for en feltstyrke på 50 V/m. Dette er under grænseværdien på 58,34 V/m, som anvendes i Danmark for en frekvens på 1,8 GHz, jf. pkt. 2.1 nedenfor.

Martin Pall 2018[35] konstaterede maj 2018, at der på det tidspunkt eksisterede minimum 35 videnskabelige undersøgelser (siden 1978), som dokumenterede, at radiofrekvent elektromagnetisk stråling var kræftfremkaldende.

Panagopoulos (2019), jf. pkt. 1.2.1. ovenfor, henviste ligeledes til "hundreder af peer reviewed-artikler", som viste en række effekter fra elektromagnetiske felter, inkl. kræft (p. 53).

Sundhedsministeren har i sin besvarelse af 12. april 2019 af spørgsmål 693 i Folketingets Sundheds- og Ældreudvalg fremlagt en opgørelse fra Sundhedsdatastyrelsens cancerregister, som viser en klar stigning i registrerede tilfælde af kræft i form af glioblastom inden for de sidste 20 år.

Stigningen er særligt markant fra 2005 til 2006 i aldersgruppen >30 år, og viser gennemsnitligt set en fordobling af tilfældene i denne aldersgruppe i perioden 2006 til 2017, set i forhold til den forudgående periode 1995 til 2006.

Samlet ses en stigning på op mod 80 % i forekomsten i de senere år 2015 – 2017, sammenlignet med det generelle niveau før 2006.

34 "NTP-studiet" er det umiddelbart ovenfor omtalte fra det amerikanske National Health Institute.

35 PhD, prof.emeritus i biokemi og Basic Medical Sciences, Martin L. Pall - *"5G: Great risk for EU, U.S. and International Health! Compelling Evidence for Eight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures and the Mechanism that Causes Them"*, pp. 15 – 16.

Tabel 1 Antal incidente tilfælde af Glioblastom, 1995-2017

Kilden : Cancerregisteret			
Udtrækskriterier :			
Der er trukket på :			
Glioblastom ICD03-morfologi = 94403			
og Giant cell glioblastom ICD03-morfologi =			
	Antal		
aar	**<= 30 år**	**>30 år**	**I alt**
1995	9	143	152
1996	11	133	144
1997	<5	176	
1998	8	200	208
1999	8	169	177
2000	<5	173	
2001	6	156	162
2002	9	163	172
2003	<5	185	
2004	7	174	181
2005	<5	174	
2006	<5	221	
2007	<5	196	
2008	6	237	243
2009	<5	252	
2010	<5	271	
2011	<5	253	
2012	9	250	259
2013	7	280	287
2014	<5	292	
2015	10	311	321
2016	7	300	307
2017	<5	288	

Kilde: Cancerregisteret, 2019

Anm.: Tal under fem er angivet med < 5 af hensyn til diskretionering og summen for i alt er i disse tilfælde fjernet

Anm.: Opdelt i kolloner mindre eller lige end 30 år, større end 30 år og i alt

Den samme fordoblingstendens er dokumenteret i England, jf. Philips et al (2018), *"Brain Tumours: Rise in Glioblastoma Multiforme Incidence in England 1995–2015 Suggests an Adverse Environmental or Lifestyle Factor"*[36], hvoraf fremgår følgende (p. 1, resumé):

*"*Results. *We report a sustained and highly statistically significant ASR*[37] *rise in glioblastomamultiforme (GBM) across all ages.The ASR for GBM more than doubled from 2.4 to 5.0, with annual case numbers rising from 983 to 2531. Overall, this rise is mostly hidden in the overall data by a reduced incidence of lower-grade tumours.* Conclusions. *The rise is of importance for clinical resources and brain tumour aetiology. The rise cannot be fully accounted for by promotion of lower–grade tumours, random chance or improvement in diagnostic techniques as it affects specific areas of the brain and only one type of brain tumour. Despite the large variation in case numbers by age, the percentage rise is similar across the age groups, which suggests widespread environmental or lifestyle factors may be responsible."* (understreget her)

I Holland er dokumenteret en stigning på 20 % over en 21-årig periode fra 1989 til 2010, jf. Vincent et al (2014), *"Changing incidence and improved survival of gliomas"*[38], p. 2311:

"The incidence rate of glioma increased from 4.9 per 100,000 inhabitants in 1989 to 5.9 in 2010..."

36 Offentliggjort i det videnskabelige tidsskrift "Journal of Environmental and Public Health" 2018, art.ID 7910754.
37 Forkortelse for "Age Standardised Rate".
38 Offentliggjort i det videnskabelige tidsskrift "European Journal of Cancer", 2014, vol. 50, pp. 2309 – 2318.

1.2.2.3. Andre helbredsskader på mennesker.

Neufeld og Kuster (2018) har i artiklen *"SYSTEMATIC DERIVATION OF SAFETY LIMITS FOR TIME-VARYING 5G RADIOFREQUENCY EXPOSURE BASED ON ANALYTICAL MODELS AND THERMAL DOSE"* konstateret, at selv ved korte eksponeringer over for stråling svarende til den planlagte 5G-stråling med højere frekvenser og/eller ændret modulation, m.v., jf. pkt. 1.1. ovenfor, vil der kunne ske vævsskader på mennesker, jf. p. 705, 706 og 711:

"Extreme broadband wireless devices operating above 10 GHz may transmit data in bursts of a few milliseconds to seconds. …these bursts may lead to short temperature spikes in the skin of exposed people. … To stay consistent with the current safety guidelines, safety factors of 10 for occupational exposure and 50 for the general public were applied. … The results also show that the peak-to-average ratio of 1,000 tolerated by the International Council on Non-Ionizing Radiation Protection guidelines may lead to permanent tissue damage after even short exposures, highlighting the importance of revisiting existing exposure guidelines.
…
THE FIFTH generation of wireless communication technology (5G) promises to facilitate transmission at data rates up to a factor of 100 times higher than 4G. For that purpose, higher frequencies (including millimeter-wave bands), broadband modulation schemes, and thus faster signals with steeper rise and fall times will be employed, potentially in combination with pulsed operation for time domain multiple access. 5G is designed as a ubiquitous communication system spanning applications such as high-bandwidth mobile data and telephony, real-time machine-to-machine communication (e.g., autonomous mobility), and the Internet of Things (IoT). Exposure to radiofrequency (RF) radiation from wireless devices to large radar installations and medical equipment can result in increases in body core temperature or cause localized temperature rises, with the potential for adverse health effects. The thresholds for frequencies above 10 MHz set in current exposure guidelines (ICNIRP 1998; IEEE 2005, 2010) are intended to limit tissue heating.
…
However, short pulses can lead to important temperature oscillations, which may be further exacerbated at high frequencies (>10 GHz, fundamental to 5G), where the shallow penetration depth leads to intense surface heating and a steep, rapid rise in temperature...
…
The recommendations in the ICNIRP guidelines limit the power density during short pulses to 1,000 times the limit for the time-averaged incident power density. The IEEE standard limits the radiant exposure (energy absorption per unit area) during any 100 ms to one-fifth of the total radiant exposure for the whole averaging time. The physical or biological rationales for these limits, however, are not provided.
…
Laakso et al. (2017) … The authors conclude that the current guidelines do not adequately prevent excessive heating from pulsed exposure, as peak temperatures can easily exceed the mean temperature by more than a factor of 3 and suggest that radiant exposure limits be introduced.
Morimoto et al. (2017) … They conclude that the thermal time constants can be as short as 30 s for narrow-beam exposures and that short pulses can carry enough energy to cause injuries;
…
Another conclusion of this study is that the current ICNIRP (1998) and IEEE (2005, 2010) guidelines urgently need to be revised, as the duty cycle of 1,000 currently tolerated can produce unacceptable temperature increases that may result in permanent tissue damage. ..." (understreget her)

Cindy Russell (2018), *"5 G wireless telecommunications expansion: Public health and environmental implications"*, p. 485:

"There are no long term exposure guidelines, nor are there guidelines for low level, non-thermal or biological effects considered in the International Commission on Non-Ionizing

Radiation Protection (ICNIRP) standards which are the basis for standards used worldwide..."

Videre ibid., p. 491:

"Although 5G technology may have many unimagined uses and benefits, it is also increasingly clear that significant negative consequences to human health and ecosystems could occur if it is widely adopted. Current radiofrequency radiation wavelengths we are exposed to appear to act as a toxin to biological systems. A moratorium on the deployment of 5G is warranted, along with development of independent health and environmental advisory boards that include independent scientists who research biological effects and exposure levels of radiofrequency radiation.
…
Public health regulations need to be updated to match appropriate independent science with the adoption of biologically based exposure standards prior to further deployment of 4G or 5G technology."

Martin Pall 2018[39] konstaterede maj 2018, at der kunne påvises følgende yderligere skadevirkninger ved radiofrekvent elektromagnetisk stråling på mennesker:

- nedsat fertilitet og kønsdrift, øgede spontane aborter, m.v. (18 studier siden 1971),
- neurologiske/neorupsykiatriske effekter (25 studier siden 1966),
- apoptose/celledød (13 studier siden 1971),
- hormonelle effekter (12 studier siden 1971), og
- forøget niveau af calciumioner intracellulært, hvilket forårsager en række sygdomme (15 studier siden 1988).

Pall anførte, ibid. p. 1 – 2:

"Each of these effects is produced via the main mechanism of action of microwave/lower frequency EMFs, activation of voltage-gated calcium channels (VGCCs) (Chapter 2). Each of them is produced via what are called downstream effects of VGCC activation. It follows from this that we have a good understanding not only that these effects occur, but also how they can occur. The extraordinary sensitivity of the VGCC voltage sensor to the forces of the EMFs tells us that the current safety guidelines allow us to be exposed to EMF levels that are something like 7.2 milion times too high. That sensitivity is predicted by the physics. Therefore, the physics and the biology are each pointing to the same mechanism of action of non-thermal EMFs. The different effects produced are obviously very deep concerns. They become much deeper and become existential threats when one considers that several of these effects are both cumulative and eventually irreversible.
…
Obviously 4G and 5G will make the situation much worse." (understreget her)

1.2.2.4. Særligt vedr. børn og kræft eller andre helbredsskader.

Der findes en række undersøgelser, hvoraf visse tillige er omtalt ovenfor, som specifikt omtaler skadevirkninger og risici for kræft eller andre helbredsskader for børn, hvoraf fremhæves:

Divan et al (2012), *"Cell phone use and behavioural problems in young children"*[40], p. 524 (resumé):

39 PhD, prof.emeritus i biokemi og Basic Medical Sciences, Martin L. Pall - *"5G: Great risk for EU, U.S. and International Health! Compelling Evidence for Eight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures and the Mechanism that Causes Them"*, pp. 8 - 15.

40 Offentliggjort i det videnskabelige tidsskrift "Journal of Epidemiology and Community Health", 2012, vol. 66, nr. 6, pp. 524 – 529.

"The findings of the previous publication were replicated in this separate group of participants demonstrating that cell phone use was associated with behavioural problems at age 7 years in children, and this association was not limited to early users of the technology. Although weaker in the new dataset, even with further control for an extended set of potential confounders, the associations remained."

Denne undersøgelse, som var en gentagelse af en tidligere undersøgelse foretaget af samme forskere, bekræftede, at der var en sammenhæng imellem adfærdsproblemer hos børn i 7-årsalderen og brug af mobiltelefoner hos moderen før fødslen samt børnenes egen brug efter fødslen, uden at det på baggrund af undersøgelsen med sikkerhed kunne lægges til grund, at der var en årsagsforbindelse, jf. p. 529. Undersøgelsen bekræftede således en mulig risiko.

Birks et al (2017), *"Maternal cell phone use during pregnancy and child behavioral problems in five birth cohorts"*[41], p. 1 (resumé, manuskriptudgave):

"Overall, 38.8% of mothers, mostly from the Danish cohort, reported no cell phone use during pregnancy and these mothers were less likely to have a child with overall behavioral, hyperactivity/inattention or emotional problems. Evidence for a trend of increasing risk of child behavioral problems through the maternal cell phone use categories was observed for hyperactivity/inattention problems (OR for problems in the clinical range: 1.11, 95%CI 1.01, 1.22; 1.28, 95%CI 1.12, 1.48, among children of medium and high users, respectively). This association was fairly consistent across cohorts and between cohorts with retrospectively and prospectively collected cell phone use data." (understreget her)

Ibid., p. 13 (konklusion, manuskriptudgave):

"Maternal cell phone use during pregnancy may be associated with an increased risk of behavioral problems, particularly hyperactivity/inattention problems, in the offspring. This is the largest study to date to evaluate these associations and to show mostly consistent results across cohorts with retrospectively and prospectively assessed maternal cell phone use. Still, the interpretation of these results is unclear and should take into consideration that uncontrolled confounding by social factors or maternal hyperactivity may influence both maternal cell phone use and child behavioral problems."

Der er således tale om et forbeholdende, men konsistent, resultat, som bekræfter, at der kan være en øget risiko for helbredsproblemer for børn ved mødres brug af mobiltelefon under graviditeten.

Sudan et al (2018), *"Maternal cell phone use during pregnancy and child cognition at age 5 years in 3 birth cohorts"*[42], p. 155 (resumé):

"We observed patterns of lower mean cognition scores among children in relation to high frequency maternal prenatal cell phone use. The causal nature and mechanism of this relationship remain unknown."

Der foreligger en række relaterede forsøg på dyrefostre, bl.a.:

41 Offentliggjort i det videnskabelige tidsskrift "Environment International", 2017, vol. 104, pp. 122 – 131.
42 Offentliggjort i det videnskabelige tidsskrift "Environment International", 2018, vol. 120, pp. 155 – 162.

Jing et al (2012), *"The influence of microwave radiation from cellular phone on fetal rat brain"*, p. 64:

"In order to protect human's health from the microwave damage, the relevant radiation limits have been given by many countries. The current limited guidelines for microwave from cellular phone in U.S. and Europe are 1.6 W/kg and 2.0 W/kg, respectively. New lower limits should also be used for children and/or pregnant women.
Due to the proximity of cellular phone antenna to the user's ear and head, the brain is inevitably exposed to EMFs with a relatively high specific absorption ratio (SAR), so the potentially danger from EMFs has been a concern of more and more people, especially by pregnant women.

…

As a whole, the results obtained in the present study indicate that exposure to EMFs of cellular phone (SAR 0.9 W/kg) could induce modifications in the fetal rat brain, not only oxidative stress system but also neurotransmitters. Because of the widespread use of cellular phones, further investigations with complementary techniques will be necessary to understand the mechanism of relation between EMFs of cellular phone and physiological implications."

Det bemærkes, at stråleabsorptionsraten ligger under den p.t. anvendte grænseværdi i Danmark, på 2 W/kg, jf. pkt. 2.1 nedenfor.

Megha et al (2015), *"Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain"*[43], p. 164 (konklusion):

"In conclusion, prolonged exposure to low intensity microwave radiation at frequencies 900, 1800 and 2450 MHz leads to oxidative stress and inflammatory imbalances which subsequently leads to DNA damage in brain. These findings suggest that microwave radiation induced oxidative stress and inflammatory imbalances may be the causative factors involved in causing DNA strand breaks in brain cells."

Aldad et al (2012), *"Fetal Radiofrequency Radiation Exposure From 800-1900 Mhz-Rated Cellular Telephones Affects Neurodevelopment and Behavior in Mice"*[44], p. 2 og 6:

"Overall, the mice exposed in-utero to radiation were hyperactive, had decreased memory, and decreased anxiety.
…
Our findings indicated significant electrophysiological and behavioral changes in mice exposed in-utero to radiation. The significant trend between the groups treated for 0, 9, 15, and 24 hours/day demonstrates that the effects are directly proportional to usage time, and suggests that safety limits, particularly for pregnant women, can be established. Though it is difficult to translate these findings to human risks and vulnerability, we identify a novel potential contribution to the increased prevalence in hyperactive children, one that is easily prevented. However, it is important to note that hyperactivity and anxiety are closely related and may confound one another.
...
In summary, we demonstrate that fetal radiofrequency radiation exposure led to neurobehavioral disorders in mice. We anticipate these findings will improve our understanding of the etiology of neurobehavioral disorders. The rise in behavioral disorders in developed countries may be, at least in part, due to a contribution from fetal cellular telephone radiation exposure. Further testing is warranted in humans and non-human primates to determine if the risks are similar and to establish safe exposure limits during pregnancy."

43 Offentliggjort i det videnskabelige tidsskrift "NeuroToxicology" 2015, vol. 51, pp. 158 – 165.
44 Offentliggjort i det videnskabelige tidsskrift "Nature Scientific Reports" 2, art.no. 312, 2012. Der er i 2013 udstedt en korrektion til artiklen, som ikke ændrer konklusionerne, der har art.nr.. 1320.

Buchner og Eger (2011), *"Changes of Clinically Important Neurotransmitters under the Influence of Modulated RF Fields—A Long-term Study under Real-life Conditions"*[45], p. 1 (oversat fra tysk):

"Since the 1960s, occupational studies on workers with continuous microwave radiation exposures (radar, manufacturing, communications) in the Soviet Union have shown that RF radiation exposures below current limits represent a considerable risk potential. A comprehensive overview is given in the review of 878 scientific studies by Prof. Hecht, which he conducted on behalf of the German Federal Institute of Telecommunications (contract no. 4231/630402) (2, 3).
As early as the 1980s, US research projects also demonstrated in long-term studies that rats raised under sterile conditions and exposed to "low-level" RF radiation showed signs of stress by increased incidences of endocrine tumors..."

Endvidere p. 9 (summary of results):

"...dopamine levels decrease substantially after the exposure begins. Even after one and a half years, the initial levels are not restored. Six months after the activation of the transmitter, PEA levels decrease continuously over the entire exposure period. Only in the exposure group above 100 µW/m2 is this effect observed immediately. All findings were observed well below current exposure limits (14)." [46]

Tillige p. 12 (epidemiological evidence):

"As part of the German Mobile Telecommunication Research Programme, approximately 3000 children and adolescents were studied in Bavaria for their individual cell phone radiation exposure levels in relation to health problems. Among the various data sets, the data set regarding behavioral problems showed a significant increased risk for both adolescents (OR: 3.7, 95%-CI: 1.6-8.4) and also children (OR: 2.9, 95%-CI: 1.4-5.9) in the highest exposure group (56). For the first time, the "Rimbach Study" provides a model of explanation in biochemical terms. "

Sudan et al (2012), *"Prenatal and Postnatal Cell Phone Exposures and Headaches in Children."* [47], p. 1 (resumé, manuskriptudgave):

"In this study, cell phone exposures were associated with headaches in children, but the associations may not be causal given the potential for uncontrolled confounding and misclassification in observational studies such as this. However, given the widespread use of cell phones, if a causal effect exists it would have great public health impact."

Byun et al (2013), *"Mobile Phone Use, Blood Lead Levels, and Attention Deficit Hyperactivity Symptoms in Children: A Longitudinal Study"* [48], p. 1:

"The results suggest that simultaneous exposure to lead and RF from mobile phone use was associated with increased ADHD symptom risk, although possible reverse causality could not be ruled out."

45 Oprindeligt offentliggjort på tysk i det videnskabelige tidsskrift "Umwelt-Medizin-Gesellschaft", 2011, vol. 24, nr. 1, pp. 44 – 57.
46 (14) er forskernes henvisning til ICNIRP-grænseværdierne, jf. pkt. 2.1. nedenfor.
47 Offentliggjort i det videnskabelige tidsskrift "The Open Pediatric Medicine Journal" 2012, nr. 6, pp. 46 – 52.
48 Offentligt i det videnskabelige onlineskrift "PLOS One" d. 21. marts 2013.

Herbert og Sage (2013), *"Autism and EMF? Plausibility of a pathophysiological link part II "*, p. 211 (resumé):

"Autism spectrum conditions (ASCs) are defined behaviorally, but they also involve multileveled disturbances of underlying biology that find striking parallels in the physiological impacts of electromagnetic frequency and radiofrequency radiation exposures (EMF/RFR).
…
Brain oxidative stress and inflammation as well as measures consistent with blood–brain barrier and brain perfusion compromise have been documented. Part II of this paper documents how behaviors in ASCs may emerge from alterations of electrophysiological oscillatory synchronization, how EMF/RFR could contribute to these by detuning the organism, and policy implications of these vulnerabilities. It details evidence for mitochondrial dysfunction, immune system dysregulation, neuroinflammation and brain blood flow alterations, altered electrophysiology, disruption of electromagnetic signaling, synchrony, and sensory processing, detuning of the brain and organism, with autistic behaviors as emergent properties emanating from this pathophysiology.
…
All of these phenomena also occur with EMF/RFR exposure that can add to system overload ('allostatic load') in ASCs by increasing risk, and can worsen challenging biological problems and symptoms; conversely, reducing exposure might ameliorate symptoms of ASCs by reducing obstruction of physiological repair.
…
With dramatic increases in reported ASCs that are coincident in time with the deployment of wireless technologies, we need aggressive investigation of potential ASC—EMF/RFR links. The evidence is sufficient to warrant new public exposure standards benchmarked to low-intensity (non-thermal) exposure levels now known to be biologically disruptive, and strong, interim precautionary practices are advocated." (understreget her)

Wiart et al (2008)[49] konstaterede, baseret på modeller af hoveder, at børns hoveder absorberede omkring 2 gange så meget stråling som voksne, jf. p. 3693:

"...The comparisons have also shown that the maximum SAR in 1 g of peripheral brain tissues of child models aged between 8 and 15 is comparable to the maximum SAR in 1 g of peripheral brain tissues of adult models while it is about two times higher for child models aged between 5 and 8. This is certainly due to the smaller thicknesses of pinna, skin and skull. … The results obtained in this study need to be confirmed since they have been derived from data sets of limited size. Nevertheless these results are comparable to those obtained in other studies involving several phantoms (Beard et al 2006, Kainz et al 2005). ..."

Hardell et al (2011), *"Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects"* [50], p. 1465 (resumé) fandt:

"...an increased risk was found for glioma and use of mobile or cordless phone. The risk increased with latency time and cumulative use in hours and was highest in subjects with first use before the age of 20."

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49 Offentligt i det videnskabelige tidsskrift "Physics in Medicine & Biology" 2008, vol. 53, nr. 13, pp. 3681 – 3695.
50 Offentligt i det videnskabelige tidsskrift "International Journal of Oncology" 2011, vol. 38, nr. 5, pp. 1465 – 1474.

Fra Miller et al november 2018, p. 676 – 677:

"In a population-based case-control study of children Li et al. (2012) included 939 leukemia and 394 brain neoplasm[51] *cases newly diagnosed between 2003 and 2007, aged 15 years or less.*

…

They reported that a higher than median averaged APD[52] *was significantly associated with an increased Adjusted Odds Ratio (AOR) for all neoplasms (1.13; 1.01–1.28), and for leukemia (1.23; 0.99–1.52), but not for all brain neoplasms (1.14, 0.83–1.55). They did not specifically analyze data on gliomas."*

Ibid., p. 681 (konklusioner):

"The precautionary principle should be applied now and suitable warning messages provided to adults and critically to children and their parents. Until technology has been devised that substantially lowers exposures, special efforts should be advanced to ensure that the exposures of children are limited to those deemed essential. Children should be encouraged to text to reduce their exposure to RFR, while every attempt should be made to reduce exposure to RFR in schools, as well as homes."

1.2.2.5. Delkonklusion.

Det fremgår klart og videnskabeligt veldokumenteret, at eksponering for radiofrekvent elektromagnetisk stråling (også under de p.t. i Danmark anvendte grænseværdier, jf. pkt. 2.1 nedenfor) kan være kræftfremkaldende, og i så henseende udgør en helbredsfare for mennesker, der kan udvikle sig livstruende.

Dertil kommer den af Pall 2018 opsummerede videnskabelige dokumentation for en række andre skader, inkl. nedsat fertilitet, spontane aborter, neurologiske/neuropsykiatriske effekter, m.v.

Endvidere må det lægges til grund, at børn er særligt sårbare, og adskillige undersøgelser peger på en mulig forbindelse mellem eksponering for radiofrekvent elektromagnetisk stråling og adfærdsvanskeligheder, autisme, forståelsesevner, m.v.

51 Neoplasmer er abnormale væv, som kan udvikle sig til svulster og i værste fald ondartede kræftsvulster.

52 Står for "Average Power Density", jf. ibid. p. 677.

1.2.3. Dyr.

Overordnet kan om dyr henvises til f.eks. Alfonso Balmoris gennemgang af den videnskabelige litteratur i *"Electromagnetic pollution from phone masts. Effects on wildlife"*[53], p. , konklusionerne (der vedrører både fugle, pattedyr og insekter):

"This literature review shows that pulsed telephony microwave radiation can produce effects especially on nervous, cardiovascular, immune and reproductive systems [111]:

- Damage to the nervous system by altering electroencephalogram, changes in neural response or changes of the blood–brain barrier.
- Disruption of circadian rhythms (sleep–wake) by interfering with the pineal gland and hormonal imbalances.
- Changes in heart rate and blood pressure.
- Impairment of health and immunity towards pathogens, weakness, exhaustion, deterioration of plumage and growth problems.
- Problems in building the nest or impaired fertility, number of eggs, embryonic development, hatching percentage and survival of chickens.
- Genetic and developmental problems: problems of locomotion, partial albinism and melanism or promotion of tumors.

In the light of current knowledge there is enough evidence of serious effects from this technology to wildlife. For this reason precautionary measures should be developed, alongside environmental impact assessments prior to installation, and a ban on installation of phone masts in protected natural areas and in places where endangered species are present. Surveys should take place to objectively assess the severity of effects."

1.2.3.1. Fugle.

Der findes en større mængde videnskabelige undersøgelser, som dokumenterer direkte skadevirkning eller risiko herfor på fugle (og følgelig også deres levesteder, hvis f.eks. en mast er placeret tilstrækkeligt nært).

I det følgende gennemgås en række heraf, med fokus på dokumentation for skadevirkninger eller risici:

Balmori (2005), *"Possible Effects of Electromagnetic Fields from Phone Masts on a Population of White Stork (Ciconia ciconia)"*[54], p. 109 og 113 – 114:

"Monitoring of a white stork population in Valladolid (Spain) in the vicinity of Cellular Phone Base Stations was carried out, with the objective of detecting possible effects.
…
Birds are especially sensitive to the magnetic fields [48]. The white stork (Ciconia ciconia) build their nests on pinnacles and other very high places with high electromagnetic contamination (exposed to the microwaves). Also, they usually live inside the urban environment, where the electromagnetic contamination is higher, and remain in the nest a lot of the time, for this reason the decrease on the brood can be a good biological indicator to detect the effects of these radiations. The results indicate a difference in total productivity but not in partial productivity between the near nests and those far from the antennae. This indicate the existence of nests without chicks, or the death of young in their first stages in the nests near cellsites (40% of nest without young, compared to 3.3% in nests further 300 m).
…
The faithfulness of the white stork to nest sites can increase the effects of the microwaves.

53 Offentligt i det videnskabelige tidsskrift *"Pathophysiology"*, 2009, vol. 16.
54 Offentligt i det videnskabelige tidsskrift "*Electromagnetic Biology and Medicine"*, 2005, vol. 24, pp. 109 – 119.

…
Other studies find a decrease of fertility, increase of deaths after the birth in rats and dystrophic changes in their reproductive organs [16]. A recent study shows a statistically significant high mortality rate of chicken embryos subjected to the radiation from a cellphone, compared to the control group [43]. ...”

Den hvide stork er optaget på fuglebeskyttelsesdirektivets ”bilag I” og hører således til de arter, for hvilke der skal træffes ”særlige beskyttelsesforanstaltninger”, jf. pkt. 2.3.2. nedenfor.

Balmori og Hallberg (2007), *”The Urban Decline of the House Sparrow (Passer domesticus): A Possible Link with Electromagnetic Radiation”*[55], p. 141 (resumé):

”During recent decades, there has been a marked decline of the house sparrow (Passer domesticus) population in the United Kingdom and in several western European countries. The aims of this study were to determine whether the population is also declining in Spain and to evaluate the hypothesis that electromagnetic radiation (microwaves) from phone antennae is correlated with the decline in the sparrow population.
Between October 2002 and May 2006, point transect sampling was performed at 30 points during 40 visits to Valladolid, Spain. At each point, we carried out counts of sparrows and measured the mean electric field strength (radiofrequencies and microwaves: 1MHz–3GHz range). Significant declines (P=0.0037) were observed in the mean bird density over time, and significantly low bird density was observed in areas with high electric field strength. The logarithmic regression of the mean bird density vs. field strength groups (considering field strength in 0.1V/m increments) was R = −0 87 P = 0 0001.
The results of this article support the hypothesis that electromagnetic signals are associated with the observed decline in the sparrow population. We conclude that electromagnetic pollution may be responsible, either by itself or in combination with other factors, for the observed decline of the species in European cities during recent years. The appearently strong dependence between bird density and field strength according to this work could be used for a more controlled study to test the hypothesis”

Uddybende i forhold til de fortsat anvendte grænseværdier, jf. pkt. 2.1. nedenfor, konstateredes p. 145 – 146:

”According to this calculation, no sparrows would be expected to be found in an area with field strength >4V/m. ... In monitored Area 14, Plaza de la Libertad, a picocell was installed at the beginning of January 2005 and removed at the end of March 2005. Between January and March 2005, the mean field strength was greater than 3V/m, and the number of sparrows decreased drastically (generally, the number of sparrows increases towards a midwinter peak). In April 2005, after the picocell was removed, the sparrows became abundant again.”

Disse elektriske feltstyrker (V/m) ligger under de af ICNIRP anbefalede og i Danmark anvendte grænseværdier, jf. pkt. 2.1. nedenfor.

Cucurachi et al (2012)[56], *”A review of the ecological effects of radiofrequency electromagnetic fields (RF-EMF)”*, p. 122:

”Balmori (2005) monitored the variation of a population of white storks (Ciconia ciconia) in the vicinity of a GSM base station i.e. 900–1800 MHz with 217 Hz modulation) in search of possible effects from the exposure. Total productivity within 200 m was on average 46% less than that found at a distance greater than 300 m from the emitting station. An analogous significant difference was found in the breeding success: in 40% more of the cases no new-born chicks were found in the nest.
…

55 Offentligt i det videnskabelige tidsskrift ”*Electromagnetic Biology and Medicine”*, 2007, vol. 26, pp. 141 – 151.
56 Offentligt i det videnskabelige tidsskrift ”Environment International”, 2013, vol. 51, pp. 116-140.

Amongst the more recent laboratory studies, evidence of an effect of RF-EMF on mortality and development of embryos was in all cases found at both high and low dosages. In all the five field studies found a significant effect of RF-EMF on breeding density, reproduction or species composition. Field observations give a closer representation of real-life exposure, thus RF-EMF, especially in the 900 MHz GSM band could be a certain factor influencing the ecology of birds."

Burlaka et al (2013), p. 223:

"In conclusion, the exposure of developing quail embryos in ovo to extremely low intensity RF-EMR of GSM 900MHz during at least one hundred and fifty-eight hours discontinuously leads to the significantly increased rates of superoxide and nitrogen oxide generation in embryo cells. This was accompanied by a significantly increased level of lipid peroxidation, a depression of key antioxidant enzymes activity,and significantly, 2–3-fold, increased level of oxidative damage of DNA in embryo cells." (understreget her)

Alfonso Balmori (2015), *"Anthropogenic radiofrequency electromagnetic fields as an emerging threat to wildlife orientation"*[57], p. 59:

Low-voltage electricity current-generated electromagnetic field can produce a significantly negative effect on the breeding success of birds (Ciconia ciconia) nesting directly on electricity lines (Vaitkuvienė and Dagys, 2014) and these same results have been found in nests exposed to radiofrequency radiation near phone masts (Balmori, 2005)."

Yakymenko et al (2015), p. 194:

"We could ascertain the signaling effects of moderate levels of free radicals from our experiments in quail embryos irradiated with the commercial cell phone. Thus, we were able to show that the prolonged exposures of embryos in ovo led to robust repression of their development (Tsybulin et al., 2013), which was concomitant with significant overproduction of superoxide radical and NO radical, increased rates of lipid peroxidation and oxidative damage of DNA (Burlaka et al., 2013; Tsybulin et al., 2012)." (understreget her)

Shende et al (2015), *"Electromagnetic Radiations: A Possible Impact on Population of House Sparrow (Passer Domesticus)"*[58], p. 45:

"By monthly monitoring in urban and rural area, it is found that the population of house sparrow is declining in the urban area, where cell phone towers are more as compared to the rural area in every season."

Et særligt fokusområde i den videnskabelige litteratur udgøres af undersøgelser af radiofrekvent elektromagnetisk strålings virkning på fugles biologisk determinerede muligheder for at orientere sig.

Fugle er – ligesom en række andre dyr, jf. pkt. 1.2.3.2. nedenfor – født med, hvad der kan beskrives som en art indbygget, magnetisk baseret kompas, som indebærer, at de kan finde vej under deres migration.

Der kan bl.a. henvises til Alfonso Balmori (2015), *"Anthropogenic radiofrequency electromagnetic fields as an emerging threat to wildlife orientation"*[59], p. 58 – 59:

"Radio frequency fields in the MHz range disrupt birds' orientation interfering directly with the primary processes of magnetoreception and therefore disable the avian compass as long as

57 Offentligt i det videnskabelige tidsskrift "Science of the Total Environment" 2015, pp. 58 – 60.
58 Offentligt i det videnskabelige tidsskrift "Engineering International", 2015, vol. 3, nr. 1, pp. 45 – 52.
59 Offentligt i det videnskabelige tidsskrift "Science of the Total Environment" 2015, pp. 58 – 60.

they are present (Wiltschko et al., 2014). Ritz et al. (2004 & 2009) reported the sensitivity for orientation of European robins (Erithacus rubecula) to radiofrequency magnetic fields. The orientation of migratory birds is disrupted when very weak high-frequency fields (broadband field of 0.1–10 MHz of 85 nT or a 1.315 MHz field of 480 nT) are added to the static geomagnetic field of 46.000 nT (Thalau et al., 2006). It was convincingly demonstrated that robins are unable to use their magnetic compass in the presence of urban electromagnetic radiofrequency noise in the frequency range of 2 kHz–5 MHz (Engels et al., 2014). Therefore, electrosmog scrambles birds' magnetic sense and this finding could inform policies written to protect the habitats of endangered species.(understreget her)

Balmori (2005), *"Possible Effects of Electromagnetic Fields from Phone Masts on a Population of White Stork (Ciconia ciconia)"*[60], p. 115:

"... The perception to the terrestrial magnetic field can be altered by the electromagnetic radiation from the antennae. The reports of carrier pigeons losing direction in the vicinity of cellsites are numerous, and more investigation is necessary. ..."

Det EU-baserede forskningsprojekt EKLIPSE udgav v/ Malkemper et al i 2018 en rapport med titlen *"The impacts of artificial Electromagnetic Radiation on wildlife (flora and fauna). Current knowledge overview: a background document to the web conference",* hvoraf bl.a. fremgår s. 15:

"...It is established that the magnetic compass of migratory birds can be disrupted by the weak RF background in larger cities (nT-intensities) but it is currently unclear which exact frequencies are most effective. ..."

Ang. denne effekt henvises endvidere til Engels et al (2014), *"Anthropogenic electromagnetic noise disrupts magnetic compass orientation in a migratory bird"*[61], p. 353 (resumé):

"...Here we show that migratory birds are unable to use their magnetic compass in the presence of urban electromagnetic noise. When European robins, Erithacus rubecula, were exposed to the background electromagnetic noise present in unscreened wooden huts at the University of Oldenburg campus, they could not orient using their magnetic compass. Their magnetic orientation capabilities reappeared in electrically grounded, aluminium-screened huts, which attenuated electromagnetic noise in the frequency range from 50 kHz to 5 MHz by approximately two orders of magnitude. When the grounding was removed or when broadband electromagnetic noise was deliberately generated inside the screened and grounded huts, the birds again lost their magnetic orientation capabilities. The disruptive effect of radiofrequency electromagnetic fields is not confined to a narrow frequency band and birds tested far from sources of electromagnetic noise required no screening to orient with their magnetic compass. These fully double-blinded tests document a reproducible effect of anthropogenic electromagnetic noise on the behaviour of an intact vertebrate." (understreget her)

1.2.3.1.1. Delkonklusion.

Ligesom tilfældet er i forhold til helbredsskader og risici herfor på mennesker, forekommer det særdeles videnskabeligt veldokumenteret, at radiofrekvent elektromagnetisk stråling, også den som holder sig inden for de af myndighederne fastlagte grænseværdier, henholdsvis er og kan være helbredsskadeligt for fugle og (in extenso) deres levesteder.

Dertil kommer det særlige forhold ved fugle i forhold til mennesker, at deres evner til at orientere sig til dels er baseret på interaktion med jordens naturligt forekommende magnetfelter. Radiofrekvent elektromagnetisk strålings virkning på fugles biologisk determinerede muligheder for at orientere sig kan være ødelæggende for bevarelsen af arten,

60 Offentligt i det videnskabelige tidsskrift "*Electromagnetic Biology and Medicine"*, 2005, vol. 24, pp. 109 – 119.
61 Offentligt i det videnskabelige tidsskrift "Nature" 2014, nr. 509, pp. 353 – 356.

herunder i de dertil særligt udpegede beskyttede yngleområder. Særligt vedr. dette emne skal dog fremhæves, at dette for indeværende ikke synes at vedrøre 5G-frekvenser, m.v., men det kunne være tilfældet. Studier herom dokumenterer p.t. så vidt ses kun, at fugles biologisk determinerede orienteringssans påvirkes negativt af radiofrekvent elektromagnetisk stråling.

1.2.3.2. Andre dyr.

For så vidt angår insekter kan bl.a. henvises til Alfonso Balmori (2015), *"Anthropogenic radiofrequency electromagnetic fields as an emerging threat to wildlife orientation"*[62], p. 59:

"As with birds, radio frequency magnetic fields disrupt magnetoreception in insects. The geomagnetic field reception in American cockroach is sensitive to weak radio frequency field causing a disruptive effect (Vacha et al., 2009), so these authors suggest that electromagnetic smog will have to be taken more seriously in animal magnetoreception experiments. In an experimentally-generated electromagnetic field of about 1 V/m with a realistic (and even lower) power intensity similar to those surrounding communication masts, the results and observations suggest that GSM (Global System for Mobile communications) 900 MHz radiation might have a severe impact on the nerve cells of exposed ants, especially affecting the visual and olfactory memory, causing the loss of their ability to use visual cues and suggesting that electromagnetic radiation may have an impact on the orientation behaviour and navigation of animals that use magnetic fields to find their way (Cammaerts et al., 2012, 2014). Honeybees are sensitive to pulsed electromagnetic fields generated by mobile phones and observable changes in the bee behaviour could be one explanation for the loss of colonies (Favre, 2011). Magnetoreception system in Monarch butterfly orientation (Guerra et al., 2014) may be also suffering interference with anthropogenic radio frequency magnetic fields and this, together with other factors (Brower et al., 2012), may be a cause of their population decline." (understreget her)

Tilsvarende i Cucurachi et al (2012)[63], p. 116:

"Information was collected from 113 studies from original peer-reviewed publications or from relevant existing reviews… The majority of the studies were conducted in a laboratory setting on birds (embryos or eggs), small rodents and plants. In 65% of the studies, ecological effects of RF-EMF (50% of the animal studies and about 75% of the plant studies) were found both at high as well as at low dosages. ..."

Ibid., p. 122 – 123:

"It has been demonstrated that insects can sense magnetic fields as a means for navigation and orientation (Abraçado et al., 2005; Kirschvink et al., 2001; Liedvogel and Mouritsen, 2010; Wajnberg et al., 2010; Winklhofer, 2010). Magneto-reception has been associated with the use of ferromagnetic iron oxide particles embedded in tissue or through pairs of molecules with unpaired electrons (known as radical pairs) that are associated with a light sensitive photoreceptor (Ritz et al., 2002; Knight, 2009; Vácha et al. 2009). The exposure to RF-EMF might disrupt this magneto-reception mechanism, which could in turn affect the survival of insects. The most commonly studied species are the honey bee (A. mellifera) and the fruit fly (Drosophila melanogaster)."

Og p. 129:

"The studies analysing the effects of RF-EMF on fruit flies found in all cases a significant effect. Results of one study show an increased reproductive success after exposure. The remaining studies, which were conducted by the same research institute in Greece, found in all cases a significant depression of growth and reproduction at both 900 and 1800 MHz. Two studies on

62 Offentligt i det videnskabelige tidsskrift "Science of the Total Environment" 2015, pp. 58 – 60.
63 Offentligt i det videnskabelige tidsskrift "Environment International", 2013, vol. 51, pp. 116-140.

the American cockroach and a species of ant analysed the effects of exposure to RF-EMF on the magneto-reception and orientation of the insects. The behaviour of target systems was disrupted by the exposure to RF-EMF."

Samt p. 136 (konklusioner):

"...The effects of RF-EMF on different biological groups were investigated. With reference to the groups under investigations in the selected studies (i.e. birds, honeybees, mammals, plants, Drosophila and others) there is ecologically relevant evidence that the RF-EMF caused an effect in about 50% of the animal studies and about 90% of the plant studies. ..."

Kumar et al (2011), *"Exposure to cell phone radiations produces biochemical changes in worker honey bees"*[64], (resumé, resultater og diskussion):

"The present study was carried out to find the effect of cell phone radiations on various biomolecules in the adult workers of Apis mellifera L. The results of the treated adults were analyzed and compared with the control. Radiation from the cell phone influences honey bees' behavior and physiology. There was reduced motor activity of the worker bees on the comb initially, followed by en masse migration and movement toward "talk mode" cell phone. The initial quiet period was characterized by rise in concentration of biomolecules including proteins, carbohydrates and lipids, perhaps due to stimulation of body mechanism to fight the stressful condition created by the radiations.
At later stages of exposure, there was a slight decline in the concentration of biomolecules probably because the body had adapted to the stimulus.
…
Very little work has been done on biochemical, metabolic and physiological influences of cell phone radiations pertaining to health risk in man.[8] Therefore, the present investigations on the influence of cell phone radiations on some biochemical and physiological aspects of honeybee biology were undertaken. That the behavior of honeybee is altered to some extent by high or low energy fields or electromagnetic radiations has been known for quite some time.[9]
During the present investigation, it was observed that there was an increase in concentration of total carbohydrates in the bees exposed to cell phone radiation for 10 min as compared to unexposed or control bees. Increasing the exposure time to 20 min resulted in further increase in the concentration, while an exposure of 40 min had a reverse effect and there was a decline in carbohydrate concentration, though it was still higher as compared to control. Hemolymph glycogen and glucose content also showed the same trend, i.e., there was increase in content up to 20 min exposure after which there was a slight decline in the concentration which remained more than the control. Sharma[10] had also reported increase in glycogen and glucose levels in the exposed pupa of A. mellifera.
Lipids are the major energy reserves of insects. Certain lipid classes are structure components of membranes while others are raw materials for a variety of hormones and pheromones. Estimation of total lipids and cholesterol during the present study showed that the trend was similar to that of carbohydrates. After an initial increase in concentration at the 10 and 20 min exposure period, a decline was observed in the concentration of total lipids and cholesterol at 40 min exposure.
It was interesting to note that during the present study as the exposure time increased, it appeared that the bees having assessed the source of the disturbance decided to move and a large scale movement of the workers toward the talk-mode (not toward the listening mobile) was observed. Also, the bees became slightly aggressive and started beating their wings in agitation. This mobility of the bees could be responsible for increase utilization of energy sources and consequent decrease in concentration of carbohydrates and lipids in the 40 min exposed sample." (understreget her)

64 Offentligt i det videnskabelige tidsskrift "Toxicology International", 2011, vol. 18, nr. 1, pp. 70 – 72.

Margaritis et al (2014), *"Drosophila oogenesis as a bio-marker responding to EMF sources"*[65], p. 165 (resumé):

"A total of 280 different experiments were performed using newly emerged flies exposed for short time daily for 3–7 d to various EMF sources including: GSM 900/1800 MHz mobile phone, 1880–1900 MHz DECT wireless base, DECT wireless handset, mobile phone-DECT handset combination, 2.44 GHz wireless network (Wi-Fi), 2.44 GHz blue tooth, 92.8 MHz FM generator, 27.15 MHz baby monitor, 900 MHz CW RF generator and microwave oven's 2.44 GHz RF and magnetic field components.
…
All EMF sources used created statistically significant effects regarding fecundity and cell death-apoptosis induction, even at very low intensity levels (0.3 V/m blue tooth radiation), well below ICNIRP's guidelines, suggesting that Drosophila oogenesis system is suitable to be used as a biomarker for exploring potential EMF bioactivity. Also, there is no linear cumulative effect when increasing the duration of exposure or using one EMF source after the other (i.e. mobile phone and DECT handset) at the specific conditions used. ..."

Studiet blev udført på bananfluer, og det blev på baggrund af fundene anbefalet, at dette insekt fremover anvendes som biologisk markør ved undersøgelser af effekter af radiofrekvent elektromagnetisk stråling.

Som det fremgår, opstod der celledød endog ved meget lave intensiteter af stråling, helt ned til 0,3 V/m fra Blue Tooth-produkter.

Undersøgelsen viser, at udstyr som ligger inden for de af ICNIRP anbefalede grænseværdier (f.eks. 61 V/m for udstyr på med et frekvensområde på 2 – 300 GHz), jf. også pkt. 2.1 nedenfor, må forventes at være stærkt skadeligt for insekter.

Visse insekter er omfattet af EU's habitatdirektivbeskyttelse, jf. pkt. 2.3.3. nedenfor.

Dertil kommer, at fugle, der lever af insekter, ligeledes vil få ødelagt deres levesteder. For indholdet EU's fuglebeskyttelsesdirektiv henvises til pkt. 2.3.2. nedenfor.

Cammaerts og Johansson (2014), *"Ants can be used as bio-indicators to reveal biological effects of electromagnetic waves from some wireless apparatus"*[66], p. 286, pkt. 3:

"All radiating sources tested in this study on the ants demonstrated clear and statistically significant effects. It was already known that a mobile phone in standby mode affects living organisms (e.g. see Cammaerts et al.,2011; Favre, 2011; Panagopoulos et al., 2004; Sharma and Kumar, 2010). In this study, we showed that a common mobile phone has an effect while in standby mode and even in off-condition. Of course, when activated, the effect of a mobile phone is stronger. Without its battery, such a phone has no longer an effect. Our ants demonstrated that a modern smartphone and even more so a DECT phone do affect living organisms. Furthermore, the electromagnetic waves generated by a WiFi router impact our ants and such an effect increases during the course of the exposure time. Persons working in rooms provided with wireless equipment should note this result. A modern personal computer also generates electromagnetic waves. This is due to the PC WiFi function, which is automatically activated. Based on these results, we advice users to deactivate the WiFi function of their PC as long as they do not use it. This can also be deduced from the study

65 Offentligt i det videnskabelige tidsskrift "Electromagnetic Biology and Medicine", 2014, vol 33, nr. 3, pp. 165 – 189.

66 Offentligt i det videnskabelige tidsskrift "Electromagnetic Biology and Medicine", 2014, vol 33, nr. 4, pp. 282 – 288.

related in http://bigbrouser.blog.lemonde.fr/2011/12/01/microonde-le-wi-fi-tueur-de-spermatozoı¨des/." (understreget her)

Særligt for så vidt angår bestøvere skal henvises til Lázaro et al (2016), *"Electromagnetic radiation of mobile telecommunication antennas affects the abundance and composition of wild pollinators"*[67], p. 322 (konklusion):

"Electromagnetic radiation from telecommunication antennas affected the abundance and composition of wild pollinators in natural habitats....Pollinators and their host plants constitute pollination networks. Although the architecture of these mutualistic networks can increase the capacity of pollinator populations to persist under harsh conditions, once a tipping point in human-induced environmental change is reached, pollinator populations may collapse simultaneously (Lever et al. 2014). Therefore, these changes in the composition of pollinator communities associated with electromagnetic smog may have important ecological and economic impacts on the pollination service that could significantly affect the maintenance of wild plant diversity, crop production and human welfare."

Studiet viser således en sammenhæng mellem stråling fra mobilmaster og antallet af (flyvende) insekter.

Sammensætningen af bestøvere må anses for et vigtigt økologisk element og vigtig økonomisk parameter for produktion af afgrøder, menneskets velfærd samt for biodiversiteten generelt.

Vilic et al (2017), *"Effects of short-term exposure to mobile phone radiofrequency (900 MHz) on the oxidative response and genotoxicity in honey bee larvae"*[68], p. 430 (resumé):

"Exposure of different animal species to radiofrequency electromagnetic fields (RF-EMF) could cause various biological effects such as oxidative stress, genotoxic effects and dysfunction of the immune system. However, there are a lack of results on oxidative stress response and genotoxicity in the honey bee (Apis mellifera) after exposure to RF-EMF. This study was performed to investigate the effects of exposure to RF-EMF on the activity of catalase, superoxide dismutase, glutathione S-transferase, lipid peroxidation level and DNA damage in honey bee larvae. Honey bee larvae were exposed to RF-EMF at 900 Mhz and field levels of 10, 23, 41 and 120 V m−1 for 2 h. At a field level of 23 V m−1 the effect of 80% AM 1 kHz sinusoidal and 217 Hz modulation was investigated as well. Catalase activity and the lipid peroxidation level decreased significantly in the honey bee larvae exposed to the unmodulated field at 10 V m−1 compared to the control. Superoxide dismutase and glutathione S-transferase activity in the honey bee larvae exposed to unmodulated fields were not statistically different compared to the control. DNA damage increased significantly in honey bee larvae exposed to modulated (80% AM 1 kHz sinus) field at 23 V m−1 compared to the control and all other exposure groups. These results suggest that RF-EMF effects in honey bee larvae appeared only after exposure to a certain EMF conditions. The increase of the field level did not cause a linear dose-response in any of the measured parameters. Modulated RF-EMF produced more negative effects than the corresponding unmodulated field. Although honey bees in nature would not be exposed to such high field levels as used in our experiments, our results show the need for further intensive research in all stages of honey bee development." (understreget her)

Ibid., p. 437 (konklusion):

"In conclusion, the results of our study showed that effects of RF-EMF at 900 MHz in honey

67 Offentligt i det videnskabelige tidsskrift "Journal of Insect Conservation", 2016, vol. 20, nr. 2, pp. 315 – 324.
68 Offentligt i det videnskabelige tidsskrift "Journal of Apicultural Research", 2017, vol. 56, nr. 4, pp. 430 – 438.

bee larvae appeared only after exposure to the certain EMF conditions. RF-EMF modulated at 1 kHz showed an increase of DNA damage, while unmodulated RF-EMF produced alteration in catalase activity and lipid peroxidation at the lowest field level of 10 V m−1. Evidently, the increase of the field level did not cause a linear dose-response relationship in any of the measured parameters. Although honey bees in nature would not be exposed to such high field levels as used in our experiments, our results show the need for further intensive research in all stages of honey bee development, as well as the intensive research on the possible existence of a "window" effect under natural conditions during the annual cycling of bees."

Thielens et al (2018), *"Exposure of Insects to Radio-Frequency Electromagnetic Fields from 2 to 120 GHz"*, p. 9 (konklusion, manuskriptudgave):

"The insects show a maximum in absorbed radio frequency power at wavelengths that are comparable to their body size. They show a general increase in absorbed radio-frequency power above 6 GHz (until the frequencies where the wavelengths are comparable to their body size), which indicates that if the used power densities do not decrease, but shift (partly) to higher frequencies, the absorption in the studied insects will increase as well. A shift of 10% of the incident power density to frequencies above 6 GHz would lead to an increase in absorbed power between 3–370%. This could lead to changes in insect behaviour, physiology, and morphology over time due to an increase in body temperatures, from dielectric heating. The studied insects that are smaller than 1 cm show a peak in absorption at frequencies (above 6 GHz), which are currently not often used for telecommunication, but are planned to be used in the next generation of wireless telecommunication systems. At frequencies above the peak frequency (smaller wavelengths) the absorbed power decreases slightly."

Som det fremgår af de sidste, understregne linjer, vedrører denne undersøgelse tillige de højere frekvenser over 6 GHz, som vil blive taget i anvendelse ved 5G.

Studier ang. effekt af radiofrekvent elektromagnetisk strålingpå flagermus[69] er gennemført i bl.a. Nicholls og Racey (2009), *"The Aversive Effect of Electromagnetic Radiation on Foraging Bats—A Possible Means of Discouraging Bats from Approaching Wind Turbines",* hvori der bl.a. findes som følger, jf. p. 1 (resumé):

"Large numbers of bats are killed by collisions with wind turbines and there is at present no accepted method of reducing or preventing this mortality. Following our demonstration that bat activity is reduced in the vicinity of large air traffic control and weather radars, we tested the hypothesis that an electromagnetic signal from a small portable radar can act as a deterrent to foraging bats. From June to September 2007 bat activity was compared at 20 foraging sites in northeast Scotland during experimental trials (radar switched on) and control trials (no radar signal). Starting 45 minutes after sunset, bat activity was recorded for a period of 30 minutes during each trial and the order of trials were alternated between nights. From July to September 2008 aerial insects at 16 of these sites were sampled using two miniature light-suction traps. At each site one of the traps was exposed to a radar signal and the other functioned as a control. Bat activity and foraging effort per unit time were significantly reduced during experimental trials when the radar antenna was fixed to produce a unidirectional signal therefore maximising exposure of foraging bats to the radar beam. However, although bat activity was significantly reduced during such trials, the radar had no significant effect on the abundance of insects captured by the traps." (understreget her)

Tilsvarende i Balmori (2009), *"Electromagnetic pollution from phone masts. Effects on*

69 15 arter af flagermus er omfattet af habitatdirektivets bestemmelser om særlige beskyttelsesforanstaltninger, jf. pkt. 2.3.3. nedenfor.

wildlife"[70], p. 4:

"Electromagnetic radiation can exert an aversive behavioral response in bats. Bat activity is significantly reduced in habitats exposed to an electromagnetic field strength greater than 2 V/m [73]. During a study in a free-tailed bat colony (Tadarida teniotis) the number of bats decreased when several phone masts were placed 80m from the colony [74]."

Ang. padder kan bl.a. henvises til Alfonso Balmori (2010), *"Mobile Phone Mast Effects on Common Frog (Rana temporaria) Tadpoles: The City Turned into a Laboratory"*[71], p. 35:

"...Most prevailing hypotheses suggest that a field acts to directionally guide the growth and migration of some embryonic cells (Hotary and Robinson, 1992).
Strong magnetic fields (1.74–16.7T) disrupt cell division of exposed frog eggs (Xenopus laevis) (Denegre et al., 1998). Valles (2002) proposed a model to explain their influence.
Several studies on effects of electromagnetic fields on amphibians have been conducted in laboratories. When amphibian eggs and embryos of Ambystoma maculatum and Rana sylvatica were exposed to high magnetic fields (6.3 103 G), a brief treatment of early embryos produced several types of abnormalities, incuding microcephaly, retarded (abnormal) growth, edema, and scoliosis (Levengood, 1969).
Adult newts (Notophthalmus viridescens) exposed to a pulsed electromagnetic field (1 T and 0.15 V/m, approx.) for the first 30 days post forelimbs were amputated and produced more abnormalities in their skeletal patterns than the native limbs or the normal regenerates. Twelve percent exhibited unique abnormalities not observed in either the native or regenerate limb population. These forelimbs demonstrated one or more of the following gross defects: acheiria (lack of carpus and digits), aphalangia, or oligodactylia (loss of digits) as well as carpal bone and long bone (radius and ulna) abnormalities (Landesman and Douglas, 1990).
Exposed frog tadpoles (Rana temporaria) developed under electromagnetic field (50Hz, 260A/m) show an increase in mortality. Exposed tadpoles developed more slowly and less synchronously than control tadpoles and remained at the early stages for longer. Tadpoles developed allergies and EMF caused changes in their blood counts (Grefner et al., 1998). These results are consistent with the observations of this work.
Deformities and disappearance of amphibians and other organisms is part of the global biodiversity crisis (Blaustein and Johnson, 2003). Some authors consider that the electromagnetic pollution is destroying nature (Warnke, 2007; Firstenberg, 1997).
Balmori (2006) proposed that electromagnetic pollution (in the microwave and radiofrequency range) along with other environmental factors is a possible cause for decline and deformations of some wild amphibian populations exposed. The results of this experiment conducted in a real situation in the city of Valladolid (Spain) indicate that the tadpoles that live near such facilities, exposed to relatively low levels of environmental electromagnetic fields (1.8–3.5V/m) may suffer adverse effects (low coordination of movements, asynchronous growth, and high mortality), and this may be a cause (together with other environmental factors) of decline of amphibian populations." (understreget her)

Studiet er således udført på, hvad der måske er Danmarks mest almindelige frø, butsnudet frø. Dyret er optaget på bilag II til EU's habitatdirektiv, og er således omfattet af særlige beskyttelsesforanstaltninger, jf. pkt. 2.3.3. nedenfor.

Undersøgelsen påpeger, udover indikation for mutationer og en lang række sundhedsskader på padder, at den elektromagnetiske forurening fra selv relativt svage elektromagnetiske felter, miljøet udsættes for, er en mulig årsag (sammen med andre miljømæssige faktorer) til tabet af krybdyrspopulationer.

På mus kan bl.a. henvises til følgende, udover de i øvrigt i responsummet omtalte artikler:

Magras og Xenos (1997), *"RF Radiation–Induced Changes in the Prenatal Development of*

70 Offentligt i det videnskabelige tidsskrift "Pathopsyiology", 2009,
71 Offentligt i det videnskabelige tidsskrift "Electromagnetic Biology and Medicine", 2010, vol. 29, pp. 31 – 35.

Mice", p. 455:

"The possible effects of radiofrequency (RF) radiation on prenatal development has been investigated in mice. This study consisted of RF level measurements and in vivo experiments at several places around an "antenna park." At these locations RF power densities between 168 nW/cm2 and 1053 nW/cm2 were measured. Twelve pairs of mice, divided in two groups, were placed in locations of different power densities and were repeatedly mated five times. One hundred eighteen newborns were collected. They were measured, weighed, and examined macro- and microscopically. A progressive decrease in the number of newborns per dam was observed, which ended in irreversible infertility. The prenatal development of the newborns, however, evaluated by the crown-rump length, the body weight, and the number of the lumbar, sacral, and coccygeal vertebrae, was improved." (understreget her)

Mekanismen for den observerede sterilitet hos musene er forklaret således i Shahin et al (2017), *"Mobile phone (1800 MHz) radiation impairs female reproduction in mice, Mus musculus, through stress induced inhibition of ovarian anduterine activity"*, p. 41 (resumé):

"Present study investigated the long-term effects of mobile phone (1800 MHz) radiation in stand-by, dialing and receiving modes on the female reproductive function (ovarian and uterine histo-architecture, andsteroidogenesis) and stress responses (oxidative and nitrosative stress). We observed that mobile phone radiation induces significant elevation in ROS, NO, lipid peroxidation, total carbonyl content and serumcorticosterone coupled with significant decrease in antioxidant enzymes in hypothalamus, ovary anduterus of mice. Compared to control group, exposed mice exhibited reduced number of developing and mature follicles as well as corpus lutea. Significantly decreased serum levels of pituitary gonadotrophins(LH, FSH), sex steroids (E2 and P4) and expression of SF-1, StAR, P-450scc, 3 -HSD, 17 -HSD, cytochrome P-450 aromatase, ER- and ER- were observed in all the exposed groups of mice, compared to control. These findings suggest that mobile phone radiation induces oxidative and nitrosative stress, which affects the reproductive performance of female mice." (understreget her)

Videre hedder det ibid., p. 57:

"...Mobile phone radiation may result in ovarian and uterine dysfunction by increasing ROS and RNS production and disturbing antioxidant status. Oxidative and nitrosative stress created at the hypothalamus and peripheral level (ovary and uterus) as a consequence of long-term mobile phone exposure may severely reduce both steroidogenesis and folliculogenesis in the ovary as well as the structural and functional status of the uterus. These results led us to conclude that chronic exposure to long-term mobile phone radiation may severely affect the ovarian and uterine activity of female mice and thus may lead to infertility. ..."

1.2.3.2.1. Delkonklusion.

Det forekommer videnskabeligt veldokumenteret, at radiofrekvent elektromagnetisk stråling, også den som holder sig inden for de af myndighederne fastlagte grænseværdier, henholdsvis er og kan være helbredsskadeligt for insekter.

Dertil kommer det særlige forhold, at også insekternes evne til at orientere sig til dels er baseret på interaktion med naturligt forekommende felter i f.eks. de blomster, som skal bestøves. Virkningen af radiofrekvent elektromagnetisk stråling på insekters biologisk determinerede muligheder for at orientere sig kan være ødelæggende for bevarelsen af arten.

Dertil kommer, at insekters forsvinden fra et område kan have afgørende betydning for insektædende fugles muligheder for at overleve som arter.

1.2.4. Yderligere om leveområder samt planter.

En del af den i pkt. 1.2.3. ovenfor omtalte forskning omhandler leveområder (habitatområder), idet den angår undersøgelser af strålings påvirkning af dyr i de områder, hvor de har deres reder, jagtområder, osv., eller belyser, hvad effekten er i også disse områder.

Dertil kan føjes videnskabelige undersøgelser foretaget af radiofrekvent elektromagnetisk stråling på planter, herunder træer:

Magone (1996), *"The effect of electromagnetic radiation from the Skrunda Radio Location Station on Spirodela polyrhiza (L.) Schleiden cultures"*[72], p. 75 (resumé):

"The effect of electromagnetic radiation from the Skrunda Radio Location Station was studied on the vegetative growth and morphology of the duckweed Spirodela polyrhiza (L.) Schleiden plant in the next generation. The impact of plant development stage and length of the exposure period were examined. The effect of short-term (5-day) exposures of Spirodela cultures depended on the stage of development at the time of exposure. Generally, the vegetative reproduction rate was accelerated in the first 20 days after the end of exposure. Exposure of plants just beginning formation lowered the vegetative growth rate. Eighty-eight-hour exposure caused the appearance of some abnormal individuals after 30 days of growth. At 55 days, various morphological and developmental abnormalities appeared in 6–10 daughter plants from 10 exposed mother plants, compared with 0.1 plants per 10 in the control condition. Plants developed completely to daughter fronds under exposure from the electromagnetic field had a shorter life-span (67 days compared to 87 days in the control) and fewer subsequent daughters (total eight compared to 10 in the control group)." (understreget her)

Undersøgelsen vedrørte andemad.

Katie Haggerty (2010), *"Adverse Influence of Radio Frequency Background on Trembling Aspen Seedlings: Preliminary Observations"*[73], p. :

"The results of this preliminary experiment indicate that the RF background may be adversely affecting leaf and shoot growth and inhibiting fall production of anthocyanins associated with leaf senescence in trembling aspen seedlings. These effects suggest that exposure to the RF background may be an underlying factor in the recent rapid decline of aspen populations. Further studies are underway to test this hypothesis in a more rigorous way."

Undersøgelsen vedrørte poppeltræer.

Waldman et al (2016), *"Radiofrequency radiation injures trees around mobile phone base stations"*, p. 554 – 555 (resumé):

"...detailed long-term (2006–2015) field monitoring study was performed in the cities of Bamberg and Hallstadt (Germany). During monitoring, observations and photographic recordings of unusual or unexplainable tree damage were taken, alongside the measurement of electromagnetic radiation. In 2015 measurements of RF-EMF (Radiofrequency Electromagnetic Fields) were carried out. A polygon spanning both cities was chosen as the study site, where 144 measurements of the radiofrequency of electromagnetic fields were taken at a height of 1.5 m in streets and parks at different locations.

72 Offentligt i det videnskabelige tidsskrift "Science of The Total Environment", 1996, vol. 180, nr. 1, pp. 75 – 80.
73 Offentligt i det videnskabelige tidsskrift "International Journal of Forestry Research" 2010, Article ID 836278.

...
The measurements of all trees revealed significant differences between the damaged side facing a phone mast and the opposite side, as well as differences between the exposed side of damaged trees and all other groups of trees in both sides. Thus, we found that side differences in measured values of power flux density corresponded to side differences in damage. The 30 selected trees in low radiation areas (no visual contact to any phonemast and power flux density under 50 μW/m2) showed no damage. Statistical analysis demonstrated that electromagnetic radiation from mobile phone masts is harmful for trees. These results are consistent with the fact that damage afflicted on trees by mobile phone towers usually start on one side, extending to the whole tree over time." (understreget her)

Malka Halgamuge (2017), *"Review: Weak radiofrequency radiation exposure from mobile phone radiation on plants"*[74], p. 213 (resumé):

"Subject and methods: In this study, we performed an analysis of the data extracted from the 45 peer-reviewed scientific publications (1996–2016) describing 169 experimental observations to detect the physiological and morphological changes in plants due to the non-thermal RF-EMF effects from mobile phone radiation. Twenty-nine different species of plants were considered in this work. Results: Our analysis demonstrates that the data from a substantial amount of the studies on RF-EMFs from mobile phones show physiological and/or morphological effects (89.9%, p < 0.001). Additionally, our analysis of the results from these reported studies demonstrates that the maize, roselle, pea, fenugreek, duckweeds, tomato, onions and mungbean plants seem to be very sensitive to RF-EMFs. Our findings also suggest that plants seem to be more responsive to certain frequencies, especially the frequencies between (i) 800 and 1500 MHz (p < 0.0001), (ii) 1500 and 2400 MHz (p < 0.0001) and (iii) 3500[75] *and 8000 MHz (p = 0.0161)."* (understreget her)

1.2.4.1. Delkonklusion.

Det forekommer videnskabeligt veldokumenteret, at radiofrekvent elektromagnetisk stråling, også den som holder sig inden for de af myndighederne fastlagte grænseværdier, henholdsvis er og kan være skadelig for planter.

Dertil kommer, at planters forsvinden fra et område kan have afgørende betydning for fugles og insekters muligheder for at overleve som arter.

74 Offentligt i det videnskabelige tidsskrift "ELECTROMAGNETIC BIOLOGY AND MEDICINE", 2017, vol. 36, nr. 2, pp. 213 – 235.

75 3,5 GHz er blandt de frekvenser, der ifølge Energistyrelsens handlingsplan af februar 2019 for 5G er afsat til dette system, jf. https://ens.dk/sites/ens.dk/files/Tele/5g-handlingsplan_for_danmark.pdf, s. 10.

1.3. Overordnet delkonklusion.

Det ovenfor gennemgåede forskningsmateriale dokumenterer efter min opfattelse en klar og bastant underbygget årsagssammenhæng imellem udsættelse af mennesker og dyr for radiofrekvent elektromagnetisk stråling på den ene side og en række skadevirkninger samt mulige skadevirkninger på begge grupper, herunder livstruende.

Der foreligger tillige en velunderbygget årsagssammenhæng for så vidt angår skader på planter.

Dette gør sig også gældende under de p.t. fastsatte grænseværdier, jf. også pkt. 2.1. nedenfor.

2. Jus.

2.1. De i Danmark anvendte grænseværdier for eksponering for radiofrekvent elektromagnetisk stråling.

Sundhedsstyrelsen anvender grænseværdier som anbefalet i 1998[76] (gentaget i 2009[77]) af organisationen ICNIRP (International Commission on Non-Ionizing Radiation Protection). Tillige opereres med en SAR-grænseværdi på 2 W/kg for producenter af trådløst udstyr. Disse grænseværdier vil ligeledes blive anvendt i forhold til 5G[78]. En særlig grænseværdi for fuldkropsbestråling er på gennemsnitligt 0,08 W/kg.

Grænseværdierne er baseret på termisk opvarmning o.l. kortsigtede og umiddelbare effekter, medens en række af de i pkt. 1.2 ovennævnte forskningsresultater dokumenterer, at radiofrekvent elektromagnetisk stråling er skadeligt uden termisk opvarmning[79], medens andre sandsynliggør dette. Det hedder i ICNIRPs retningslinjer, p. 496:

*"**BASIS FOR LIMITING EXPOSURE***
These guidelines for limiting exposure have been developed following a thorough review of all published scientific literature. The criteria applied in the course of the review were designed to evaluate the credibility of the various reported findings (Repacholi and Stolwijk 1991; Repacholi and Cardis 1997); only established effects were used as the basis for the proposed exposure restrictions. Induction of cancer from long-term EMF exposure was not considered to be established, and so these guidelines are based on short-term, immediate health effects such as stimulation of peripheral nerves and muscles, shocks and burns caused by touching conducting objects, and elevated tissue temperatures resulting from absorption of energy during exposure to EMF. In the case of potential long-term effects of exposure, such as an increased risk of cancer, ICNIRP concluded that available data are insufficient to provide a basis for setting exposure restrictions, although epidemiological research has provided suggestive, but unconvincing, evidence of an association between possible carcinogenic effects and exposure at levels of 50/60 Hz magnetic flux densities substantially lower than those recommended in these guidelines.
..." (understreget her)

Sundhedsstyrelsen anfører nærmere om 5G på sin hjemmeside bl.a.:

"...Helt overordnet er det Sundhedsstyrelsens vurdering, at der ikke er grund til at være bekymret for, at der skulle være en sundhedsrisiko forbundet med 5G. Målinger viser, at den samlede stråling fra mobiltelefoner, wifi og andet apparatur, som i dag udsender ikke-ioniserende stråling, er svag, og ligger langt under grænseværdierne for, hvad der er sundhedsskadeligt. Baseret på den tilgængelige viden har vi ingen grund til at tro, at 5G vil ændre på det.
I lovgivningen om radioudstyr er der fastsat regler om, at radioudstyr skal være konstrueret, så det sikrer menneskers sundhed. Det betyder, at antenner mv. til 5G skal følge de samme fælleseuropæiske grænseværdier som alt andet nuværende udstyr til telekommunikation. ..."

Ved sammenholdelsen mellem den i pkt. 1.2. ovenfor omtalte forskning og de af Sundhedsstyrelsen anvendte grænseværdier er beregningerne i følgende skema lagt til grund[80]:

76 "ICNIRP guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 Ghz)", offentliggjort i det videnskabelige tidsskrift "Health Physics" (1998) nr. 74, pp. 494 – 522.

77 "ICNIRP statement on the 'guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 Ghz)'", offentliggjort i det videnskabelige tidsskrift "Health Physics" (2009) nr. 97, p. 257 – 258.

78 Jf. Energi-, Forsynings- og Klimaministerens svar af 1. april 2019 på spørgsmål 226 i samme folketingsudvalg samt Sundhedsstyrelsens hjemmeside: https://www.sst.dk/da/straalebeskyttelse/mobiltelefoni,-traadloese-netvaerk-med-mere/5g

79 Se f.eks. Philips et al (2009), p. 83, med referencer til to studier.

80 Kilden er Rådet for Helbredssikker Telekommunikation.

ICNIRP 1998 grænser

(frekvens) MHz	(feltstyrke) V/m	(effekttæthed) µW/m2	mW/m2	W/m2	
10	28,00	2.079.575,60	2079,58	2,08	→ 10 – 400MHz defineret som 28 V/m (ICNIRP har vist rundet op)
400	27,50	2.005.968,17	2005,97	2,01	
450	29,17	2.256.999,73	2257,00	2,26	
500	30,75	2.508.123,34	2508,12	2,51	
550	32,25	2.758.786,47	2758,79	2,76	
600	33,68	3.008.865,78	3008,87	3,01	
650	35,06	3.260.487,00	3260,49	3,26	
700	36,38	3.510.621,75	3510,62	3,51	
750	37,66	3.762.004,24	3762,00	3,76	
800	38,89	4.011.756,23	4011,76	4,01	
850	40,09	4.263.151,46	4263,15	4,26	
900	41,25	4.513.428,38	4513,43	4,51	
950	42,38	4.764.096,55	4764,10	4,76	
1000	43,48	5.014.616,45	5014,62	5,01	
1050	44,56	5.266.826,53	5266,83	5,27	I dette interval er grænsen frekvensafhængig Udregnes som: $1.375\,x10^{-3}\sqrt{frekvens}$
1100	45,60	5.515.543,77	5515,54	5,52	
1150	46,63	5.767.524,93	5767,52	5,77	
1200	47,63	6.017.551,46	6017,55	6,02	
1250	48,61	6.267.724,40	6267,72	6,27	
1300	49,58	6.520.361,80	6520,36	6,52	
1350	50,52	6.769.948,01	6769,95	6,77	
1400	51,45	7.021.492,04	7021,49	7,02	
1450	52,36	7.272.067,90	7272,07	7,27	
1500	53,25	7.521.385,94	7521,39	7,52	
1550	54,13	7.772.034,22	7772,03	7,77	
1600	55,00	8.023.872,68	8023,87	8,02	
1650	55,85	8.273.799,73	8273,80	8,27	
1700	56,69	8.524.551,99	8524,55	8,52	
1750	57,52	8.775.995,76	8776,00	8,78	
1800	58,34	9.027.998,94	9028,00	9,03	→ Her rammer vi 9 mio µW/m2 ved 1800 MHz som var den højeste GSM frekvens i anvendelse omkring 98'
1850	59,14	9.277.293,37	9277,29	9,28	
1900	59,93	9.526.803,45	9526,80	9,53	
1950	60,72	9.779.624,40	9779,62	9,78	
2000	61,49	10.029.231,03	10029,23	10,03	
300000	61,00	9.870.027,00	9870,03	9,87	→ 2 – 300GHz defineret som 61 V/m (ICNIRP har vist rundet ned)

En række af de i pkt. 1.2 – 1.4 ovennævnte forskningsresultater dokumenterer, at stråling under de pågældende grænseværdier kan være helbredsskadelig for mennesker og/eller dyr, bl.a.:

Balmori og Hallberg 2007, p. 145 – 146.
Blank og Goodman 2011, p. 413.
Jing et al 2012, p. 64.
REFLEX-studiet p. 109 og 223.
Yakymenko et al 2015, p. 186.
Lerchl et al 2015, p. 585.
Falcioni et al. 2018, p. 499.
Russell 2018, p. 485.
Neufeld og Kuster 2018, p. 711.

Dertil kommer, at en række studier er udført med kommercielt tilgængelige produkter inkl. følgende fra pkt. 1.2 – 1.4 ovenfor:

D'Silva et al 2017, p. 6.
Panagopoulos 2019, p. 55 (v.sp., nederst).

Pr. 1. september 2018 havde 244 forskere fra over 40 lande, som tilsammen havde offentliggjort over 2.000 forskningsartikler om elektromagnetiske felter, underskrevet en appel til FN og WHO, hvori de opfordrede til, at der etableres en bedre beskyttelse af menneskers (og dyrs) helbred i forhold til brugen af elektromagnetiske installationer såsom 5G.[81] Disse 244 forskere har i appellens afsnit med overskriften *"Inadequate non-ionizing EMF international guidelines"* anført som deres opfattelse, at:

"The various agencies setting safety standards have failed to impose sufficient guidelines to protect the general public, particularly children who are more vulnerable to the effects of EMF.
...
It is our opinion that, because the ICNIRP guidelines do not cover long-term exposure and low-intensity effects, they are insufficient to protect public health. "

De 244 forskeres *"...opfattelse..."* er spejlet i den i pkt. 1.2 ovenfor nævnte videnskabelige litteratur.

Pall 2018 konkluderede i sin gennemgang bl.a. følgende om ICNIRPs grænseværdier:

"Each of these reviews, typically cite from 5 to over 100 primary literature citations, each showing that non-thermal EMF exposures produce the effect under which they are listed. It follows from this, that there are not only 11 or more reviews documenting each of these effects, but there is also a massive primary literature documenting these effects as well. It follows from this that the ICNIRP, FCC and International Safety Guidelines, which are entirely based only on thermal effects are inadequate and there have been petitions and other statements of international groups of scientists expressing great concern about this. It follows that the ICNIRP, FCC and International safety guidelines are completely unscientific and cannot be relied upon to protect our safety." (understreget her)

Europarådet har i resolution 1815 af 2011, pkt. 8.1.2. anbefalet medlemsstaterne følgende:

"8.1.2. reconsider the scientific basis for the present standards on exposure to electromagnetic fields set by the International Commission on Non-Ionising Radiation Protection, which have serious limitations, and apply ALARA[82] principles, covering both thermal effects and the athermic or biological effects of electromagnetic emissions or radiation;"

81 Jf. https://emfscientist.org/index.php/emf-scientist-appeal
82 Står for "As Low As Reasonably Achievable".

2.2. Retsbeskyttelsen af mennesker (menneskerettigheder).

2.2.1. Den Europæiske Menneskerettighedskonvention (EMRK).

Konventionen blev inkorporeret direkte i dansk ret ved lov nr. 285 af 1992 og er således en del af "almindelig" national ret, som kan påberåbes direkte for de danske domstole.

Dertil kommer, at den ifølge Højesterets praksis anvendes på den måde, at andre lovbestemmelser "fortolkes i lyset af" konventionen og den dertilhørende praksis.

Det er et generelt fortolkningsprincip ved anvendelsen af bestemmelserne i konventionen, at de skal fortolkes således, at rettighederne indeholdt heri er praktisk anvendelige og effektive, jf. f.eks. Storkammerdom af 27. september 1995 i sagen McCann m.fl. mod Storbritannien, præmis 146:

"146. The Court's approach to the interpretation of Article 2 (art. 2) must be guided by the fact that the object and purpose of the Convention as an instrument for the protection of individual human beings requires that its provisions be interpreted and applied so as to make its safeguards practical and effective (see, inter alia, the Soering v. the United Kingdom judgment of 7 July 1989, Series A no. 161, p. 34, para. 87, and the Loizidou v. Turkey (Preliminary Objections) judgment of 23 March 1995, Series A no. 310, p. 27, para. 72)."

2.2.1.1. Art. 2 – retten til livet og statens positive forpligtelser.

EMRK art. 2 lyder:

*"**Artikel 2.** Ethvert menneskes ret til livet skal beskyttes ved lov. Ingen må forsætligt berøves livet undtagen ved fuldbyrdelse af en dødsdom, afsagt af en domstol i tilfælde, hvor der ved lov er fastsat dødsstraf for den pågældende forbrydelse.*
Stk. 2. Berøvelse af livet betragtes ikke som sket i modstrid med denne artikel, når den er en følge af magtanvendelse, der ikke gå ud over det absolut nødvendige:
a) for at forsvare nogen mod ulovlig vold;
b) for at iværksætte en lovlig anholdelse eller forhindre flugt
fra lovlig frihedsberøvelse;
c) for lovligt at undertrykke optøjer eller opstand."

Det er således alene stk. 1, 1. pkt. samt 2. pkt., 1. led, der har betydning for den retlige problemstilling i nærværende responsum.

Den Europæiske Menneskerettighedsdomstol (EMD) har i sin praksis fortolket bestemmelsen således, at den rummer en række materielle, positive forpligtelser (dvs.: handlepligter), bl.a. under visse betingelser at hindre, at mennesker dør som følge af forurening, der har været tilladt eller ikke hindret af staten.

Forpligtelserne i henhold til art. 2 skal – henset til vigtigheden af den rettighed, den skal beskytte – fortolkes strengt, jf. f.eks. Storkammerdom af 27. september 1995 i sagen McCann m.fl. mod Storbritannien, præmis 147:

"147. It must also be borne in mind that, as a provision (art. 2) which not only safeguards the right to life but sets out the circumstances when the deprivation of life may be justified, Article 2 (art. 2) ranks as one of the most fundamental provisions in the Convention - indeed one which, in peacetime, admits of no derogation under Article 15 (art. 15). Together with Article 3 (art. 15+3) of the Convention, it also enshrines one of the basic values of the democratic societies making up the Council of Europe (see the above-mentioned Soering judgment, p. 34, para. 88). As such, its provisions must be strictly construed."

I relation til nærværende responsums emne bør sagen Öneryildiz mod Tyrkiet, Storkammerdom af 30. november 2004, fremhæves[83].

Sagen drejede sig om en dødelig eksplosion på en losseplads, der kostede i alt 39 mennesker livet, inkl. flere af klagerens nære familiemedlemmer.

Det blev bl.a. statueret, at staten har en positiv forpligtelse til at opstille retlige og administrative rammer, som er designet til at medføre et effektivt værn imod livsfare.

Præmis 89 – 90 (med yderligere praksishenvisninger):

*"**(a) General principles applicable in the present case**(i) Principles relating to the prevention of infringements of the right to life as a result of dangerous activities: the substantive aspect of Article 2 of the Convention*

89. The positive obligation to take all appropriate steps to safeguard life for the purposes of Article 2 (see paragraph 71 above) entails above all a primary duty on the State to put in place a legislative and administrative framework designed to provide effective deterrence against threats to the right to life (see, for example, mutatis mutandis, Osman, cited above, p. 3159, § 115; Paul and Audrey Edwards, cited above, § 54; İlhan v. Turkey [GC], no. 22277/93, § 91, ECHR 2000-VII; Kılıç v. Turkey, no. 22492/93, § 62, ECHR 2000-III; and Mahmut Kaya v. Turkey, no. 22535/93, § 85, ECHR 2000-III).

90. This obligation indisputably applies in the particular context of dangerous activities, where, in addition, special emphasis must be placed on regulations geared to the special features of the activity in question, particularly with regard to the level of the potential risk to human lives. They must govern the licensing, setting up, operation, security and supervision of the activity and must make it compulsory for all those concerned to take practical measures to ensure the effective protection of citizens whose lives might be endangered by the inherent risks.

Among these preventive measures, particular emphasis should be placed on the public's right to information, as established in the case-law of the Convention institutions. The Grand Chamber agrees with the Chamber (see paragraph 84 of the Chamber judgment) that this right, which has already been recognised under Article 8 (see Guerra and Others, cited above, p. 228, § 60), may also, in principle, be relied on for the protection of the right to life, particularly as this interpretation is supported by current developments in European standards (see paragraph 62 above).

In any event, the relevant regulations must also provide for appropriate procedures, taking into account the technical aspects of the activity in question, for identifying shortcomings in the processes concerned and any errors committed by those responsible at different levels." (understreget her)

Der skal således være effektive hindringer imod trusler over for retten til livet, inkl. farlige aktiviteter, såsom den i sagen omhandlede drift af losseplads.

Når der er tale om farlige aktiviteter, skal der lægges særligt vægt på regler, som er egnede til de særlige forhold, som den pågældende aktivitet frembyder, og da især under hensyntagen til den potentielle fare for menneskeliv.

Det er således ikke et krav i art. 2s forstand, at der kan påvises en sikker skadevirkning, som kan være eller med sikkerhed er livstruende. Det er tilstrækkeligt, for at den pågældende adfærd falder ind under beskyttelsen af retten til livet, at der kan påvises en risiko for fare for menneskeliv.

Staten skal pålægge aktørerne, og herunder således dem, der måtte ønske at opføre den relevante infrastruktur, at der foretages praktiske foranstaltninger, som effektivt beskytter imod de fareelementer, som kan koste mennesker livet.

83 Der kan tillige henvises til f.eks. Kolyadenko m.fl. mod Rusland, præmis 157 – 161.

Henset til det foreliggende forskningsmateriale, jf. pkt. 1.2. ovenfor, er det min vurdering, at staten klart ikke på nuværende tidspunkt har efterkommet sine forpligtelser til at opstille relevante grænseværdier, endsige forbyde aktiviteter, som indebærer en klart dokumenteret risiko for fare for menneskeliv.

Art. 2 finder ikke kun anvendelse, hvor der er tale om en pludselig hændelse, som resulterer i dødsfald. Udsættelse for forurening over en længere periode er også omfattet.

EMD bringer bestemmelsen i anvendelse, uanset om klager er afgået ved døden eller "blot" er i alvorlig livsfare.[84]

Det er ikke afgørende, om det er en offentlig eller privat forurener.

Hvor det må lægges til grund, at det er denne forurening, der har medført dødsfaldet eller livsfaren, og hvor staten ikke har handlet over for en kendt fare (f.eks. fordi forureningen ikke har været i strid med gældende, national ret, herunder grænseværdier), vil det som udgangspunkt udgøre en krænkelse af retten til livet, jf. i det hele f.eks. dom af 24. juli 2014 i sagen Brincat m.fl. mod Malta, præmis 79 – 81 og 83 (med yderligere praksishenvisninger):

"79. The Court reiterates that Article 2 does not solely concern deaths resulting from the use of unjustified force by agents of the State but also, in the first sentence of its first paragraph, lays down a positive obligation on States to take appropriate steps to safeguard the lives of those within their jurisdiction (see, for example, L.C.B. v. the United Kingdom, 9 June 1998, § 36, Reports 1998-III, and Paul and Audrey Edwards, cited above, § 54).

80. This obligation is construed as applying in the context of any activity, whether public or not, in which the right to life may be at stake, and a fortiori in the case of industrial activities which by their very nature are dangerous, such as the operation of waste-collection sites (see Öneryıldız v. Turkey [GC], no. 48939/99, §71, ECHR 2004-XII) or nuclear testing (see L.C.B. cited above, § 36) or cases concerning toxic emissions from a fertiliser factory (see Guerra and Others v. Italy, 19 February 1998, §§ 60 and 62, Reports 1998-I, although in this case the Court found that it was not necessary to examine the issue under Article 2, it having been examined under Article 8).

81. The Court considers that the same obligations may apply in cases, such as the present one, dealing with exposure to asbestos at a workplace which was run by a public corporation owned and controlled by the Government.

82. The Court reiterates that it has applied Article 2 both where an individual has died (see, for example, Öneryıldız, cited above) and where there was a serious risk of an ensuing death, even if the applicant was alive at the time of the application. Examples include cases where the physical integrity of an applicant was threatened by the action of a third party (see Osman v. the United Kingdom, 28 October 1998, §§ 115-122, Reports 1998-VIII) or as a result of a natural catastrophe which left no doubt as to the existence of a threat to the applicants' physical integrity (see Budayeva and Others v. Russia, nos. 15339/02, 21166/02, 20058/02, 11673/02 and 15343/02, § 146, ECHR 2008 (extracts)). More particularly, the Court has repeatedly examined complaints under Article 2 from persons suffering from serious illnesses. Such cases include G.N. and Others v. Italy (no. 43134/05, 1 December 2009) in which the applicants suffered from the potentially life-threatening disease hepatitis C; L.C.B. v. the United Kingdom (cited above), where the applicant suffered from leukaemia diminishing her chances of survival, Hristozov and Others v. Bulgaria, nos. 47039/11 and 358/12, ECHR 2012

84 Udover Brincat m.fl. mod Malta kan f.eks. henvises til Vilnes m.fl. mod Norge, præmis 219: *"...the applicant was found to be the victim of conduct which by its very nature had put his life at risk, even though he survived. The Court found there that Article 2 was applicable and sees no reason for arriving at a different conclusion in the present case."*

(extracts), concerning applicants suffering from different types of terminal cancer; Karchen and Others v. France ((dec.), no. 5722/04, 4 March 2008) and Oyal v. Turkey (no. 4864/05, 23 March 2010), in which the applicants had been infected with the HIV virus, which endangered their life; Nitecki v. Poland ((dec.), no. 65653/01, 21 March 2002), in which the applicant suffered from amyotrophic lateral sclerosis; Gheorghe v. Romania ((dec.), no. 19215/04, 22 September 2005), in which the applicant suffered from haemophilia; and De Santis and Olanda v. Italy ((dec.), 35887/11, 9 July 2013) in which the applicant – who was severely disabled – suffered a cerebral haemorrhage as a consequence of an infection acquired in hospital.

83. The medical certification indicated that Mr Attard's death was likely to be a result of asbestos exposure; malignant mesothelioma is known to be a rare cancer associated with asbestos exposure. The Court observes that it has not been contested or denied that Mr Attard worked at Malta Drydocks for more than a decade (1959-1974), during which time he was repeatedly exposed to asbestos. Neither has it been shown that Mr Attard could have been contaminated elsewhere or that he was affected by other factors that could have led to the disease. In these circumstances, and given that Mr Attard has died as a result of his cancer, the Court considers that Article 2 is applicable to the complaint brought by the applicants in application no. 62338/11 relating to the death of the said Mr Attard." (understreget her)

2.2.1.1.1. Delkonklusion.

Set på baggrund af de i pkt. 1.2 ovenfor gennemgåede forskningsresultater er der efter min vurdering ikke nogen rimelig tvivl om, at 5G-systemet udgør en industriel aktivitet, som er farlig for mennesker.

Så længe de nuværende grænseværdier (som meddelt af Sundhedsstyrelsen, jf. pkt. 2.1 ovenfor) anvendes, må livstruende helbredstilstande forårsaget af radiofrekvent elektromagnetisk stråling ved iværksættelse af 5G-systemet ganske klart forventes, hvilket vil være i strid med den danske stats positive forpligtelser efter EMRK art. 2.

Da det må lægges til grund, at risikoen er velkendt af den danske stat, er det endvidere oplagt, at der i relation til 5G-systemet[85] vil skulle indtræde ansvar efter EMRK art. 2, senest når de livstruende helbredstilstande viser sig.

2.2.1.2. Art. 8 – retten til respekt for privat- og familieliv.

EMRK art. 8 lyder:

*"**Artikel 8.** Enhver har ret til respekt for sit privatliv og familieliv, sit hjem og sin korrespondance.*

Stk. 2. Ingen offentlig myndighed må gøre indgreb i udøvelsen af denne ret, medmindre det sker i overensstemmelse med loven og er nødvendigt i et demokratisk samfund af hensyn til den nationale sikkerhed, den offentlige tryghed eller landets økonomiske velfærd, for at forebygge uro eller forbrydelse, for at beskytte sundheden eller sædeligheden eller for at beskytte andres rettigheder og friheder."

Alvorlig miljøforurening kan påvirke individers velbefindende og forhindre dem i at udøve deres privat- og familieliv. En sådan tilstand vil udgøre et indgreb i borgernes rettigheder efter EMRK art. 8, jf. f.eks. Guerra m.fl. v. Italien, Storkammerdom af 19. februar 1998[86].

85 Det falder udenfor området for nærværende responsum at fremkomme med tilsvarende vurderinger ang. 2G, 3G og 4G, m.v.

86 Fordi Storkammeret fandt en krænkelse af art. 8, var det ikke nødvendigt at vurdere en klage over krænkelse af retten til livet, jf. EMRK art. 2, p.g.a. de samme kræftdødsfald.

Præmis 60:

"60. The Court reiterates that severe environmental pollution may affect individuals' well-being and prevent them from enjoying their homes in such a way as to affect their private and family life adversely (see, mutatis mutandis, the López Ostra judgment cited above, p. 54, § 51). In the instant case the applicants waited, right up until the production of fertilisers ceased in 1994, for essential information that would have enabled them to assess the risks they and their families might run if they continued to live at Manfredonia, a town particularly exposed to danger in the event of an accident at the factory.
The Court holds, therefore, that the respondent State did not fulfil its obligation to secure the applicants' right to respect for their private and family life, in breach of Article 8 of the Convention.

There has consequently been a violation of that provision."

De positive forpligtelser i så henseende overlapper i vidt omfang de ovenfor nævnte efter art. 2, jf. f.eks. dom af 20. marts 2008 i sagen Budayeva m.fl. mod Rusland, præmis 133 og dom af 28. februar 2012 i sagen Kolyadenko m.fl. mod Rusland, præmis 216.

Retten til beskyttelse af privat- og familielivet kan bringes i anvendelse, hvor en sygdomstilstand ikke har udviklet sig livsfarligt og heller ikke nødvendigvis gør det. I denne sammenhæng anvendes bestemmelsen af EMD som en slags "mindre i det mere" i forhold til art. 2, jf. ovenfor.

Dette blev f.eks. fremgangsmåden for alle klager undtagen én i dom af 24. juli 2014 i sagen Brincat m.fl. mod Malta, præmis 85:

"85. However, in the context of dangerous activities, the scope of the positive obligations under Article 2 of the Convention largely overlaps with that of those under Article 8 (see Öneryıldız, cited above, §§ 90 and 160). The latter provision has allowed complaints of this nature to be examined where the circumstances were not such as to engage Article 2, but clearly affected a person's family and private life under Article 8 (see López Ostra v. Spain, 9 December 1994, Series A no. 303-C and Guerra and Others, cited above). The Court therefore considers it appropriate to examine the complaints in respect of the remaining applicants under Article 8, which is applicable in the present case (see also Roche v. the United Kingdom [GC], no. 32555/96, §§ 155-156, ECHR 2005-X)."

Der skal foreligge en sygdomstilstand, som har en sådan karakter, at den vil udgøre et indgreb i vedkommendes privat- eller familieliv. Sygdomme som nødvendiggør f.eks. langvarige eller hyppige hospitalsindlæggelser, varige og indgribende funktionsnedsættelser (herunder lidelser såsom EHS, der indebærer overfølsomhed over for udstyr, der afgiver elektromagnetisk stråling, evt. også i meget små doser), markant nedsat fertilitet eller spontane aborter, m.v., kunne være egnede eksempler.

Hvor en begivenhed eller tilstand indtræffer, som gør indgreb i retten til privat- eller familielivet, vil EMD kunne forventes at påse, om f.eks. de tekniske forudsætninger var til stede for, at begivenheden eller tilstanden ikke indtraf, og om dette burde have været forudset af staten.

Finder EMD, at dette er tilfældet, vil det som udgangspunkt udgøre en krænkelse af statens positive forpligtelser efter art. 8. Der kan fra praksis henvises til dom af 28. februar 2012 i sagen Kolyadenko m.fl. mod Rusland, præmis 215 – 216 (sagen drejede sig om brud på et vandreservoir, som medførte livsfare og skader på menneskers hjem):

"215. The Court further notes that the Government seem to have argued, with reference to the findings of the domestic courts in the applicants' civil cases, that the alleged infringements of their rights under Article 8 and Article 1 of Protocol No. 1 were the result of a natural disaster, in the form of exceptionally heavy rain, which could not have been foreseen, and

could therefore not be imputed to the State. The Court cannot accept this argument. It reiterates in this connection that, being sensitive to the subsidiary nature of its role and cautious about taking on the role of a first-instance tribunal of fact, the Court nevertheless is not bound by the findings of domestic courts and may depart from them where this is rendered unavoidable by the circumstances of a particular case (see, for example, Matyar v. Turkey, no. 23423/94, § 108, 21 February 2002). In the present case, the Court has established in paragraphs 162-165 above that the flooding of 7 August 2001 occurred after the urgent large-scale evacuation of water from the Pionerskoye reservoir, the likelihood and potential consequences of which the authorities should have foreseen. The Court has furthermore established that the main reason for the flood, as confirmed by the expert reports, was the poor state of repair of the Pionerskaya river channel because of the authorities' manifest failure to take measures to keep it clear and in particular to make sure its throughput capacity was adequate in the event of the release of water from the Pionerskoye reservoir. The Court has concluded that this failure as well as the authorities' failure to apply town planning restrictions corresponding to the technical requirements of the exploitation of the reservoir put the lives of those living near it at risk (see paragraphs 168-180 and 185 above).

216. The Court has no doubt that the causal link established between the negligence attributable to the State and the endangering of the lives of those living in the vicinity of the Pionerskoye reservoir also applies to the damage caused to the applicants' homes and property by the flood. Similarly, the resulting infringement amounts not to "interference" but to the breach of a positive obligation, since the State officials and authorities failed to do everything in their power to protect the applicants' rights secured by Article 8 of the Convention and Article 1 of Protocol No. 1 (see Öneryıldız, cited above, § 135). Indeed, the positive obligation under Article 8 and Article 1 of Protocol No. 1 required the national authorities to take the same practical measures as those expected of them in the context of their positive obligation under Article 2 of the Convention (see, mutatis mutandis, Öneryıldız, cited above, § 136). Since it is clear that no such measures were taken, the Court concludes that the Russian authorities failed in their positive obligation to protect the applicants' homes and property.

217. There has, accordingly, been a violation of Article 8 of the Convention and Article 1 of Protocol No. 1 to the Convention in the present case."

EMD foretog ikke en udtrykkelig proportionalitetsafvejning i den pågældende sag, hvilket synes at være konsekvensen af, at staten intet havde foretaget sig, uanset den burde have forudset den skadegørende hændelse og kunne have handlet til afværgelse heraf.

2.2.1.2.1. Delkonklusion.

Der gælder i det væsentlige de samme positive forpligtelser efter art. 8 som art. 2 m.h.t. beskyttelse af mennesker over for forurening, jf. pkt. 2.2.1.1.1. Allerede derfor må iværksættelsen af 5G-netværket, ved brug af de nugældende grænseværdier, klart forventes at medføre sådanne forstyrrelser af borgeres privat- og familieliv p.g.a. sygdomme, at der tillige vil ske en krænkelse af disse menneskers rettigheder i henhold til art. 8.

2.2.2. FN's børnekonvention.

Konventionen af 20. december 1989 om barnets rettigheder blev ratificeret ved kgl. resolution af 5. juli 1991. Den er ikke inkorporeret i dansk ret, og gældende ret skal så vidt muligt fortolkes i overensstemmelse med de forpligtelser, Danmark har valgt at påtage sig ved ratifikationen (Dette gælder, så længe det ikke er nødvendigt direkte at tilsidesætte en lovbestemmelse i national ret.)

Art. 24, stk. 1 og 2, litra (c), i konventionen lyder:

*"**1.** Deltagerstaterne anerkender barnets ret til at nyde den højest opnåelige sundhedstilstand, adgang til at få sygdomsbehandling og genoprettelse af helbredet. Deltagende stater skal stræbe mod at sikre, at intet barn fratages sin ret til adgang til at opnå sådan behandling og pleje.*

***2.** Deltagerstaterne skal arbejde for fuld gennemførelse af denne ret og især tage passende forholdsregler for:*

- *...*
- *(c) at bekæmpe sygdom og underernæring, herunder inden for rammerne af den primære sundhedspleje, blandt andet ved anvendelse af let tilgængelig teknologi og gennem ydelse af tilstrækkelig og nærende mad og rent drikkevand under hensyntagen til de farer og risici, der er knyttet til forurening af miljøet;*
- *..."*

Statens egentlige forpligtelse går ud på, at den skal "stræbe mod at sikre" sådan behandling og pleje, og at "arbejde for fuld gennemførelse" af barnets ret til den højest opnåelige sundhedstilstand.

Den højest opnåelige sundhedstilstand kan ikke indebære, at staten tillader børn (som tilhører en særligt sårbar gruppe også i denne henseende) at blive udsat for stråling af helbredsskadelig karakter eller styrke.

Der foreligger videnskabelig dokumentation for, at en etablering af 5G-systemet, der vil indebære udsættelse for dels kraftigere og dels mere farlig[87] radiofrekvent elektromagnetisk stråling end de allerede etablerede 2G-, 3G- og 4G-systemer (som ifølge den foreliggende dokumentation i sig selv er skadegørende eller indebærer en risiko herfor), i sin nuværende form, jf. pkt. 1.1. ovenfor, klart forventeligt vil være direkte helbredsskadelig og indebære risiko for skader, og som sådan i strid med Danmarks forpligtelser efter art. 24 i FN's børnekonvention.

FN's børnekomité, som også træffer afgørelse i konkrete klagesager, har udstedt en "general comment" nr. 15 i 2013, som er retningslinjer for, hvorledes komitéen fortolker konventionens art. 24.

Det fremgår pkt. III.A, om artiklens normative indhold, at:

"The notion of "the highest attainable standard of health" takes into account both the child's biological, social, cultural and economic preconditions and the State's available resources, supplemented by resources made available by other sources, including nongovernmental organizations, the international community and the private sector.

Children's right to health contains a set of freedoms and entitlements. ... The entitlements include access to a range of facilities, goods, services and conditions that provide equality of opportunity for every child to enjoy the highest attainable standard of health." (understreget

87 Dele af den stråling, der ifølge det foreliggende vil blive udsendt fra 5G-systemet, vil have en mindre styrke, men vil pga. de øvrige karakteristika ikke desto mindre være farligere end den nuværende fra 2G-, 3G- og 4G-systemerne. Se Kuster et al (2018) i pkt. 1.2.2.1.3. ovenfor.

her)

Teleselskaberne har i forvejen stillet effektive kommunikationsmidler til rådighed, som ikke indebærer en implementering 5G-systemet.

Yderligere om stk. 2, litra (c), ang. forurening af miljøet har komitéen anført følgende, jf. ibid., s. 6 – 7:

"States should take measures to address the dangers and risks that local environmental pollution poses to children's health in all settings. ... States should regulate and monitor the environmental impact of business activities that <u>may</u> compromise children's right to health,..." (understreget her)

Formuleringen "may compromise" indikerer kraftigt, at komitéen anvender et forsigtighedsprincip, og at konstateringen af en risiko er tilstrækkelig til at staten skal regulere og monitorere sådanne aktiviteter.

Ud fra en formålsfortolkning må dette indebære, at hensynet til børns helbred (der i sig selv må antages at veje særdeles tungt, særligt over for økonomiske interesser) skal føre til, at staten forbyder former for forurening, som kan skade børns helbred. Dette vil i henhold til den foreliggende videnskabelige dokumentation indebære, at børnekonventionen er til hinder for iværksættelse af 5G-systemet, hvis systemet blot skal overholde de af ICNIRP anbefalede grænseværdier.

Der foreligger kun én afgørelse fra FN's børnekomité, der vedrører art. 24 (kommunikésagsnr. 35/2017). Sagen, der ikke er indholdsmæssigt beskrevet på komitéens hjemmeside, ses ikke ud fra de sparsomme beskrivelser (flygtningebarn) at have relevans for nærværende responsums emne.

Der er ikke taget stilling til spørgsmålet i national, dansk ret.

Der er således ikke en autoritativ retskilde, som kan bekræfte ovenstående fortolkning.

<u>2.2.2.1. Delkonklusion.</u>

Ud fra en fortolkning af FN's børnekonventions ordlyd og formål, sammenholdt med den foreliggende videnskabelige dokumentation for såvel skadevirkninger som skaderisici, er det min vurdering, at en aktivering af 5G-systemet, som det foreligger beskrevet, jf. pkt. 1.1. ovenfor, vil være i strid med den danske stats forpligtelser efter konventionens art. 24.

2.3. Miljøretlige regler.

2.3.1. Forsigtighedsprincippet i EU-retten.

Det EU-retlige forsigtighedsprincip er i dag at finde i Traktaten om den Europæiske Unions Funktionsmåde (TFEU) art. 191, stk. 2:

"Unionens politik på miljøområdet tager sigte på et højt beskyttelsesniveau under hensyntagen til de forskelligartede forhold, der gør sig gældende i de forskellige områder i Unionen. Den bygger på forsigtighedsprincippet og princippet om forebyggende indsats, ..."

I henhold til Europa-Kommissionens meddelelse af 20. februar 2000 om forsigtighedsprincippet, s. 9 – 10, kan det anvendes, *"...hvor de videnskabelige data er utilstrækkelige, foreløbige eller usikre, og den indledende objektive videnskabelige undersøgelse tyder på, at der er rimelig grund til bekymring for, at mulige farlige følger for miljø samt menneskers, dyrs og planters sundhed ikke stemmer overens med det valgte beskyttelsesniveau."*

For så vidt angår nærværende responsums emneområde, vil princippet klart være relevant at bringe i anvendelse, hvis det måtte lægges til grund, at der ikke foreligger tilstrækkelig videnskabelig sikkerhed for at konkludere, at radiofrekvent elektromagnetisk stråling inden for de p.t. anvendte grænseværdier, jf. pkt. 2.1. ovenfor, vil være helbredsskadeligt for (in casu) fugle, dyr og planter omfattet af de i det følgende behandlede miljøretlige direktiver.

2.3.2. Fuglebeskyttelsesdirektivet.

EU-direktivet "om beskyttelse af vilde fugle", kodificeret udgave af 30. november 2009, indeholder en række forpligtelser for EU-lande til at "træffe alle nødvendige foranstaltninger" til "beskyttelse" (herunder bevarelse) af fugle, deres æg, reder og levesteder, jf. art. 1.

De for nærværende responsums problemstilling relevante bestemmelser i direktivet er på det foreliggende grundlag følgende (understregninger indsat her), hvortil der er indsat løbende kommentarer:

Art. 1:

"1. Dette direktiv vedrører beskyttelse af alle de fuglearter, som i vild tilstand har deres naturlige ophold på medlemsstaternes område i Europa, hvor traktaten finder anvendelse. Det omhandler bevarelse, forvaltning og regulering af de pågældende arter og fastsætter regler for udnyttelse af de nævnte arter.
2. Dette direktiv gælder for fugle samt for deres æg, reder og levesteder."

Dette vil sige, at direktivbeskyttelsen omfatter enhver vild fugleart og deres levesteder.

Art. 2:

"Medlemsstaterne træffer alle nødvendige foranstaltninger til at opretholde eller tilpasse bestanden af samtlige de i artikel 1 omhandlede arter på et niveau, som især imødekommer økologiske, videnskabelige og kulturelle krav og samtidig tilgodeser økonomiske og rekreative hensyn."

Art. 3, stk. 1:

"Medlemsstaterne træffer ud fra de i artikel 2 omhandlede hensyn alle nødvendige foranstaltninger for at beskytte, opretholde eller genskabe tilstrækkeligt forskelligartede og vidtstrakte levesteder for alle de i artikel 1 omhandlede fuglearter."

De i pkt. 1.2.3.1. ovenfor i øvrigt citerede undersøgelser kan i princippet være relevante på samtlige fugle omfattet af direktivet.

Henvisningen til art. 2 giver medlemsstaterne en vis skønsmæssig beføjelse til, hvorledes hensynene i art. 3 skal varetages, uanset det er klart udtrykt i art. 2, at de økonomiske hensyn ikke må være de mest tungtvejende.

Det mest sandsynlige baseret på den i pkt. 1.2.3.1. ovenfor gennemgåede forskning er, at en indførelse af 5G-systemet i områder, hvor fuglene har deres levesteder, vil udgøre en overtrædelse af denne bestemmelse.

Art. 4, stk. 1 og 4:

"1. For arter, som er anført i bilag I, træffes der særlige beskyttelsesforanstaltninger med hensyn til deres levesteder for at sikre, at de kan overleve og formere sig i deres udbredelsesområde.
I denne forbindelse tages der hensyn til:

a) arter, der trues af udslettelse

b) arter, der er følsomme over for bestemte ændringer af deres levesteder

…

4. Medlemsstaterne træffer egnede foranstaltninger med henblik på i de i stk. 1 og 2 nævnte beskyttede områder at undgå forurening eller forringelse af levestederne samt forstyrrelse af fuglene, i det omfang en sådan forurening, forringelse eller forstyrrelse har væsentlig betydning for formålet med denne artikel. Medlemsstaterne bestræber sig på at undgå forurening eller forringelse af levesteder også uden for disse beskyttede områder."

Der er tale om en vidtfavnende beskyttelse, som bl.a. omfatter den hvide stork, der var genstand for den videnskabelige undersøgelse, der er refereret til ovenfor pkt. 1.2.3.1. (Balmori 2005). Undersøgelsen påviste bl.a., at der var forskelle i mængden af afkom, og at der var en sammenhæng med nærheden til telemaster, og at nogle reder således var helt uden afkom. Undersøgelsens resultater var endvidere underbygget af eksperimentelle studier på fugleæg.

De i pkt. 1.2.3.1. ovenfor i øvrigt citerede undersøgelser kan i princippet være relevante på samtlige fugle omfattet af bilag I.

Da undersøgelserne samtidig udgør en bastant, videnskabelig dokumentation for, at radiofrekvent elektromagnetisk stråling både kan reducere afkommet, mutere det og påføre skader på levende fugle (og muligvis herunder hindre deres navigationsevne), er det min vurdering, at der ved indførelsen af den påtænkte 5G-system sker en overtrædelse af Danmarks forpligtelser i henhold til fuglebeskyttelsesdirektivets art. 4, stk. 1, idet det ikke "sikres", at de beskyttede fugle kan overleve og formere sig.

Danmark vil ligeledes heller ikke have truffet egnede foranstaltninger til at undgå forurening eller forringelse af fuglenes levesteder eller forstyrrelse af fuglene, uanset dette vil have væsentlig betydning for formålet med art. 4.

Der vil heller ikke være sket nogen bestræbelse på at undgå forurening eller forringelse af levesteder for disse fugle også uden for de beskyttede områder, jf. stk. 4, in fine.

Væsentlig nedbringelse af bestanden af dyr, som insektædende fugle skal kunne leve af, jf. pkt. 1.2.3.2. ovenfor, må ligeledes forventes at have den betydning, at fuglenes levesteder forstyrres i en sådan grad, at det vil have væsentlig betydning for deres overlevelsesmuligheder.

Art. 5, stk. 1, litra a), b) og d):

"Med forbehold af artikel 7 og 9 træffer medlemsstaterne de nødvendige foranstaltninger til at indføre en generel ordning til beskyttelse af alle de i artikel 1 omhandlede fuglearter, herunder især forbud mod:

a) forsætligt at dræbe eller indfange dem, uanset hvilken metode der anvendes

b) forsætligt at ødelægge eller beskadige deres reder og æg samt fjerne deres reder

...

d) forsætligt at forstyrre fuglene navnlig i yngletiden, i det omfang, en sådan forstyrrelse har væsentlig betydning for formålet med dette direktiv

..."

Artikel 7, der drejer sig om jagt, og artikel 9, der indeholder en række undtagelsesbestemmelser uden betydning for etablering af 5G-netværk, er ikke relevante i nærværende sammenhæng.

Artiklen forpligter medlemsstaterne til at etablere generelle beskyttelsesordninger til beskyttelse af de i artikel 1 omhandlede fugle, og det er særligt fremhævet, at der skal være forbud imod bl.a. forsætligt drab på fugle, uanset hvilken metode, der anvendes, og forsætligt at ødelægge eller beskadige reder og æg.

Uanset det ikke er formålet med opstillingen af f.eks. 5G-telemaster at dræbe fugle eller at ødelægge deres reder og æg, er dette en klar og forudsigelig effekt, hvis de opstilles i tilpas nærhed af fuglenes levesteder.

Art. 8, stk. 1:

"For så vidt angår jagt på, fangst af eller drab på fugle i overensstemmelse med dette direktiv forbyder medlemsstaterne anvendelse af alle midler, indretninger eller metoder til massefangst eller -drab eller ikke-selektiv fangst eller drab, som kan medføre, at en art forsvinder lokalt; de forbyder herunder navnlig anvendelse af de i bilag IV, litra a), nævnte midler, indretninger og metoder."

Art. 8 omhandler alene drab, der i forvejen foretages i overensstemmelse med direktivet.

Det bemærkelsesværdige ved formuleringen er, at forbuddet omfatter ikke-selektiv drab, som kan medføre, at en art forsvinder lokalt. Dvs. at bestemmelsen er risikobaseret, således at den blotte fare for, at indretningen eller metoden kan medføre, at en art forsvinder lokalt, er tilstrækkelig til, at den pågældende indretning eller metode skal forbydes. Der fremgår ikke en sådan direkte udtrykt risikobaseret beskyttelse af de i øvrigt ovenfor citerede artikler. I stedet

anvendes udtryk såsom ”beskyttelse”, ”alle nødvendige foranstaltninger”, ”sikre, at de kan overleve”, o.l. Disse anderledes formuleringer lægger i større eller mindre grad op til, at der i disse andre bestemmelser ligeledes skal indfortolkes såvel en risikobaseret beskyttelse som anvendelse af forsigtighedsprincippet. Formuleringen af art. 8, der oven i købet vedrører arter omfattet af den lavere rangerende beskyttelse i direktivets ”bilag II”, underbygger en sådan fortolkning af de øvrige bestemmelser, hvilket endvidere vil være i god overensstemmelse med direktivets beskyttelsesformål.

2.3.2.1. Delkonklusion.

Det er på baggrund af den i pkt. 1.2 ovenfor refererede forskning min vurdering, at hvis 5G-systemet aktiveres, så vil det medføre eller kunne medføre væsentlig skade på de beskyttede vildfugle, der har deres levesteder tilstrækkeligt tæt på f.eks. en relevant telemast.

Denne virkning vil blive forstærket af, at disse installationer påviseligt også har betydelig skadevirkning på de dyr, som insektædende fugle skal leve af, jf. pkt. 1.2.3.2. ovenfor.

Det må følgelig også være min vurdering, at aktiveringen heraf vil udgøre en overtrædelse af Danmarks forpligtelser efter fuglebeskyttelsesdirektivets art. 4 og 5, samt formentlig art. 3.

Hvis det lagdes til grund, at der fortsat består en videnskabelig usikkerhed, bør anvendelsen af forsigtighedsprincippet føre til samme delkonklusioner.

2.3.3. Habitat-direktivet

EU-direktivet "om bevaring af naturtyper samt vilde dyr og planter" af 21. maj 1992 indeholder en række forpligtelser for EU-lande til at "sikre" opretholdelse af gunstig bevaringsstatus for de af direktivet omfattede naturtyper og levesteder for beskyttede arter, og at "sikre sig" ikke at skade de beskyttede lokaliteters integritet eller at "forstyrre" arterne på en måde, som har betydelige konsekvenser for direktivets formål.

Det er således ikke alle dyr og planter, der er omfattet af beskyttelsen. Imidlertid kan den i pkt. 1.3. ovenfor omtalte forskning ikke tages til indtægt for, at den alene finder anvendelse på de specifikke undersøgte arter. Dette gør sig særligt gældende, al den stund mange af dem vedrører forstyrrelse af almene mekanismer, ligesom det i flere tilfælde er udtrykkeligt anført, at de undersøgte arter (f.eks. bananfluer) vil udgøre "gode indikatorer".

De for nærværende responsums problemstilling relevante bestemmelser i direktivet er på det foreliggende grundlag følgende (understregninger indsat her), hvortil der er indsat løbende kommentarer:

Art. 2:

"1. Formålet med dette direktiv er at bidrage til at sikre den biologiske diversitet ved at bevare naturtyperne samt de vilde dyr og planter inden for det af medlemsstaternes område i Europa, hvor Traktaten finder anvendelse.

2. De foranstaltninger, der træffes efter dette direktiv, tager sigte på at opretholde eller genoprette en gunstig bevaringsstatus for naturtyper samt vilde dyre- og plantearter af fællesskabsbetydning.

3. De foranstaltninger, der træffes efter dette direktiv, tager hensyn til de økonomiske, sociale og kulturelle behov og til regionale og lokale særpræg."

Art. 3, stk. 1:

"Der oprettes et sammenhængende europæisk økologisk net af særlige bevaringsområder under betegnelsen Natura 2000. Dette net, der består af lokaliteter, der omfatter de naturtyper, der er nævnt i bilag I, og levesteder for de arter, der er nævnt i bilag II, skal sikre opretholdelse eller i givet fald genopretning af en gunstig bevaringsstatus for de pågældende naturtyper og levestederne for de pågældende arter i deres naturlige udbredelsesområde.

Natura 2000-nettet omfatter ligeledes de særligt beskyttede områder, som medlemsstaterne har udlagt i medfør af direktiv 79/409/EØF."

I henhold til denne bestemmelse skal staterne "sikre" opretholdelse/genopretning af en gunstig bevaringsstatus for levestederne for de af bilag II omfattede arter. Dette gælder bl.a. den hvide stork og de flagermus, som det fremgår af afsnit 1.2.3.1. og 1.2.3.2. ovenfor.

Når der henses til den videnskabelige dokumentation for skadevirkningerne, forekommer dette ikke at være muligt.

Dertil kommer, at en evt. (tilstrækkeligt videnskabeligt funderet) usikkerhed vil skulle afklares.

Art. 6, stk. 2 til 4:

"2. Medlemsstaterne træffer passende foranstaltninger for at undgå forringelse af naturtyperne og levestederne for arterne i de særlige bevaringsområder samt forstyrrelser af de arter, for hvilke områderne er udpeget, for så vidt disse forstyrrelser har betydelige konsekvenser for

dette direktivs målsætninger.

3. Alle planer eller projekter, der ikke er direkte forbundet med eller nødvendige for lokalitetens forvaltning, men som i sig selv eller i forbindelse med andre planer og projekter kan påvirke en sådan lokalitet væsentligt, vurderes med hensyn til deres virkninger på lokaliteten under hensyn til bevaringsmålsætningerne for denne. På baggrund af konklusionerne af vurderingen af virkningerne på lokaliteten, og med forbehold af stk. 4, giver de kompetente nationale myndigheder først deres tilslutning til en plan eller et projekt, når de har sikret sig, at den/det ikke skader lokalitetens integritet, og når de - hvis det anses for nødvendigt - har hørt offentligheden.

4. Hvis en plan eller et projekt, på trods af at virkningerne på lokaliteten vurderes negativt, alligevel skal gennemføres af bydende nødvendige hensyn til væsentlige samfundsinteresser, herunder af social eller økonomisk art, fordi der ikke findes nogen alternativ løsning, træffer medlemsstaten alle nødvendige kompensationsforanstaltninger for at sikre, at den globale sammenhæng i Natura 2000 beskyttes. Medlemsstaten underretter Kommissionen om, hvilke kompensationsforanstaltninger der træffes.
Hvis der er tale om en lokalitet med en prioriteret naturtype og/eller en prioriteret art, kan der alene henvises til hensynet til menneskers sundhed og den offentlige sikkerhed eller væsentlige gavnlige virkninger på miljøet, eller, efter udtalelse fra Kommissionen, andre bydende nødvendige hensyn til væsentlige samfundsinteresser."

Særligt ad stk. 3:

Forpligtelsen går ud på, at myndighederne skal sikre sig, at et projekt m.v. (f.eks. indførelse af 5G-systemet ved opførelse af nye telemaster eller opsættelse af 5G-sendere på eksisterende telemaster) ikke skader lokalitetens integritet.

Når der henses til den videnskabelige dokumentation for skadevirkningerne, forekommer dette ikke at være muligt.

Dertil kommer, at en evt. (tilstrækkeligt videnskabeligt funderet) usikkerhed vil skulle afklares.

Særligt ad stk. 4:

I og med at Sundhedsstyrelsen ikke anerkender henholdsvis skadevirkninger og -risici som gennemgået ovenfor, er der heller ikke grundlag for at antage, at staten har truffet "alle nødvendige kompensationsforanstaltninger", jf. stk. 4, hvis det må lægges til grund, at forskningen i pkt. 1.2 ovenfor er retvisende.

For så vidt angår lokaliteter med en prioriteret naturtype og/eller prioriteret art, finder ingen af de særlige undtagelser anvendelse. Etablering af et 5G-netværk har således ikke nogen væsentlig gavnlig virkning for menneskers sundhed, den offentlige sikkerhed eller miljøet, herunder når der sammenlignes med andre teknologiske muligheder. For så vidt angår hensynet til menneskers sundhed, er det tværtimod klart, at det vil have en skadelig virkning. Der foreligger heller ikke nogen udtalelse fra Kommissionen desangående.

Art. 7:

"Forpligtelserne i artikel 6, stk. 2, 3 og 4, i nærværende direktiv træder i stedet for forpligtelserne i artikel 4, stk. 4, første punktum, i direktiv 79/409/EØF, for så vidt angår de områder, der er udlagt som særligt beskyttede efter artikel 4, stk. 1, eller tilsvarende anerkendt efter artikel 4, stk. 2, deri, fra datoen for nærværende direktivs iværksættelse eller fra den dato, hvor en medlemsstat har udlagt eller anerkendt områderne efter direktiv 79/409/EØF, hvis denne dato er senere."

Direktiv 79/409/EØF er det ovenfor omtalte fuglebeskyttelsesdirektiv (nu: kodificeret ved direktiv 2009/147/EF). Det nye fuglebeskyttelsesdirektiv er også omfattet af henvisningsbestemmelsen i habitatdirektivets art. 7, jf. fuglebeskyttelsesdirektivets art. 18, stk. 2.

Art. 12, stk. 1, 3 og 4:

"1. Medlemsstaterne træffer de nødvendige foranstaltninger til at indføre en streng beskyttelsesordning i det naturlige udbredelsesområde for de dyrearter, der er nævnt i bilag IV, litra a), med forbud mod:
a) alle former for forsætlig indfangning eller drab af enheder af disse arter i naturen

b) forsætlig forstyrrelse af disse arter, i særdeleshed i perioder, hvor dyrene yngler, udviser yngelpleje, overvintrer eller vandrer

c) forsætlig ødelæggelse eller indsamling af æg i naturen

d) beskadigelse eller ødelæggelse af yngle- eller rasteområder.

...

3. Forbuddene i stk. 1, litra a) og b), samt stk. 2 gælder for alle livsstadier hos de dyr, der er omfattet af denne artikel.

4. Medlemsstaterne indfører en ordning med tilsyn med uforsætlig indfangning eller drab af de dyrearter, der er nævnt i bilag IV, litra a). På grundlag af de indhentede oplysninger gennemfører medlemsstaterne de yderligere undersøgelser eller træffer de bevaringsforanstaltninger, der er nødvendige for at sikre, at uforsætlig indfangning eller drab ikke får en væsentlig negativ virkning for de pågældende dyrearter."

Uanset det ikke er formålet med opstillingen af f.eks. 5G-telemaster at dræbe dyr eller at ødelægge deres reder og æg, er dette en klar og forudsigelig effekt af, hvis de opstilles i tilpas nærhed af de beskyttede dyrs levesteder.

Beskyttelsen i habitatdirektivet gælder udtrykkeligt for alle livsstadier hos de omfattede dyr, hvor det i fuglebeskyttelsesdirektivet er anført, at beskyttelsen gælder fugle, deres reder og æg. Der er næppe tilsigtet nogen forskellig anvendelse af bestemmelserne, henset til, at et "hul" i beskyttelsen af de pågældende arter ville kunne gøre reglerne ineffektive.

Art. 13, stk. 1, litra a) og stk. 2:

"1. Medlemsstaterne træffer de nødvendige foranstaltninger for at indføre en streng beskyttelsesordning for de plantearter, der er nævnt i bilag IV, litra b), med forbud mod:
a) forsætlig plukning, indsamling, afskæring, oprivning med rod eller ødelæggelse af disse vildtvoksende planter i naturen

¨...

2. Forbuddene i stk. 1, litra a) og b), gælder for alle livsstadier for de planter, der er omfattet af denne artikel."

Art. 15:

"Ved indfangning eller drab af de vilde dyrearter, som er nævnt i bilag V, litra a), og ved anvendelse efter artikel 16 af fravigelser i forbindelse med indsamling, indfangning eller drab af de arter, der er nævnt i bilag IV, litra a), forbyder medlemsstaterne anvendelse af alle ikke-selektive midler, der lokalt kan medføre, at bestande af en art forsvinder eller udsættes for

alvorlige forstyrrelser, navnlig
a) anvendelse af de indfangnings- og drabsmetoder, der er nævnt i bilag VI b)
...”

I bilag VI (rettelig er der tale om bilag VI a), ikke b)) er bl.a. nævnt *”...elektriske og elektroniske apparater, som kan dræbe eller lamme...”* Det er ikke afklaret, om der i denne definition kan inkluderes apparater såsom telemaster, antenner, m.v., som over en længere eller meget lang periode kan gøre dødelig skade på de af direktivet omfattede dyr. Det kan ikke udelukkes, uanset der med selve formuleringen formentlig er ment apparater, som mere umiddelbart kan dræbe eller lamme. Med anvendelsen af udtrykket ”navnlig” i selve art. 15 anføres imidlertid, at de i bilag VI a) nævnte midler ikke er udtømmende, og at forbuddet omfatter ethvert middel, som lokalt vil kunne medføre, at en artsbestand forsvinder eller forstyrres alvorligt.

Det forekommer således oplagt, at påtænkte 5G-installationer er i strid med selve art. 15, uanset de evt. også kan henføres til de specifikke apparater i bilag VI a).

Art. 16, stk. 1, litra c):

”1. Hvis der ikke findes nogen anden brugbar løsning, og fravigelsen ikke hindrer opretholdelse af den pågældende bestands bevaringsstatus i dens naturlige udbredelsesområde, kan medlemsstaterne fravige bestemmelserne i artikel 12, 13, 14 og 15, litra a) og b):
...
c) af hensyn til den offentlige sundhed og sikkerhed eller af andre bydende nødvendige hensyn til væsentlige samfundsinteresser, herunder af social og økonomisk art, og hensyn til væsentlige gavnlige virkninger på miljøet
...”

Der findes andre, brugbare løsninger.

Dertil kommer, at det på baggrund af det i pkt. 1.2 behandlede forskningsmateriale må være min vurdering, at en fravigelse af beskyttelsen med stor sandsynlighed over tid vil kunne hindre opretholdelse af bestandene.

2.3.3.1. Delkonklusion.

Det er på baggrund af den i pkt. 1.2 ovenfor refererede forskning min vurdering, at hvis 5G-systemet aktiveres, så vil det medføre eller kunne medføre væsentlig skade på de beskyttede dyre- og plantearter, der har deres levesteder tilstrækkeligt tæt på f.eks. en telemast.

Denne virkning vil blive forstærket af, at disse installationer påviseligt også har betydelig skadevirkning på insektædere, jf. pkt. 1.2.3.2. ovenfor.

Det må følgelig også være min vurdering, at aktiveringen heraf vil udgøre en overtrædelse af Danmarks forpligtelser efter habitatdirektivets art. 6, stk. 2 – 4.

Hvis det lagdes til grund, at der fortsat består en videnskabelig usikkerhed, bør anvendelsen af forsigtighedsprincippet føre til samme delkonklusioner.

2.4. Bern-konventionen

Konvention af 19. september 1979 "om beskyttelse af Europas vilde dyr og planter samt naturlige levesteder" (herefter: *"Bern-konventionen"*) blev ratificeret af Danmark i henhold til kongelig resolution af 5. juli 1982. Den er ikke inkorporeret i dansk ret, og gældende ret skal så vidt muligt fortolkes i overensstemmelse med de forpligtelser, Danmark har valgt at påtage sig ved ratifikationen (Dette gælder, så længe det ikke er nødvendigt direkte at tilsidesætte en lovbestemmelse i national ret.)

Konventionen indeholder bl.a. en række bestemmelser, hvormed de kontraherende stater har forpligtet sig til at *"...sikre..."* beskyttelsen af en række vilde dyr og planter, således at bestanden opretholdes, samtidig med at der "tages hensyn til de økonomiske behov", alt jf. art. 2.

De for nærværende responsums problemstilling relevante bestemmelser i konventionen er på det foreliggende grundlag følgende (understregninger indsat her), hvortil der er indsat løbende kommentarer:

Art. 2:
"De kontraherende parter skal træffe de nødvendige foranstaltninger for at opretholde bestanden af vilde dyr og planter på, eller at tilpasse den til, et niveau, som svarer til de særlige økologiske, videnskabelige og kulturelle behov, idet der samtidig tages hensyn til de økonomiske og rekreative behov og behov hos underarter, geografiske racer eller former, som trues lokalt."

Art. 3, stk. 2:
"Hver kontraherende part forpligter sig til i sin planlægnings- og egnsudviklingspolitik og i sine foranstaltninger mod forurening at tage hensyn til beskyttelsen af vilde dyr og planter."

Art. 4, stk. 1 til 3:
"1. Hver kontraherende part skal træffe passende og nødvendige lovgivningsmæssige og administrative foranstaltninger for at sikre beskyttelsen af levesteder for vilde dyre- og plantearter, navnlig de i liste I og II anførte, og beskyttelsen af truede naturlige levesteder.

2. De kontraherende parter skal i deres planlægnings- og egnsudviklingspolitik tage hensyn til beskyttelsesbehovene i de områder, som skal beskyttes i henhold til stk. 1, således at man undgår eller så vidt muligt begrænser en hvilken som helst forringelse af sådanne områder.

3. De kontraherende parter forpligter sig til at tage særligt hensyn til beskyttelsen af områder, som er af betydning for de migrerende arter, der er anført i liste II og III, og som har en passende beliggenhed i forhold til migrationsruter som overvintringsområder, rastepladser, fourageringspladser, yngleområder eller fældningsområder."

Art. 5, 1. pkt.:
"Hver kontraherende part skal træffe passende og nødvendige lovgivningsmæssige og administrative foranstaltninger for at sikre en særlig beskyttelse af de vilde plantearter, som er anført i liste I. ..."

Art. 6:
"Hver kontraherende part skal træffe passende og nødvendige lovgivningsmæssige og administrative foranstaltninger for at sikre en særlig beskyttelse af de vilde dyrearter, som er anført i liste II. Navnlig skal der i forbindelse med disse arter være forbud mod følgende:

-a.- alle former for forsætlig indfangning og fangenskabshold samt forsætlig ihjelslagning,

-b. forsætlig skade på eller ødelæggelse af yngle- og rastepladser,

-c.- forsætlig forstyrrelse af vilde dyr, i særdeleshed i perioder, når de yngler, udviser ynglepleje og overvintrer, for så vidt som forstyrrelsen måtte være væsentlig i forbindelse med denne konventions målsætninger,

…"

Art. 7, stk. 1 og 2:
"Hver kontraherende part skal træffe passende og nødvendige lovgivningsmæssige og administrative foranstaltninger for at sikre beskyttelse af de vilde dyrearter, som er anført i liste III.

Enhver udnyttelse af de vilde dyrearter, som er anført i liste III, skal under hensyntagen til bestemmelserne i artikel 2 reguleres med henblik på at forebygge, at bestandene bliver truet."

Til art. 4 til 7:

Den forudsatte "sikring" af beskyttelsen af de i liste I og II anførte arter er efter min vurdering på baggrund af den i pkt. 1.2. ovenfor gennemgåede forskning sammenholdt med de p.t. værende grænseværdier ikke mulig ved indførelsen af det påtænkte 5G-system.

Dette er særligt tydeligt for så vidt angår forpligtelsen i art. 4, stk. 2, til at undgå eller så vidt muligt begrænse en hvilken som helst forringelse af sådanne områder.

Art. 8:
"I forbindelse med indfangning eller ihjelslagning af de vilde dyrearter, som er anført i liste III, og i tilfælde af benyttelsen af undtagelser i medfør af artikel 9 på de arter, som er anført i liste II, skal de kontraherende parter forbyde anvendelsen af ikke-selektive fangst- og drabsmetoder og af alle midler, som vil kunne medføre, at bestande af en art forsvinder i et lokalt område eller forstyrres alvorligt, samt navnlig anvendelsen af de midler, som er opregnet i liste IV."

Bestemmelsen er formuleret som risikobaseret, således at forbuddet gælder den blotte mulighed for, at den ikke-selektive drabsmetode eller middel vil kunne medføre bestandenes forsvinden. (Det er således særligt oplagt at bringe forsigtighedsprincippet i anvendelse, hvis det lægges til grund, at der fortsat foreligger videnskabelig uklarhed.)

Endvidere indeholder art. 8 en reference til en *"Liste IV"* med angivelse af forbudte midler og metoder til ihjelslagning, m.v., af vilde dyr omfattet af konventionens *"Liste III*" (jf. art. 7 ovenfor). Listen omfatter bl.a. et forbud imod at anvende *"Elektriske apparater, som kan dræbe eller lamme"* i forhold til både pattedyr og fugle. Det er ikke afklaret, om der i denne definition kan inkluderes apparater såsom telemaster, antenner, m.v., som over en længere eller meget lang periode kan gøre dødelig skade på de i liste III omfattede dyr. Det kan ikke udelukkes, uanset der med selve formuleringen formentlig er ment apparater, som mere umiddelbart kan dræbe eller lamme. Med anvendelsen af udtrykket "navnlig" i selve art. 8 anføres imidlertid, at de i liste IV nævnte midler og metoder ikke er udtømmende, og at forbuddet omfatter ethvert middel, som vil kunne medføre at en artsbestand forsvinder eller forstyrres alvorligt i det lokale område.

Art. 9, stk. 1:
"Hver kontraherende part kan gøre undtagelser fra bestemmelserne i artiklerne 4, 5, 6 og 7, og fra forbudet mod anvendelse af de midler, der er nævnt i artikel 8, hvis der ikke findes andre tilfredsstillende løsninger, og hvis undtagelsen ikke er til skade for den berørte bestands overlevelse:

- for at beskytte dyr og planter,

- for at forhindre alvorlig skade på afgrøder, besætning, skove, fiskeri, vand og andre former for ejendom,

- af hensyn til den offentlige sundhed og sikkerhed, sikkerheden for luftfarten eller andre offentlige interesser, der måtte gå forud,

...”

Der findes andre tilfredsstillende løsninger.

Dertil kommer, at det på baggrund af det i pkt. 1.2 behandlede forskningsmateriale må være min vurdering, at en fravigelse af beskyttelsen med stor sandsynlighed vil være til skade for bestandenes overlevelse.

2.4.1. Delkonklusion.

Den forudsatte ”sikring” af beskyttelsen af de i liste I og II anførte arter er efter min vurdering på baggrund af den i pkt. 1.2. ovenfor gennemgåede forskning sammenholdt med de p.t. værende grænseværdier ikke mulig ved indførelsen af det påtænkte 5G-system.

Det forekommer sandsynligt, at beskyttelsen efter art. 8 af ”liste III-arter” ligeledes ikke vil blive respekteret ved etableringen af 5G-systemet sammenholdt med de p.t. værende grænseværdier.

2.5. Bonn-konventionen

Konvention af 23. juni 1979 "om beskyttelse af migrerende arter af vilde dyr" (herefter: *"Bonn-konventionen"*) blev ratificeret af Danmark i henhold til kongelig resolution af 5. juli 1982, samme dato som Bern-konventionen ovenfor. Den er ikke inkorporeret i dansk ret, og gældende ret skal så vidt muligt fortolkes i overensstemmelse med de forpligtelser, Danmark har valgt at påtage sig ved ratifikationen (Dette gælder, så længe det ikke er nødvendigt direkte at tilsidesætte en lovbestemmelse i national ret.)

Konventionen indeholder bl.a. en række bestemmelser, hvormed de kontraherende stater har forpligtet sig til "i passende omfang" at "tage skridt til" at bevare truede, migrerende dyrearter, samt deres bosteder, jf. art. 2, stk. 1.

De for nærværende responsums problemstilling relevante bestemmelser i konventionen er på det foreliggende grundlag følgende (understregninger indsat her), hvortil der er indsat løbende kommentarer:

Art. 2, stk. 1 og 2:

"1. The Parties acknowledge the importance of migratory species being conserved and of Range States agreeing to take action to this end whenever possible and appropriate, paying special attention to migratory species the conservation status of which is unfavourable, and taking individually or in co-operation appropriate and necessary steps to conserve such species and their habitat.

2. The Parties acknowledge the need to take action to avoid any migratory species becoming endangered." (understreget her)

Den forudsatte beskyttelse af migrerende arter er efter min vurdering på baggrund af den i pkt. 1.2. ovenfor gennemgåede forskning sammenholdt med de p.t. værende grænseværdier ikke mulig ved indførelsen af det påtænkte 5G-system.

Dertil kommer, at fastholdelsen af de nuværende grænseværdier efter min vurdering vil udelukke, at Danmark kan anses for at have taget "de nødvendige skridt" til opretholdelse af de migrerende arter.

Art. 3, stk. 4:

"Parties that are Range States of a migratory species listed in Appendix I shall endeavour:

a) to conserve and, where feasible and appropriate, restore those habitats of the species which are of importance in removing the species from danger of extinction;

b) to prevent, remove, compensate for or minimize, as appropriate, the adverse effects of activities or obstacles that seriously impede or prevent the migration of the species; and

c) to the extent feasible and appropriate, to prevent, reduce or control factors that are endangering or are likely to further endanger the species, including strictly controlling the introduction of, or controlling or eliminating, already introduced exotic species."

Formuleringen *"to the extent … appropriate"* (i passende omfang), som begrænser forpligtelsen i henhold til litra c, er ikke at finde i litra a og b.

Den forudsatte beskyttelse af migrerende arter er efter min vurdering på baggrund af den i pkt. 1.2. ovenfor gennemgåede forskning sammenholdt med de p.t. værende grænseværdier

ikke mulig ved indførelsen af det påtænkte 5G-system.

2.5.1. Delkonklusion.

Den forudsatte beskyttelse af migrerende arter er efter min vurdering på baggrund af den i pkt. 1.2. ovenfor gennemgåede forskning sammenholdt med de p.t. værende grænseværdier ikke mulig ved indførelsen af det påtænkte 5G-system.

3. Konklusion og afsluttende bemærkninger.

Det konkluderes i nærværende responsum, at etablering og aktivering af et 5G-netværk, således som det p.t. foreligger beskrevet, vil være i strid med gældende menneskeretlige og miljøretlige regler i EMRK, FN's børnekonvention, EU-regler og Bern- og Bonn-konventionerne.

Årsagen hertil er den meget betydelige, videnskabelige dokumentation, der foreligger for, at radiofrekvent elektromagnetisk stråling er helbredsskadelig og farlig for mennesker (og særligt for børn), dyr og planter.

Dette gælder også, når strålingen holder sig inden for de retningslinjer, som anbefales af ICNIRP, og som anvendes af Danmark og bredt i EU.

De nøjagtige helbredsmæssige skadevirkninger af 5G-systemet er ikke kendte, idet der ikke er tale om et eksakt defineret system, men det er på baggrund af den foreliggende forskning i radiofrekvent elektromagnetisk strålings påvirkninger af f.eks. menneskers og dyrs krop, herunder ved fremkaldelsen af DNA-skader og oxidativt stress, stærkt usandsynligt, at det ikke skulle medføre tilsvarende skadevirkninger som de hidtidige systemer, særligt al den stund det er baseret på samme grundlæggende stråling.

Den danske stat tjener betydelige beløb på at tillade oprettelse og drift af kommunikationssystemerne, bl.a. ved beskatning af overskud og auktioner over de frekvensbånd, som teleselskaber benytter til at opbygge den kommunikationsinfrastruktur, der kan indbringe selskaberne selv milliarder.

Alfonso Balmori er én blandt mange forskere, der har udtalt sig på følgende måde om den iboende interessekonflikt i dette strukturelle problem, jf. Balmori 2005 p. 116:

"Controversy is frequent when the scientists recognize serious effects on health and on the environment that cause high economic losses."

Holte, d. 4. maj 2019

Christian F. Jensen
advokat (L)

BILAG 1 TIL RESPONSUM

Agarwal A. et al. 2009, Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study

https://www.sciencedirect.com/science/article/pii/S0015028208033566

"Result(s) Samples exposed to RF-EMW showed a significant decrease in sperm motility and viability, increase in ROS level, and decrease in ROS-TAC score. Levels of TAC [Total Antioxidant Capacity] and DNA damage showed no significant differences from the unexposed group."

Aitken R.J. et al. 2005, Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline.

"In this study, mice were exposed to 900 MHz RFEMR at a specific absorption rate of approximately 90 mW/kg inside a waveguide for 7 days at 12 h per day. Following exposure, DNA damage to caudal epididymal spermatozoa was assessed by quantitative PCR (QPCR) as well as alkaline and pulsed-field gel electrophoresis... This study suggests that while RFEMR does not have a dramatic impact on male germ cell development, a significant genotoxic effect on epididymal spermatozoa is evident and deserves further investigation."

Fulltext & PDF: https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1365-2605.2005.00531.x

Avendaño C. et al. 2011, Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation

exposure of human spermatozoa to a wireless internet-connected laptop decreased motility and induced DNA fragmentation by a nonthermal effect.

Fulltext & PDF: https://www.fertstert.org/article/S0015-0282(11)02678-1/fulltext

Behari J. et al. 2006, Single strand DNA breaks in rat brain cells exposed to microwave radiation

This study shows that the chronic exposure to these radiations (2.45 and 16.5 GHz, SAR 1.0 and 2.01 W/kg, respectively) cause statistically significant ($p < 0.001$) increase in DNA single strand breaks in brain cells of rat.

https://www.sciencedirect.com/science/article/pii/S0027510705005361

Belyaev I.Y. et al. 2009, Microwaves from UMTS/GSM mobile phones induce long lasting inhibition of 53BP1/γ H2AX DNA repair foci in human lymphocytes

"All data were pooled and highly significant inhibitory effects on formation of DNA repair foci were found as analyzed immediately after 1 h exposure to UMTS, 915 MHz and heat shock...The most striking observation was that these MW-
induced inhibitory effects continued up to 3 days following 1 h exposure to MWs... These effects depended on carrier frequency and type of signal and suggested misbalance between DNA damage and DNA repair"

https://onlinelibrary.wiley.com/doi/abs/10.1002/bem.20445

Blank M. et al. 2011, DNA is a fractal antenna in electromagnetic fields

Since DNA can interact with EMF over a wide range of frequencies, and does not appear to be limited to an optimal frequency, it has the functional properties of a fractal antenna....From the above analysis of the effect of EMF on the stress response, DNA strand breaks and cancer epidemiology, the fractal property of DNA is apparent in the ELF and RF ranges.

https://www.tandfonline.com/doi/full/10.3109/09553002.2011.538130

Burlaka A. et al. 2013, Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation

CONCLUSION: Exposure of developing quail embryos to extremely low intensity RF-EMR of GSM 900 MHz during at least one hundred and fifty-eight hours leads to a significant overproduction of free radicals/reactive oxygen species and oxidative damage of DNA in embryo cells. These oxidative changes may lead to pathologies up to oncogenic transformation of cells.

Fulltext & PDF: http://exp-oncology.com.ua/article/6079

Busljeta I. et al. 2004, Erythropoietic changes in rats after 2.45 GJz nonthermal irradiation.

"Adult male Wistar rats (N=40) were exposed to 2.45 GHz continuous RF/MW fields for 2 hours daily, 7 days a week, at 5-10 mW/cm2... In the applied experimental condition, RF/MW radiation might cause disturbance in red cell maturation and proliferation, and induce micronucleus formation in erythropoietic cells."

https://www.ncbi.nlm.nih.gov/pubmed/15729835

Cam ST et al. 2011, Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation

Conclusions: A short-term exposure (15 and 30 min) to RFR (900-MHz) from a mobile phone caused a significant increase in DNA single-strand breaks in human hair root cells located around the ear which is used for the phone calls.

https://www.tandfonline.com/doi/full/10.3109/09553002.2012.666005

Campisi A. et al. 2010, Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field

Our data demonstrate, for the first time, that even acute exposure to low intensity EMF induces ROS production and DNA fragmentation in astrocytes in primary cultures, which also represent the principal target of modulated EMF. Our findings also suggest the hypothesis that the effects could be due to hyperstimulation of the glutamate receptors, which play a crucial role in acute and chronic brain damage. Furthermore, the results show the importance of the amplitude modulation in the interaction between EMF and neocortical astrocytes.

https://www.sciencedirect.com/science/article/abs/pii/S030439401000176X

d'Ambrosio G et al. 2002, Cytogenetic damage in human lymphocytes following GMSK phase modulated microwave exposure

Human peripheral blood cultures were exposed to 1.748 GHz, either continuous wave (CW) or phase only modulated wave (GMSK), for 15 min. The maximum specific absorption rate (approximately 5 W/kg) was higher than that occurring in the head of mobile phone users; however, no changes were found in cell proliferation kinetics after exposure to either CW or GMSK fields. As far as genotoxicity is concerned, the micronucleus frequency result was not affected by CW exposure; however, a statistically significant micronucleus effect was found following exposure to phase modulated field. These results would suggest a genotoxic power of the phase modulation per se.

https://www.ncbi.nlm.nih.gov/pubmed/11793401

De Luliis G.N. et al. 2009, Mobile Phone Radiation Induces Reactive Oxygen Species Production and DNA Damage in Human Spermatozoa In Vitro

"Principal Findings: Purified human spermatozoa were exposed to radio-frequency electromagnetic radiation (RF-EMR) tuned to 1.8 GHz and covering a range of specific absorption rates (SAR) from 0.4 W/kg to 27.5 W/kg. In step with increasing SAR, motility and vitality were significantly reduced after RF-EMR exposure, while the mitochondrial generation of reactive oxygen species and DNA fragmentation were significantly elevated ($P<0.001$). Furthermore, we also observed highly significant relationships between SAR, the oxidative DNA damage bio-marker, 8-OH-dG, and DNA fragmentation after RF-EMR exposure."

"Conclusions: RF-EMR in both the power density and frequency range of mobile phones enhances mitochondrial reactive oxygen species generation by human spermatozoa, decreasing the motility and vitality of these cells while stimulating DNAbase adduct formation and, ultimately DNA fragmentation. These findings have clear implications for the safety of extensive mobile phone use by males of reproductive age, potentially affecting both their fertility and the health and wellbeing of their offspring."

Fulltext & PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2714176/

Diem E. et al. 2005, Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro.

"Abstract: Cultured human diploid fibroblasts and cultured rat granulosa cells were exposed to intermittent and continuous radiofrequency electromagnetic fields (RF-EMF) used in mobile phones, with different specific absorption rates (SAR) and different mobile-phone modulations. DNA strand breaks were determined by means of the alkaline and neutral comet assay. RF-EMF exposure (1800 MHz; SAR 1.2 or 2 W/kg; different modulations; during 4, 16 and 24h; intermittent 5 min on/10 min off or continuous wave) induced DNA single- and double-strand breaks. Effects occurred after 16 h exposure in both cell types and after different mobile-phone modulations. The intermittent exposure showed a stronger effect in the comet assay than continuous exposure. Therefore we conclude that the induced DNA damage cannot be based on thermal effects."

https://www.sciencedirect.com/science/article/abs/pii/S1383571805000896

D'Silva M.H. et al. 2017, Effect of Radiofrequency Radiation Emitted from 2G and 3G Cell Phone on Developing Liver of Chick Embryo – A Comparative Study

Conclusion: The chronic exposure of chick embryo liver to RFR emitted from 2G and 3G cell phone resulted in various structural changes and DNA damage. The changes were more pronounced in 3G experimental group. Based on these findings it is necessary to create awareness among public about the possible ill effects of RFR exposure from cell phone.

Fulltext og PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5583901/

Fucić A. et al. 1992, X-rays, microwaves and vinyl chloride monomer: their clastogenic and aneugenic activity, using the micronucleus assay on human lymphocytes.

"In our study we chose the micronucleus assay with a new mathematical approach to separate clastogenic from aneugenic activity of three well-known mutagens (vinyl chloride monomer, X-rays and microwaves) on the genome of human somatic cells... Microwaves possess some mutagenic characteristics typical of chemical mutagens."

https://www.sciencedirect.com/science/article/pii/0165799292901333

Gajski G. et al. 2009, Radioprotective effects of honeybee venom (Apis mellifera) against 915-MHz microwave radiation-induced DNA damage in wistar rat lymphocytes: in vitro study.

The aim of this study is to investigate the radioprotective effect of bee venom against DNA damage induced by 915-MHz microwave radiation (specific absorption rate of 0.6 W/kg) in Wistar rats... Bee venom is demonstrated to have a radioprotective effect against basal and oxidative DNA damage. Furthermore, bee venom is not genotoxic and does not produce oxidative damage in the low concentrations used in this study.

https://journals.sagepub.com/doi/full/10.1177/1091581809335051

Gandhi G. et al. 2005, Cytogenetic Damage in Mobile Phone Users: Preliminary Data.

"The aim of the present study hence was to detect any cytogenetic damage in mobile phone users by analysing short term peripheral lymphocytes cultures for chromosomal aberrations and the buccal mucosal cells for micronuclei (aneugenicity and clastogenicity). The results revealed increased number of micronucleated buccal cells and cytological abnormalities in cultured lymphocytes indicating the genotoxic response from mobile phone use."

https://www.tandfonline.com/doi/abs/10.1080/09723757.2005.11885936
PDF: http://www.krepublishers.com/02-Journals/IJHG/IJHG-05-0-000-000-2005-Web/IJHG-05-4-225-288-2005-Abst-PDF/IJHG-05-4-259-265-2005-210-Gandhi-G/IJHG-05-4-259-265-2005-210-Gandhi-G.pdf

Gandhi G. et al. 2015, A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.

Genetic damage parameters of DNA migration length, damage frequency (DF) and damage index were significantly (p = 0.000) elevated in the sample group compared to respective values in healthy controls.

https://www.tandfonline.com/doi/abs/10.3109/15368378.2014.933349

Garaj-Vrhovac V et al. 1992, The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro.

Human whole-blood samples were exposed to continuous microwave radiation, frequency 7.7 GHz, power density 0.5, 10 and 30 mW/cm2 for 10, 30 and 60 min. A correlation between specific chromosomal aberrations and the incidence of micronuclei after in vitro exposure was observed. In all experimental conditions, the frequency of all types of chromosomal aberrations was significantly higher than in the control samples... The results of the study indicate that microwave radiation causes changes in the genome of somatic human cells and that the applied tests are equally sensitive for the detection of the genotoxicity of microwaves.

https://www.sciencedirect.com/science/article/pii/0165799292900064

Gorpinchenko I. et al. 2014, The influence of direct mobile phone radiation on sperm quality.

CONCLUSIONS: A correlation exists between mobile phone radiation exposure, DNA-fragmentation level and decreased sperm motility.

Fulltext & PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4074720/

Güler G et al. 2012, The effect of radiofrequency radiation on DNA and lipid damage in female and male infant rabbits.

CONCLUSION: Consequently, it can be concluded that GSM-like RF radiation may induce biochemical changes by increasing free radical attacks to structural biomolecules in the rabbit as an experimental animal model.

https://www.tandfonline.com/doi/full/10.3109/09553002.2012.646349

Gursatej A.G. 2005, Genetic damage in mobile phone users: some preliminary findings.

"In the present study, DNA and chromosomal damage investigations were carried out on the peripheral blood lymphocytes of individuals using mobile phones, being exposed to MW frequency ranging from 800 to 2000 MHz.... CONCLUSIONS: These results highlight a correlation between mobile phone use (exposure to RFR) and genetic damage and require interim public health actions in the wake of widespread use of mobile telephony."

https://tspace.library.utoronto.ca/handle/1807/5943
PDF: https://tspace.library.utoronto.ca/bitstream/1807/5943/1/hg05022.pdf

Karaca E. et al. 2011, The genotoxic effect of radiofrequency waves on mouse brain

Brain cell cultures of the mice were exposed to 10.715 GHz with specific absorbtion rate (SAR) 0.725 W/kG signals for 6 h in 3 days at 25°C to check for the changes in the micronucleus (MNi) assay and in the expression of 11 proapoptotic and antiapoptotic genes. It was found that MNi rate increased 11-fold and STAT3 expression decreased 7-fold in the cell cultures which were exposed to RF. Cell phones which spread RF may damage DNA and change gene expression in brain cells.

https://link.springer.com/article/10.1007%2Fs11060-011-0644-z

Kesari K.K. et al. 2013, Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the hsp27/p38MAPK stress pathway in rat brain.

Result shows that microwave radiation emitted from 3G mobile phone significantly induced DNA strand breaks in brain.

https://link.springer.com/article/10.1007%2Fs12013-013-9715-4

Kumar S. et al. 2014, Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.

"Significant decrease in sperm count, increase in the lipid peroxidation damage in sperm cells, reduction in seminiferous tubules and testicular weight and DNA damage were observed following exposure to EMF in male albino rats. The results suggest that mobile phone exposure adversely affects male fertility."

http://nopr.niscair.res.in/bitstream/123456789/29335/1/IJEB%2052%289%29%20890-897.pdf

Liu C. et al. 2013, Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin.

RESULTS:The levels of DNA damage were significantly increased following exposure to Mobile Phone Radiation(MPR) in the listen, dialed and dialing modes. Moreover, there were significantly higher increases in the dialed and dialing modes than in the listen mode. Interestingly, these results were consistent with the radiation intensities of these modes. However, the DNA damage effects of MPR in the dialing mode were efficiently attenuated by melatonin pretreatment.

Link:https://www.ncbi.nlm.nih.gov/pubmed/23952262

Lu Y. et al. 2012, Reactive Oxygen Species Formation and Apoptosis in Human Peripheral Blood Mononuclear Cell Induced by 900 MHz Mobile Phone Radiation

Abstract: We demonstrate that reactive oxygen species (ROS) plays an important role in the process of apoptosis in human peripheral blood mononuclear cell (PBMC) which is induced by the radiation of 900 MHz radiofrequency electromagnetic field (RFEMF) at a specific absorption rate (SAR) of ~0.4 W/kg when the exposure lasts longer than two hours. The apoptosis is induced through the mitochondrial pathway and mediated by activating ROS and caspase-3, and decreasing

the mitochondrial potential. The activation of ROS is triggered by the conformation disturbance of lipids, protein, and DNA induced by the exposure of GSM RFEMF. Although human PBMC was found to have a self-protection mechanism of releasing carotenoid in response to oxidative stress to lessen the further increase of ROS, the imbalance between the antioxidant defenses and ROS formation still results in an increase of cell death with the exposure time and can cause about 37% human PBMC death in eight hours.

Fulltext & PDF: https://www.hindawi.com/journals/omcl/2012/740280/

Markova E. et al. 2010, Microwaves from Mobile Phones Inhibit 53BP1 Focus Formation in Human Stem Cells More Strongly Than in Differentiated Cells: Possible Mechanistic Link to Cancer Risk

"We studied whether microwaves from mobile telephones of the Global System for Mobile Communication (GSM) and the Universal Global Telecommunications System (UMTS) induce DSBs or affect DSB repair in stem cells... Microwaves from mobile phones inhibited formation of 53BP1 foci in human primary fibroblasts and mesenchymal stem cells. These data parallel our previous findings for human lymphocytes. Importantly, the same GSM carrier frequency (915 MHz) and UMTS frequency band (1947.4 MHz) were effective for all cell types. Exposure at 905 MHz did not inhibit 53BP1 foci in differentiated cells, either fibroblasts or lymphocytes, whereas some effects were seen in stem cells at 905 MHz. Contrary to fibroblasts, stem cells did not adapt to chronic exposure during 2 weeks."

Fulltext & PDF: https://ehp.niehs.nih.gov/doi/abs/10.1289/ehp.0900781

Megha K. et al. 2015, Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain.

In conclusion, the present study suggests that low intensity microwave radiation induces oxidative stress, inflammatory response and DNA damage in brain by exerting a frequency dependent effect. The study also indicates that increased oxidative stress and inflammatory response might be the factors involved in DNA damage following low intensity microwave exposure.

https://www.sciencedirect.com/science/article/pii/S0161813X15300097

Panagopoulos D.J. 2007, Cell death induced by GSM 900-MHz and DCS 1800-MHz mobile telephony radiation.

Results (uddrag): The data reveal that both GSM 900 and DCS 1800 mobile telephony radiations strongly induce cell death (DNA fragmentation) in ovarian egg chambers of the exposed groups, (63.01% in 900, 45.08% in 900A and 39.43% in 1800), while in the SE [Sham Exposed] and C [Control] groups the corresponding percentage of cell death was only 7.78% and 7.75%, respectively.

https://www.sciencedirect.com/science/article/abs/pii/S1383571806003202

Pandey N. et al. 2016 Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice

"Result: Swiss albino mice were exposed to RFR (900 MHz) for 4 h and 8 h duration per day for 35 days. One group of animals was terminated after the exposure period, while others were kept for an

additional 35 days post-exposure. RFR exposure caused depolarization of mitochondrial membranes resulting in destabilized cellular redox homeostasis. Statistically significant increases in the damage index in germ cells and sperm head defects were noted in RFR-exposed animals."

https://journals.sagepub.com/doi/abs/10.1177/0748233716671206

Phillips JL et al. 2009, Electromagnetic fields and DNA damage

This review describes the comet assay and its utility to qualitatively and quantitatively assess DNA damage, reviews studies that have investigated DNA strand breaks and other changes in DNA structure, and then discusses important lessons learned from our work in this area. Dette er jo kun et review. det er ikke en artikel der repræsenterer et studie hvor sammenhængen er påvist. MEN DEN ERVIGTIG fordi den giver en oversigt over hvilke faktorer der er afgørende for om der findes genotoxiske effekter eller ej i videnskabelige forsøg. (feks celletype og strålingens pulsering)

Fulltext & PDF: https://www.pathophysiologyjournal.com/article/S0928-4680(09)00014-5/fulltext

Salford L et al. 2003, Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones

Three groups each of eight rats were exposed for 2 hr to Global System for Mobile Communications (GSM) mobile phone electromagnetic fields of different strengths [0.24. 2.4, and 24 W/m^2]. We found highly significant ($p < 0.002$) evidence for neuronal damage in the cortex, hippocampus, and basalganglia in the brains of exposed rats.

Fulltext & PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241519/

Schwarz C. 2008, Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes.

CONCLUSION: UMTS [3G] exposure may cause genetic alterations in some but not in all human cells in vitro.

https://link.springer.com/article/10.1007%2Fs00420-008-0305-5

Semra T.C. et al. 2012, Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation

Conclusions: A short-term exposure (15 and 30 min) to RFR (900-MHz) from a mobile phone caused a significant increase in DNA single-strand breaks in human hair root cells located around the ear which is used for the phone calls.

https://www.tandfonline.com/doi/abs/10.3109/09553002.2012.666005

Tice R.R. et al. 2002, Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells.

This research demonstrates that, under extended exposure conditions, RF signals at an average SAR of at least 5.0 W/kg are capable of inducing chromosomal damage in human lymphocytes.

https://onlinelibrary.wiley.com/doi/abs/10.1002/bem.104

Trosic I et al. 2011, Effect of electromagnetic radiofrequency radiation on the rats' brain, liver and kidney cells measured by comet assay

The results of this study suggest that, under the experimental conditions applied, repeated 915 MHz irradiation could be a cause of DNA breaks in renal and liver cells, but not affect the cell genome at the higher extent compared to the basal damage.

https://www.researchgate.net/publication/221683991_Effect_of_Electromagnetic_Radiofrequency_Radiation_on_the_Rats'_Brain_Liver_and_Kidney_Cells_Measured_by_Comet_Assay

Verschaeve L. 2009, Genetic damage in subjects exposed to radiofrequency radiation

A majority of these studies do show that RF-exposed individuals have increased frequencies of genetic damage (e.g., chromosomal aberrations) in their lymphocytes or exfoliated buccal cells.

https://www.sciencedirect.com/science/article/pii/S1383574208001415

Xu S. et al. 2010, Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons.

In this study, we exposed primary cultured cortical neurons to pulsed RF electromagnetic fields at a frequency of 1800 MHz modulated by 217 Hz at an average special absorption rate (SAR) of 2 W/kg. At 24 h after exposure, we found that RF radiation induced a significant increase in the levels of 8-hydroxyguanine (8-OHdG), a common biomarker of DNA oxidative damage, in the mitochondria of neurons. Concomitant with this finding, the copy number of mtDNA and the levels of mitochondrial RNA (mtRNA) transcripts showed an obvious reduction after RF exposure. Each of these mtDNA disturbances could be reversed by pretreatment with melatonin, which is known to be an efficient antioxidant in the brain. Together, these results suggested that 1800 MHz RF radiation could cause oxidative damage to mtDNA in primary cultured neurons. Oxidative damage to mtDNA may account for the neurotoxicity of RF radiation in the brain.

https://www.sciencedirect.com/science/article/pii/S0006899309022999

Yakymenko I. et al, 2010, Risks of carcinogenesis from electromagnetic radiation of mobile telephony devices.

Among reproducible biological effects of low-intensive MWs are reactive oxygen species overproduction, heat shock proteins expression, DNA damages, apoptosis. The lack of generally accepted mechanism of biological effects of low-intensive non-ionizing radiation doesn't permit to disregard the obvious epidemiological and experimental data of its biological activity.

Fulltext &
PDF: https://www.researchgate.net/publication/45538585_Risks_of_carcinogenesis_from_electromagnetic_radiation_of_mobile_telephony_devices

Zalata A. et al. 2015 , In vitro effect of cell phone radiation on motility, DNA fragmentation and clusterin gene expression in human sperm.

CONCLUSION: Cell phone emissions have a negative impact on exposed sperm motility index, sperm acrosin activity, sperm DNA fragmentation and seminal CLU gene expression, especially in OAT* cases.
*** = tilfælde hvor koncentration af sædceller er lav.**

Fulltext & PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4410031/

Zothansiama et al. 2017, Impact of radiofrequency radiation on DNA damage and antioxidants in peripheral blood lymphocytes of humans residing in the vicinity of mobile phone base stations

Testpersoner bosat indenfor 80m radius af mobilmast havde signifikant flere mikronuklei (blod markør for DNA-skader) og nedsat anti-oxidant status end i personer bosat udenfor 300m radius.

https://www.tandfonline.com/doi/abs/10.1080/15368378.2017.1350584

Zotti-Martelli L. et al. 2005, Individual responsiveness to induction of micronuclei in human lymphocytes after exposure in vitro to 1800-MHz microwave radiation.

The results show that microwaves are able to induce MN [micronuclei] in short-time exposures to medium power density fields. Our data analysis highlights a wide inter-individual variability in the response, which was confirmed to be a characteristic reproducible trait by means of the second experiment.

https://www.sciencedirect.com/science/article/abs/pii/S138357180500032X

Nº Expediente: 18011666

Sr. D.
ANGEL CRUZ BAYÓN GOZALO
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REGISTRO

SALIDA
21/08/2019 - 19080322

Estimado Sr.:

En relación con la queja arriba indicada, se le comunica que se ha recibido un informe elaborado por la Dirección General de Telecomunicaciones, dependiente de la Secretaría de Estado para el Avance Digital, que puede resumirse como sigue:

I. El Plan Nacional 5G, publicado por el anterior Ministerio de Energía, Turismo y Agenda Digital en diciembre de 2017, pretende contribuir a la consecución de los objetivos marcados a nivel europeo en el Plan de Acción de 5G para Europa aprobado por las instituciones comunitarias en abril de 2016 como estrategia para mejorar la competitividad europea en el desarrollo de esta tecnología emergente, en el que se incluye diversos objetivos para los Estados Miembros, entre ellos, la aprobación de planes de trabajo nacionales.

El Plan se enmarca en un contexto internacional en el que los principales países europeos (Alemania, Francia, Reino Unido, etc.) y de otras regiones del mundo (USA, Japón, Corea, etc.) están aprobando sus estrategias para el desarrollo del 5G, y España no podía quedarse atrás.

No obstante, el Plan Nacional 5G no es una norma que establezca preceptos jurídicos relacionados con los despliegues 5G, que deban ser objeto de una evaluación específica y previa a su aprobación y publicación, ni un documento vinculante que genere efectos frente a terceros. El Plan es únicamente un documento de carácter general y de referencia que define y trata de impulsar una estrategia de una política pública, en el que se identifican los tipos de medidas, de carácter estratégico, que se considera se deben a llevar a cabo para que España no quede retrasada en el desarrollo de esta nueva tecnología, y pueda perder las oportunidades que proporcionará en el futuro. Los aspectos ambientales deben ser analizados cuando se proceda al despliegue real de las infraestructuras que sirvan de soporte para la tecnología 5G.

II. Así, el Plan Nacional 5G no se ha sometido a evaluación ambiental estratégica. Se trata de un plan que contiene los principios programáticos y relaciona las líneas estratégicas que se considera deben ser ejecutadas para lograr el impulso de esta

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"nueva tecnología disruptiva" a nivel social y económico. Es un documento estratégico que identifica las líneas directrices y distintas políticas públicas que deberían ser tenidas en cuenta para situar a nuestro país en una situación de liderazgo en la adopción y consolidación de la tecnología 5G. No es un norma ni un documento vinculante que genere efectos frente a terceros, como ser un plan general de ordenación urbana, que sea directamente aplicable y ejecutable, y que, en consecuencia, deba haber efectuado un evaluación medioambiental. Por tanto, el Plan Nacional 5G, atendiendo a su naturaleza y finalidad, "*no ha sido sometido a evaluación de impacto ambiental*" (sic).

Otra cuestión es el despliegue en concreto de las infraestructuras de red, en particular, de las estaciones base, en que se plasmará finalmente los recursos necesarios para poder utilizar la tecnología 5G a través la prestación de una serie de servicios a instituciones, empresas y ciudadanos.

En tal sentido, el artículo 34.6 de la Ley 9/2014, de 9 de mayo, General de Telecomunicaciones, establece:

> "*6. Para la instalación de las estaciones o infraestructuras radioeléctricas utilizadas para la prestación de servicios de comunicaciones electrónicas disponibles para el público a las que se refiere la disposición adicional tercera de la Ley 12/2012, de 26 de diciembre, de medidas urgentes de liberalización del comercio y de determinados servicios, no podrá exigirse la obtención de licencia previa de instalaciones, de funcionamiento o de actividad, ni otras de clase similar o análogas, en los términos indicados en la citada ley.*
>
> *Para la instalación de redes públicas de comunicaciones electrónicas o de estaciones radioeléctricas en dominio privado distintas de las señaladas en el párrafo anterior, no podrá exigirse por parte de las administraciones públicas competentes la obtención de licencia o autorización previa de instalaciones, de funcionamiento o de actividad, o de carácter medioambiental, ni otras licencias o aprobaciones de clase similar o análogas que sujeten a previa autorización dicha instalación, en el caso de que el operador haya presentado a la administración pública competente para el otorgamiento de la licencia o autorización un plan de despliegue o instalación de red de comunicaciones electrónicas, en el que se contemplen dichas infraestructuras o estaciones, y siempre que el citado plan haya sido aprobado por dicha administración.*
>
> *(…)*
>
> *Las licencias o autorizaciones previas que, de acuerdo con los párrafos anteriores, no puedan ser exigidas, serán sustituidas por declaraciones responsables, de conformidad con lo establecido en el artículo 71 bis de la Ley 30/1992, de 26 de noviembre, de Régimen Jurídico de las administraciones públicas y del Procedimiento Administrativo Común, relativas al cumplimiento de las previsiones legales establecidas en la normativa vigente. En todo caso, el declarante deberá estar en posesión del justificante de pago del tributo correspondiente cuando sea preceptivo.*
>
> *(…)*
>
> *La presentación de la declaración responsable, con el consiguiente efecto de habilitación a partir de ese momento para ejecutar la instalación, no prejuzgará en modo alguno la situación y efectivo acomodo de las condiciones de la infraestructura o estación radioeléctrica a la normativa aplicable, ni limitará el ejercicio de las potestades administrativas de comprobación, inspección,*

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sanción, y, en general, de control que a la administración en cualquier orden, estatal, autonómico o local, le estén atribuidas por el ordenamiento sectorial aplicable en cada caso".

En todo caso, el inicio de la implantación de la tecnología 5G a corto y medio plazo se va a efectuar a través de la infraestructuras y estaciones bases actualmente desplegadas, que suelen estar utilizando la tecnología 4G en las mencionadas bandas de frecuencia que son objeto de uso a nivel masivo por diferentes servicios radioeléctricos comerciales (700 MHz, 800 MHz, 900 MHz, 1,8 GHz, 2,1 GHz, 2,6 GHz, 3,5 GHz), estaciones base que ya previamente han sido autorizadas tanto en lo relativo a su proyecto técnico como en su puesta en servicio.

Así, cabe traer a colación nuevamente la Ley General de Telecomunicaciones, cuyo artículo 34.7 prevé expresamente el supuesto de una renovación puramente tecnológica de una estación base previamente instalada, no sujetándola a un riguroso control administrativo en diferentes ámbitos de actuación, incluido el medioambiental:

"7. En el caso de que sobre una infraestructura de red pública de comunicaciones electrónicas, fija o móvil, incluidas las estaciones radioeléctricas de comunicaciones electrónicas, ya esté ubicada en dominio público o privado, se realicen actuaciones de innovación tecnológica o adaptación técnica que supongan la incorporación de nuevo equipamiento o la realización de emisiones radioeléctricas en nuevas bandas de frecuencias o con otras tecnologías, sin variar los elementos de obra civil y mástil, no se requerirá ningún tipo de concesión, autorización o licencia nueva o modificación de la existente o declaración responsable o comunicación previa a las administraciones públicas competentes por razones de ordenación del territorio, urbanismo o medioambientales".

Así se concluye que el Plan Nacional 5G, atendiendo a su naturaleza y finalidad, no ha sido sometido a evaluación de impacto ambiental ni se solicitó su evaluación.

III. El Plan Nacional 5G de España prevé la realización de proyectos piloto para probar las nuevas funcionalidades que ofrecerá la tecnología 5G. En particular, se han convocado 2 proyectos piloto por la entidad pública Red.es, que han sido adjudicados por dicha entidad mediante Resolución del 30 de abril de 2019.

Una parte de estas pruebas se desarrollan introduciendo nuevas funcionalidades en estaciones 4G ya existentes y en servicio. Cuando se requiere la instalación y operación durante un tiempo de una nueva estación, para la instalación y uso de la misma es aplicable la regulación del uso del dominio público radioeléctrico, que garantiza un control exhaustivo en la instalación y uso en el tiempo de todo centro emisor o estación base radioeléctrica.

IV. Los límites de las emisiones radioeléctricas no dependen de la tecnología utilizada en la comunicación (2G, 3G, 4G o 5G) sino de las características específicas de cada banda de frecuencias. Las bandas de frecuencias que se van a utilizar para la tecnología 5G, en un primer estadio o posterior, van a ser las que ya están siendo utilizadas masivamente por diferentes servicios radioeléctricos comerciales (700 MHz, 800 MHz, 900 MHz, 1,8 GHz, 2,1 GHz, 2,6 GHz, 3,5 GHz) con el único añadido de la

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banda de 26 GHz. Esta última banda no ha sido aún adjudicada para el uso de 5G en España para servicios comerciales masivos.

Los organismos internacionales de normalización ya están trabajando en una norma técnica que cubra el cálculo y el procedimiento de medida de emisiones en las bandas milimétricas, como es esta de 26 GHz. En concreto, la Comisión Electrotécnica Internacional (IEC) está desarrollando la norma IEC 62232 (*Determination of RF field strength, power density and SAR in the vicinity of radiocommunication base stations for the purpose of evaluating human exposure*), en avanzado estado de elaboración, que permitirá la evaluación de los niveles de emisión en estas bandas milimétricas.

Asimismo, la Comisión Internacional sobre la Protección contra la Radiación No Ionizante (International Commission on Non-Ionizing Radiation Protection, ICNIRP), está estudiando y revisando las directrices científicas por ella. Estas directrices derivadas de los estudios de la exposición a campos electromagnéticos sirvieron de base para Recomendación 1999/519/CE del Consejo de Sanidad de la Unión Europea, de 12 de julio de 1999, relativa a la exposición del público en general a campos electromagnéticos desde 0 Hz a 300 GHz. Este es el marco europeo que garantiza el nivel de protección de la población en la exposición a los campos electromagnéticos procedentes de productos y aparatos eléctricos o electrónicos.

La ICNIRP publicará, en caso de que lo considere necesario, una actualización de las directrices científicas. Y en el caso de que se produzca esta revisión, se espera que consecuentemente sea revisada la Recomendación 1999/519/CE del Consejo de Sanidad de la Unión Europea, así como el Real Decreto 1066/2001 por el que se aprueba el Reglamento que establece condiciones de protección del dominio público radioeléctrico, restricciones a las emisiones radioeléctricas y medidas de protección sanitaria frente a emisiones radioeléctricas. Este decreto incorpora los criterios de protección sanitaria establecidos en la Recomendación, y su anexo II establece los límites de exposición, en el que se incluyen los límites máximos de emisión para las bandas de frecuencias usadas por las comunicaciones móviles, que son las que utilizará también la tecnología 5G. Así, este Real Decreto establece que el Ministerio de Sanidad y Consumo adaptará al progreso científico el mencionado anexo II, teniendo en cuenta el principio de precaución y las evaluaciones realizadas por las organizaciones nacionales e internacionales competentes.

Las directrices científicas de la ICNIRP son los estándares internacionales que tienen el respaldo de la Organización Mundial de la Salud (OMS). La OMS recomienda una estricta adhesión a los estándares internacionales citados con anterioridad, que han sido desarrollados para proteger tanto a los usuarios de telefonía móvil, como a las personas que trabajan cerca o viven alrededor de estaciones base de telefonía móvil, y a la gente que no hace uso de este tipo de comunicación. La OMS también recomienda que no se impongan límites arbitrarios, desautorizando o desconfiando de la regulación existente, pues esta se basa en el conocimiento científico.

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V. El Reglamento sobre el uso del dominio público radioeléctrico, aprobado por el Real Decreto 123/2017, prevé mecanismos de seguimiento de los niveles de exposición, mediante la presentación de certificaciones e informes por parte de operadores de telecomunicaciones, la realización planes de inspección y la elaboración de un informe anual.

El Reglamento sobre el uso del dominio público radioeléctrico, establece que los operadores que establezcan determinadas redes, entre ellas las redes de telefonía móvil, tienen que elaborar un estudio detallado, realizado por técnico competente, que indique los niveles de exposición radioeléctrica en áreas cercanas a sus instalaciones radioeléctricas, en las que puedan permanecer habitualmente personas, y que este estudio tiene que ser presentado ante el Ministerio de Economía y Empresa incorporado en el proyecto que se tiene que presentar para solicitar la autorización de las instalaciones radioeléctricas.

Asimismo, con carácter previo al inicio de emisiones, los operadores tienen que obtener la autorización de puesta en servicio de las estaciones prevista en el Título IV del mencionado reglamento sobre el uso del dominio público radioeléctrico, y para otorgarla los servicios de inspección de la Secretaría de Estado para el Avance Digital comprueban el cumplimiento de los niveles de emisiones en el caso de estaciones que requieren de reconocimiento técnico, o los operadores tienen que presentar un certificado de niveles de exposición realizado por un técnico competente en materia de telecomunicaciones, en el caso de estaciones a las que sea aplicable el procedimiento de certificación sustitutiva del reconocimiento técnico.

Además de lo anterior, de acuerdo con lo establecido en el "Dispongo" cuatro de la Orden CTE/23/2002, de 11 de enero, por la que se establecen condiciones para la presentación de determinados estudios y certificaciones por operadores de servicios de radiocomunicaciones los operadores tienen que remitir al Ministerio de Economía y Empresa, en el primer trimestre de cada año natural, una certificación emitida por técnico competente de que se han respetado durante al año anterior los límites de exposición establecidos en el anexo II del Reglamento que establece las restricciones a las emisiones radioeléctricas y medidas de protección sanitaria frente a emisiones radioeléctricas, aprobado por el Real Decreto 1066/2001.

Adicionalmente, el Ministerio lleva a cabo unos planes de inspección anuales. En ellos se incluyen la realización de medidas en el entorno de una muestra de estaciones radioeléctricas y una auditoria de las certificaciones presentadas por los operadores.

Asimismo, y debido a la importancia que otorga la normativa española a los espacios sensibles (guarderías, centros de educación infantil, primaria, centros de enseñanza obligatoria, centros de salud, hospitales, parques públicos, residencias o centros geriátricos, etc) también se realizan trabajos específicos para comprobar los niveles de exposición radioeléctrica en dichos lugares sensibles.

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Se concluye que la regulación actual sobre el uso del dominio público radioeléctrico y control de los niveles de emisión es adecuada, incluye las decisiones adoptadas por los organismos internacionales competentes e incorpora el principio de precaución en la delimitación y fijación de los límites de exposición a las emisiones radioeléctricas, garantizándose el oportuno y estricto control en su cumplimiento.

Hasta aquí la información recibida de la Secretaría de Estado para el Avance Digital, a la que esta institución ha dirigido, con esta misma fecha, las siguientes consideraciones:

"**1.** Lo primero que debe señalarse, es que la evaluación ambiental de planes tiene un objeto distinto y está sujeta a un procedimiento diferente a de la evaluación de impacto ambiental proyectos.

La Ley 21/2013 define con carácter general la evaluación ambiental como el proceso a través del cual se analizan los efectos significativos que tienen o pueden tener los planes y proyectos sobre el medio ambiente, antes de su adopción, aprobación o autorización. Ese análisis incluye los efectos de aquellos sobre la población, la salud humana, la flora, la fauna, la biodiversidad, la geodiversidad, la tierra, el suelo, el subsuelo, el aire, el agua, el clima, el cambio climático, el paisaje, los bienes materiales, incluido el patrimonio cultural, y la interacción entre todos los factores mencionados.

Partiendo de la definición general, la Ley distingue entre la evaluación ambiental estratégica, a la que deben someterse los planes, y la evaluación de impacto ambiental, que procede respecto de los proyectos.

Para aclarar la cuestión, la Ley define qué debe entenderse por plan y qué debe entenderse por proyecto.

El artículo 5.2 b) de la Ley define un plan como el conjunto de estrategias, directrices y propuestas destinadas a satisfacer necesidades sociales, no ejecutables directamente, sino a través de su desarrollo por medio de uno o varios proyectos. Por su parte, en el artículo 5.3 b) de la Ley, un proyecto se define como cualquier actuación que consista en: 1º la ejecución, explotación, desmantelamiento o demolición de una obra, una construcción, o instalación, o bien 2º cualquier intervención en el medio natural o en el paisaje, incluidas las destinadas a la explotación o al aprovechamiento de los recursos naturales o del suelo y del subsuelo, así como de las aguas continentales o marinas.

Definido el objeto de la evaluación, la Ley define la finalidad específica de una y otra evaluación según recaiga sobre un plan o un proyecto. La evaluación ambiental estratégica requiere incorporar los criterios de sostenibilidad en la toma de decisiones estratégicas. Ello significa que en la elaboración del plan deben valorarse y tenerse en cuenta los aspectos medioambientales, junto a los económicos y sociales, con el fin de

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alcanzar un elevado grado de protección ambiental y promover el desarrollo sostenible (artículo 1 de la Ley 21/2013).

A través de la evaluación de proyectos, se garantiza una adecuada prevención de los impactos ambientales concretos que se puedan generar, al tiempo que se establecen las medidas necesarias de corrección o compensación de dichos impactos.

La distinción entre ambos tipos de evaluación tiene su reflejo en una diferente tramitación. Así, el procedimiento para evaluar los planes se regula en el artículo 6 y en el Capítulo I del Título II de la Ley 21/2013; y el de los proyectos, en el artículo 7 y en el Capítulo II del Título II.

Centrándonos ahora en la evaluación ambiental estratégica, esta requiere, en síntesis, la elaboración del estudio ambiental estratégico; la celebración de un trámite de información pública y de consultas a las Administraciones públicas afectadas y personas interesadas y una declaración ambiental estratégica en la que se incluyan las determinaciones, medidas o condiciones que deban incorporarse en el plan que finalmente se apruebe o adopte para garantizar su sostenibilidad.

2. Hecha la distinción entre ambas formas de evaluación, para averiguar si el Plan Nacional 5G debe someterse a evaluación ambiental debe comprobarse si cumple los requisitos establecidos en la legislación.

1º Como se ha dicho, el artículo 5.2 b) de la Ley define un plan como el conjunto de estrategias, directrices y propuestas destinadas a satisfacer necesidades sociales, no ejecutables directamente, sino a través de su desarrollo por medio de uno o varios proyectos. De la definición dada por esa Secretaría de Estado para el Avance Digital, a través del informe de la Dirección General de Telecomunicaciones, y del contenido del propio plan se deduce que el Plan Nacional 5G se ajusta a esta definición.

Así según la DG, *"El Plan Nacional 5G tiene como objetivo situar a España entre los países más avanzados en el desarrollo de esta nueva tecnología, de manera que cuando la 5G alcance su madurez tecnológica y comercial, España esté lo mejor preparada posible para aprovechar al máximo las oportunidades de este nuevo paradigma tecnológico.*

El Plan Nacional (…) es un documento que define y trata de impulsar una estrategia de una política pública que va a provocar efectos beneficiosos para la economía y sociedad españolas,...(…) Dicho Plan es únicamente un documento de carácter general en el que se identifican los tipos de medidas, de carácter estratégico, que se considera se deben a llevar a cabo para que España no quede retrasada en el desarrollo de esta nueva tecnología, y pueda perder las oportunidades que proporcionará en el futuro.

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Por ello, este Plan se enmarca en un contexto internacional en el que los principales países europeos (Alemania, Francia, Reino Unido, etc.) y de otras regiones del mundo (USA, Japón, Corea, etc.) están aprobando sus estrategias para el desarrollo del 5G, y España no podía quedarse atrás en estos aspectos. Estas estrategias nacionales, al igual que el Plan de acción 5G para Europa, y el Plan nacional 5G publicado en España, constituyen documentos de referencia en cada uno de los países, en los que se identifican las grandes medidas estratégicas a llevar a cabo para el desarrollo del 5G, pero no se trata de medidas normativas específicas que deban ser objeto para su aprobación y publicación de una evaluación específica y sobre un caso concreto en lo que a su impacto en diferentes aspectos se refiere".

Conforme a lo anterior, el Plan define los objetivos estratégicos, las medidas estratégicas (entre ellas acciones para la gestión y planificación del espectro radioeléctrico, puesta en marcha de proyectos piloto y desarrollo de instrumentos legales que proporcionen seguridad jurídica para facilitar las inversiones en el despliegue) y la hoja de ruta para el despliegue de las acciones clave. De lo anterior se concluye que el Plan Nacional 5G se ajusta sin dificultad a la definición legal y es un plan a los efectos de la Ley 21/2013, pues constituye un conjunto de directrices y propuestas destinadas a satisfacer necesidades sociales, no ejecutables directamente.

2º El artículo 6.1 determina los planes que deben ser sometidos a evaluación ambiental estratégica ordinaria conforme a lo siguiente:

- *Que se adopten o aprueben por una Administración pública.* En este caso, esa Secretaría de Estado para el Avance Digital no ha informado de que el Plan haya sido aprobado formalmente por el Consejo de Ministros mediante un acuerdo o mediante una orden o resolución de un órgano del Ministerio de Economía y Empresa. No obstante, no está del todo claro que la Ley 21/2013 exija un acto de aprobación formal en sentido estricto, pues basta que el plan haya sido "adoptado" por una Administración pública, lo cual ocurre en este caso, pues como acaba de indicarse, y así lo reconoce la Dirección General de Telecomunicaciones, el Plan es un documento de referencia elaborado y difundido por el Ministerio, el cual define los pasos que han de darse y el calendario que ha de cumplirse para el despliegue de la tecnología 5G.

- *Que su elaboración y aprobación venga exigida por una disposición legal o reglamentaria o por acuerdo del Consejo de Ministros o del Consejo de Gobierno de una comunidad autónoma.* Este precepto traspone el artículo 2 a) segundo guión de la Directiva 2001/42/CE de evaluación ambiental de planes y programas. Esta Directiva se refiere a los planes que sean exigidos por disposiciones legales, reglamentarias o administrativas. Conforme a lo anterior, debe interpretarse que las disposiciones que prevén la adopción de un plan no se circunscriben al ámbito estatal o autonómico como dice la Ley 21/2013, sino que su necesidad puede derivarse también de disposiciones o actos emanados de las instituciones comunitarias. Según indica la Dirección General de

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Telecomunicaciones, el Plan Nacional 5G, es consecuencia de la necesidad de cumplir con los objetivos marcados a nivel europeo en el Plan de Acción de 5G para Europa aprobado por las instituciones comunitarias en abril de 2016 "*como estrategia para mejorar la competitividad europea en el desarrollo de esta tecnología emergente, en el que se incluye diversos objetivos para los Estados Miembros, entre ellos, la aprobación de planes de trabajo nacionales*". Efectivamente, el Plan de Acción 5G para Europa fue adoptado por la Comisión Europea mediante la Comunicación 2016 (588). Las comunicaciones son actos atípicos propios del derecho derivado de la UE. Por tanto, el Plan Nacional 5G entra dentro de la categoría de disposiciones a las que hace referencia la Directiva y, por tanto, también cumple el requisito señalado.

- *Que los planes establezcan el marco para la futura autorización de proyectos legalmente sometidos a evaluación de impacto ambiental y se refieran entre otras materias a las telecomunicaciones, a la ordenación del territorio urbano y rural, o del uso del suelo*. La materia objeto del Plan se encuadra dentro de la categoría "telecomunicaciones" y obviamente establece el marco para la autorización de proyectos, pues tal y como se desprende de la información suministrada por la Dirección General de Telecomunicaciones, el Plan Nacional 5G de España prevé la realización de proyectos piloto para probar las nuevas funcionalidades que ofrecerá la tecnología 5G. En particular, al amparo de Plan Nacional 5G, se han convocado 2 proyectos piloto por la entidad pública Red.es, que han sido adjudicados por dicha entidad mediante Resolución del 30 de abril de 2019. Por tanto, el Plan sirve de marco para la autorización de proyectos.

La Ley establece otros requisitos alternativos para planes que también deben someterse a evaluación, por ejemplo, que afecten de forma apreciable a espacios Red Natura 2000.

A la vista de las dudas que pueden suscitarse sobre la concurrencia de los requisitos necesarios para efectuar dicha evaluación, resulta significativo que esa Secretaría de Estado no consultara en su momento al órgano ambiental acerca de la procedencia de tramitar dicha evaluación. Debe recordarse que la Administración sirve con objetividad los intereses generales y actúa de acuerdo con los principios de eficacia y coordinación, con sometimiento pleno a la ley y al Derecho, lo cual habilita el órgano sustantivo para consultar al órgano ambiental si existen dudas sobre la procedencia de aplicar la Ley 21/2013, cuyos preceptos vinculan a todas las administraciones públicas. El artículo 2 h) de la Ley establece un principio de colaboración activa entre los órganos que intervienen en el proceso de evaluación; y según el artículo 3.1, las Administraciones que puedan estar interesadas en el plan, debido a sus responsabilidades medioambientales, serán consultadas sobre la información proporcionada por el promotor y sobre la solicitud de adopción o aprobación de un plan. Finalmente, la Ley prevé la posibilidad de que el órgano ambiental decida someter a una evaluación simplificada un plan cuando se cumplan los requisitos del Anexo V de la Ley 21/2013 entre los que se incluyen los riesgos para la salud humana o el medio

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ambiente y la magnitud y el alcance espacial de los efectos (área geográfica y tamaño de la población que puedan verse afectadas). Para ello debe tener conocimiento del Plan, el cual debe ser remitido por el órgano sustantivo y promotor del Plan.

También debe recordarse que la necesidad de someter el Plan Nacional 5G a este tipo de evaluación se alegó por varias asociaciones en el trámite de información pública del Plan. Esta institución no tiene constancia, pues esa Secretaría de Estado no ha procedido a informar de ello, de cómo se han valorado dichas alegaciones ni si se ha dado una respuesta motivada acerca de la manera en que se han tenido en cuenta en la adopción del Plan. Hay que destacar que una de las finalidades de la evaluación ambiental estratégica es garantizar la participación pública en la toma de decisiones y garantizar que la variable ambiental se tiene en cuenta adecuadamente en ese proceso (artículos 2 i) y k), 17.1 d) y siguientes de la Ley 21/2013).

Ello exige que esa Administración incluya el resultado de la información pública y la manera en que se han tenido en consideración las alegaciones tanto en el análisis técnico del expediente como en la declaración ambiental estratégica, en los términos establecidos en los artículos 24 y 25 de la Ley 21/2013 y 3.2 y conexos de la Ley 27/2006 de acceso a la información, participación pública y acceso a la justicia en materia de medio ambiente. Además, el órgano sustantivo debe publicar en el Boletín Oficial del Estado, junto con la adopción del Plan, un extracto que incluya los siguientes aspectos: 1º De qué manera se han integrado en el plan los aspectos ambientales; y 2º Cómo se ha tomado en consideración en el plan, entre otras cuestiones, los resultados de la información pública y de las consultas.

En el documento en el que resume el trámite de información pública, disponible en la página web del Ministerio de Economía y Empresa, no se da respuesta a las alegaciones planteadas sobre la necesidad de someter el Plan Nacional 5G a evaluación. Así, aunque se haya celebrado un trámite de información pública, la ausencia de valoración de alegaciones y de información al público de cómo se han tenido en cuenta esas alegaciones, la participación pública en el proceso de toma de decisiones ha resultado incompleta.

Tampoco esa Secretaría de Estado ha informado de que diera traslado al órgano ambiental de esas alegaciones. Con esta forma de proceder también ha ignorado lo establecido en el artículo 3.4 de la Ley 21/2013, según el cual, el órgano sustantivo debe informar al órgano ambiental de cualquier incidencia que se produzca durante la tramitación del procedimiento administrativo sustantivo de adopción de un plan que tenga relevancia a los efectos de la tramitación de la evaluación ambiental.

3. Las consideraciones anteriores conducen a la conclusión de que el Plan Nacional 5G no se sometió a evaluación ambiental estratégica y que dicha decisión se adoptó unilateralmente por el órgano sustantivo (y promotor del Plan) sin consultar con el órgano ambiental sobre la procedencia de efectuar dicha evaluación, aunque el Plan

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podía cumplir los requisitos exigidos de la Ley 21/2013, en incluso podrían suscitarse dudas en relación con la falta de aprobación formal.

Respecto a esta última cuestión (la necesidad de aprobación formal de los planes) debe manifestarse que si bien no es irrazonable interpretar que los planes deben ser aprobados por la Administración que lo elabora (u otra) para ser evaluados ambientalmente, también es cierto que por la vía de no dictar una norma o un acto que apruebe formalmente un plan, podría eludirse fácilmente la evaluación, incluso de planes que cumplieran los demás requisitos señalados por la Ley 21/2013. En el caso del Plan Nacional 5G se da la contradicción de que siendo indispensable para "*situar a nuestro entre los países más avanzados en el desarrollo de esta nueva tecnología de manera que cuando la 5G alcance su madurez tecnológica y comercial, España esté preparada para aprovechar al máximo las oportunidades de este paradigma tecnológico*", no haya sido objeto de aprobación formal y que la Dirección General de Telecomunicaciones afirme que carece de efectos vinculantes, aunque su contenido se está cumpliendo.

En todo caso, existen planes estatales (por ejemplo, planes de infraestructuras), con una naturaleza, finalidad y estructura similar al Plan Nacional 5G (aunque más detallados y completos), que se han aprobado formalmente y que han sido sometidos a evaluación ambiental estratégica. Esa Secretaría de Estado sin embargo, no ha explicado las razones de la falta de aprobación formal del Plan Nacional 5G, pese a la importancia que atribuye al cumplimiento de su contenido para alcanzar los objetivos propuestos.

4. En todo caso, tener en cuenta las consideraciones ambientales en la toma de decisiones es una exigencia del desarrollo sostenible y, por tanto, los efectos ambientales de las decisiones públicas deben valorarse aunque para ello no se siga un procedimiento reglado. El artículo 3 del Tratado de la Unión Europea incluye entre sus objetivos el establecimiento de "un nivel elevado de protección y mejora de la calidad del medio ambiente". Y en el mismo sentido, el artículo 11 del Tratado de Funcionamiento de la UE señala que "las exigencias de la protección del medio ambiente deberán integrarse en la definición y en la realización de las políticas y acciones de la Unión, en particular con objeto de fomentar un desarrollo sostenible". A la luz de lo anterior, puede concluirse que la Secretaría de Estado no ha tenido en cuenta las consideraciones ambientales a la hora de decidir y aprobar el Plan Nacional 5G.

Esta institución no puede deducir del contenido del Plan Nacional 5G si este afecta o no apreciablemente al medio ambiente. Sin perjuicio de lo referido a las emisiones radioeléctricas (sobre lo que se volverá más adelante), la ausencia de información sobre los lugares de despliegue de nuevas infraestructuras, sobre posibles afecciones a espacios protegidos, o al paisaje, o al uso del suelo, ponen en evidencia que la variable ambiental no se ha tomado en consideración. Sin embargo, no puede obviarse que propio Plan Nacional 5G señala que "el desarrollo de los servicios 5G supondrá el despliegue masivo de nuevos elementos de red en el territorio español, ya

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sea en *nuevos emplazamientos* o en los emplazamientos que se utilizan para otras tecnologías y servicios". Por ello, hubiera sido deseable un mayor grado de definición de la estrategia de despliegue del tecnología y, como se ha dicho, que se hubiera recabado el criterio del órgano ambiental respecto a la necesidad de practicar una evaluación reglada del Plan. Incluso en el caso de que la afección ambiental del Plan Nacional 5G no sea significativa, la valoración de la variable ambiental debería haberse incluido en el proceso de adopción del Plan y haberse tomado en consideración, al menos para justificar que no era necesaria una evaluación ambiental estratégica, conforme a los requisitos exigidos por la Ley 21/2013.

5. Señalado lo anterior, existe escaso margen de maniobra para subsanar la falta de evaluación y de valoración de los aspectos ambientales en la adopción del Plan nacional 5G. Por un lado, el artículo 9 de la Ley 21/2013, determina la falta de validez de los planes que han omitido la evaluación ambiental estratégica preceptiva. Sin embargo, puesto que el Plan Nacional 5G no ha sido objeto de aprobación formal y carece de efectos jurídicos vinculantes, la falta de validez pierde su sentido en este caso. Además, el Plan diseña las directrices de actuación hasta 2020, de manera que el ámbito temporal de la planificación está próximo a su fin. Dada esta situación, esta institución solo puede concluir sobre este punto que el Plan Nacional no ha tenido en consideración los aspectos ambientales del Plan y no los ha valorado ni siquiera a efectos de justificar que no era exigible una evaluación reglada conforme a la Ley 21/2013.

6. En relación con la evaluación de impacto ambiental de los proyectos piloto adjudicados, debe señalarse que esa Secretaría de Estado no se ha referido en ningún momento al artículo 7 de la Ley 21/2013 en el que se establecen los requisitos de evaluación de impacto ambiental proyectos. Conforme a este artículo, deben ser objeto de evaluación de impacto ambiental ordinaria los comprendidos en el anexo I; y deben ser objeto de evaluación de impacto ambiental simplificada, entre otros, cualquier proyecto que pueda afectar de forma apreciable a lugares de la Red Natura 2000 que no esté incluido en el anexo I.

De la información disponible sobre los dos proyectos piloto adjudicados en Andalucía y Galicia, no parece que se incluyan en alguno de los supuestos enumerados en el anexo I de la Ley. Por otro lado parece que las actuaciones se van a desarrollar en entornos urbanos, en parques tecnológicos, en instalaciones portuarias y otros entornos altamente antropizados, de manera que es posible que los proyectos piloto no afecten apreciablemente a lugares de la Red Natura 2000. No obstante, ello es una mera suposición, dada la falta total de valoración de los efectos ambientales de los proyectos por parte del órgano sustantivo o del órgano ambiental, incluso para justificar que dicha evaluación no es necesaria, de acuerdo con lo exigido por la Ley 21/2013.

Debe destacarse que para justificar la falta de evaluación de los proyectos piloto, esa Secretaría de Estado se refiere en todo momento a lo dispuesto en la Ley 9/2014 General de Telecomunicaciones y no a los requisitos exigidos en la Ley 21/2013.

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Respecto a esta cuestión es necesario aclarar que la exención de *licencia ambiental* que efectúa la Ley General de Telecomunicaciones respecto a los proyectos en esta materia no alcanza al procedimiento de evaluación de impacto ambiental, que será obligatoria cuando los proyectos reúnan los requisitos establecidos en la Ley 21/2013.

La legislación estatal en materia de evaluación ambiental es transposición de una Directiva comunitaria de obligado cumplimiento que no ampara que las normas excluyan una determinada categoría de proyectos del deber de evaluar si estos pudieran tener efectos negativos y apreciables sobre el medio ambiente. Es decir, por aplicación de la Ley de Telecomunicaciones no puede argumentarse *a priori* que ningún proyecto en la materia tiene efectos apreciables sobre el medio ambiente y, que por tanto, en ningún caso será exigible la evaluación que regula la Ley 21/2013. Al contrario, los proyectos en materia de telecomunicaciones deberán someterse a evaluación de impacto ambiental reglada si cumplen los requisitos establecidos en la Ley 21/2013; y no deberán someterse si no los cumplen.

Otra cosa distinta es que, caso por caso, la Administración decida motivadamente que un proyecto concreto no debe ser sometido a evaluación de impacto ambiental antes de su autorización (aunque reúna los requisitos legales para ello), cuando se dé alguno de los supuestos regulados en el artículo 8 de la Ley 21/2013, es decir, proyectos en materia de defensa, emergencia civil, infraestructuras críticas etcétera. Pero incluso en estos casos es preciso que el órgano sustantivo valore si es necesario someter el proyecto a otra forma alternativa de evaluación y que dicha valoración se ponga a disposición del público y se comunique a la Unión Europea antes de su aprobación.

7. En relación con el estado del conocimiento científico sobre los efectos de la tecnología 5G en la salud, debe reiterarse que esta institución confía en la información suministrada por las autoridades científicas y sanitarias. No obstante, también debe señalarse lo siguiente:

1º Sin perjuicio de la Recomendación 1999/519CE del Consejo de Sanidad de la UE, España es también miembro del Consejo de Europa y debe seguir las recomendaciones que aprueba, entre ellas la Recomendación 1815 (2011) sobre los peligros potenciales de los campos electromagnéticos y sus efectos sobre el medio ambiente. En esa Recomendación se insta a los Estados miembros, entre otras cosas, a tener en consideración los principios de precaución y ALARA (mantener los niveles de exposición tan bajos como sea posible); a adoptar todas las medidas razonables para reducir la exposición a los CEM, en particular de niños y jóvenes; a prestar especial atención a las personas electrosensibles que sufren síndrome de intolerancia a los campos electromagnéticos; a llevar a cabo los procedimientos de evaluación de riesgos apropiados y mejorar los estándares de evaluación; y a mantener las instalaciones eléctricas a una distancia segura de las viviendas. La Recomendación requiere por tanto que determinados colectivos vulnerables tengan una consideración específica más allá de los niveles de exposición establecidos para el conjunto de la población. Dicha

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atención específica no se encuentra ni en el Plan Nacional 5G ni en la adjudicación de los proyectos piloto.

Si bien esta institución comparte el criterio de que no deben imponerse límites arbitrarios, desautorizando o desconfiando de la regulación existente basada en el conocimiento científico, tampoco la Administración ha justificado concluyentemente que no deba atenderse la Recomendación del Consejo de Europa de prestar especial atención a determinados colectivos vulnerables.

2º Esa Secretaría de Estado dice que, en lo que se refiere a los límites de las emisiones radioeléctricas, estos límites no dependen de la tecnología utilizada (4G o 5G) sino de las características específicas de cada banda de frecuencias; y que las bandas que se van a utilizar para la tecnología 5G van a ser las que ya se utilizan a nivel masivo en España, con la única excepción de la banda de 26 GHz. Esta banda, según afirma esa Administración, aún no ha sido adjudicada para el uso de 5G para servicios comerciales masivos; y los organismos internacionales de normalización ya están trabajando en una norma técnica que cubra el cálculo y procedimiento de medida de emisiones en la banda de 26 GHz.

Esta información ratifica lo señalado por el Comité Científico sobre Radiofrecuencias y Salud (CCARS) en su informe en esta materia para 2013-2016 en el que se refiere expresamente a la ausencia de datos sobre límites de exposición de la tecnología 5G en bandas superiores a 6GHz "*pues están aún en proceso de discusión, definición y posterior estandarización, la cual no se espera cerrar antes del año 2020.*".

Dicho lo anterior, debe señalarse que si bien la utilización de la banda de 26 GHz puede no ser masiva, como dice esa Secretaría de Estado, esta institución ha podido comprobar que las bases reguladoras de la adjudicación de los proyectos piloto contempla la utilización de la banda de 26 GHz y esta se valora "*particularmente*" en los criterios de selección de proyectos (artículo 13 b) y anexo III) de la Orden ECE/1016/2018, de 28 de septiembre, por la que se establecen las bases reguladoras de la concesión de subvenciones a proyectos piloto de tecnología 5G).

Además, en los proyectos piloto hasta ahora adjudicados en Andalucía y Galicia, según la información suministrada por Red.es (una entidad pública empresarial del Ministerio de Economía y Empresa que depende de esa Secretaría de Estado) se prevé que el despliegue de la tecnología 5G se realice en las bandas 3.7 y 26 GHz. Ello significa que se va a utilizar una banda –la de 26 GHz- para la cual aún no se han fijado los niveles de exposición seguros.

Este supuesto es el ámbito propio de la aplicación del principio de precaución o cautela. Según este principio, cuando una actividad representa una amenaza o un daño para la salud humana o el medio ambiente, hay que tomar medidas de precaución incluso cuando la relación causa-efecto no haya podido demostrarse científicamente de forma concluyente.

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El principio de precaución se menciona en el artículo 191 del Tratado de Funcionamiento de la Unión Europea con el fin de garantizar un elevado nivel de protección del medio ambiente aunque su ámbito de aplicación es más amplio y se extiende a la salud humana, animal y vegetal.

Por su parte, el Real Decreto 1066/2001, por el que se aprueba el Reglamento que establece condiciones de protección del dominio público radioeléctrico, restricciones a las emisiones radioeléctricas y medidas de protección sanitaria frente a emisiones radioeléctricas, alude a este principio en el artículo 7, cuando impone al Ministerio de Sanidad el deber de adaptar al progreso científico los límites de exposición a las emisiones radioeléctricas, teniendo en cuenta el principio de precaución y las evaluaciones realizadas por las organizaciones nacionales e internacionales competentes.

La Comunicación de la Comisión sobre el recurso al principio de precaución (COM (2000) 1 final de 2.2.2000), señala que las medidas basadas en el principio de precaución deberán ser proporcionales al nivel de protección elegido, no discriminatorias en su aplicación, coherentes con medidas similares ya adoptadas, basadas en el examen de los posibles beneficios y los costes de la acción o de la falta de acción, estar sujetas a revisión, a la luz de los nuevos datos científicos, y ser capaces de designar a quién incumbe aportar las pruebas científicas necesarias para una evaluación del riesgo más completa.

Obviamente, el análisis de estos aspectos y del potencial riesgo para la salud del empleo de la tecnología 5G a través de la banda de 26 GHz excede las funciones de esta institución y requiere la intervención de órganos y administraciones públicas distintas de esa Secretaría de Estado, entre ellas las sanitarias. La disposición adicional décima de la Ley de Telecomunicaciones prevé la creación de la Comisión Interministerial sobre Radiofrecuencias y Salud que debe ser el órgano, a nivel estatal, apto para abordar estas cuestiones de manera coordinada. La Comisión tiene por función asesorar e informar a la ciudadanía, al conjunto de las administraciones públicas y a los diversos agentes de la industria sobre las restricciones establecidas a las emisiones radioeléctricas, las medidas de protección sanitaria aprobadas frente a emisiones radioeléctricas y los múltiples y periódicos controles a que son sometidas las instalaciones generadoras de emisiones radioeléctricas, en particular, las relativas a las radiocomunicaciones.

Asimismo, dicha Comisión debe realizar y divulgar estudios e investigaciones sobre las emisiones radioeléctricas y sus efectos y cómo las restricciones a las emisiones, las medidas de protección sanitaria y los controles establecidos preservan la salud de las personas, así como, a la vista de dichos estudios e investigaciones, realizará propuestas y sugerirá líneas de mejora en las medidas y controles a realizar.

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De la Comisión interministerial formarán parte en todo caso, además del órgano competente en materia de telecomunicaciones, el Ministerio de Sanidad y el Instituto de Salud Carlos III. Contará además con un grupo asesor o colaborador en materia de radiofrecuencias y salud, con participación de comunidades autónomas, de la asociación de entidades locales de ámbito estatal con mayor implantación y un grupo de expertos independientes, sociedades científicas y representantes de los ciudadanos, para hacer evaluación y seguimiento periódico de la prevención y protección de la salud de la población en relación con las emisiones radioeléctricas, proponiendo estudios de investigación, medidas consensuadas de identificación, elaboración de registros y protocolos de atención al ciudadano.

Dicha Comisión aún no se ha constituido a pesar de haberlo sugerido esta institución al entonces Ministerio de Sanidad, Servicios Sociales e Igualdad en el año 2017 y sin que dicho departamento haya ofrecido razones que justifiquen desatender el mandato de la Ley. A la vista de las consideraciones expuestas, en particular el despliegue de la tecnología 5G a través de una banda para la que no se han establecido niveles de exposición a emisiones radioeléctricas seguras, su constitución resulta ineludible, con el fin de que se pronuncie sobre la aplicación del principio de precaución en este caso.

A juicio de esta institución no puede ignorarse que existe una preocupación ciudadana por los efectos del despliegue de esta nueva tecnología y que sus reclamaciones sobre los efectos de los campos electromagnéticos en la salud empiezan a ser atendidas por los tribunales de justicia, los cuales reconocen, en virtud de los informes médicos aportados, determinadas patologías por exposición a emisiones radioeléctricas.

La Administración puede estar convencida de la inocuidad de las emisiones cuando se respetan los valores de seguridad fijados por la Comisión Internacional para la Protección ante Radiaciones No Ionizantes (ICNIRP), por debajo de los cuales no se han reproducido efectos biológicos en las personas. Pero ello no le exime de poner en marcha los mecanismos previstos en la legislación en materia de investigación, asesoramiento, participación e información pública, ni de valorar y aplicar las medidas de precaución necesarias para asegurarse de que el despliegue de la nueva tecnología no suponga perjuicios para la salud, especialmente cuando aún no se han determinado los niveles de exposición segura para una determinada banda de frecuencia, cuyo uso, sin embargo, se ha autorizado.”.

En virtud de las consideraciones expuestas, y de conformidad con los artículos 28 y 30 de la Ley Orgánica 3/1981, de 6 de abril, el Defensor del Pueblo ha resuelto dirigir a la Secretaría de Estado para el Avance Digital las siguientes resoluciones:

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RECORDATORIO DE DEBERES LEGALES

"Someter los planes y proyectos en materia de telecomunicaciones a evaluación ambiental estratégica y evaluación de impacto ambiental respectivamente, cuando reúnan los requisitos establecidos en la Ley 21/2013 de evaluación ambiental".

SUGERENCIAS

1. **"Elaborar, conjuntamente con el Ministerio de Sanidad, Consumo y Bienestar Social el proyecto de reglamento por el que debe regularse la Comisión Interministerial sobre Radiofrecuencias y Salud y, tras cumplimentar los trámites preceptivos, elevarlo al Consejo de Ministros para su aprobación.**

2. **Una vez constituida, someter a consulta de la Comisión la forma de proceder respecto a la aplicación del principio de precaución en el desarrollo de proyectos que impliquen el uso de la banda de 26 GHz, en tanto no se determinen los límites seguros de exposición a emisiones radioeléctricas exigibles para dicha frecuencia".**

Asimismo se ha solicitado a la Secretaría de Estado que informe de las medidas adoptadas para evaluar los posibles efectos sobre la salud que pudieran derivarse de los proyectos piloto en Andalucía y Galicia; si se ha consultado a dichas Comunidades Autónomas sobre estos proyectos; y si se han adjudicado o se van a adjudicar otros nuevos durante el ámbito de aplicación temporal del Plan Nacional 5G.

De la respuesta que a dichas resoluciones se reciba, se le informará, así como de las actuaciones que procedan.

Le saluda muy atentamente,

Francisco Fernández Marugán
Defensor del Pueblo (e.f.)

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BILAG 5

Faktaboks med overblik over Johansens industriarbejde

▪ **1994/95:** Modtager forskningsmidler fra teleindustrien – Sonofon og Tele Danmark Mobil – til en abonnentsdatabase, der anvendes til det såkaldte Interphone-studie. Den danske del af studiet, "The Danish Cohort", kritiseres for selve anvendelsen af kontrolgruppen, hvori erhvervsbrugere indgår sammen med borgere, der ikke bruger mobiltelefon.
▪ **1994-2004:** Modtager midler fra Dansk Energi. Bestilt forskning vedrører skadeligheden af højspændingsmaster og børn med bopæl tæt herpå samt de ansatte ved elselskaberne.
▪ **1994-2007:** Modtager tre personlige honorarer fra COWI. Disse har også relation til højspænding.
▪ **1994-2010:** Modtager fire personlige honorarer af ukendt størrelse fra Energinet.dk, en interesseorganisation for danske energileverandører, heriblandt elselskaber. Hans honorarer uddeles for konsulentbistand til VVM-rapporter om højspænding, fremgår det.
▪ **2006:** Modtager fornyet forskningsbevilling fra Energinet.dk. .
▪ **2017, efterår:** Indgår ny kontrakt med COWI og orienterer SST herom. Emnet er rådgivning inden for elektromagnetiske felter, som han også rådgiver Sundhedsstyrelsen om.

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Adverse health effects of 5G mobile networking technology under real-life conditions

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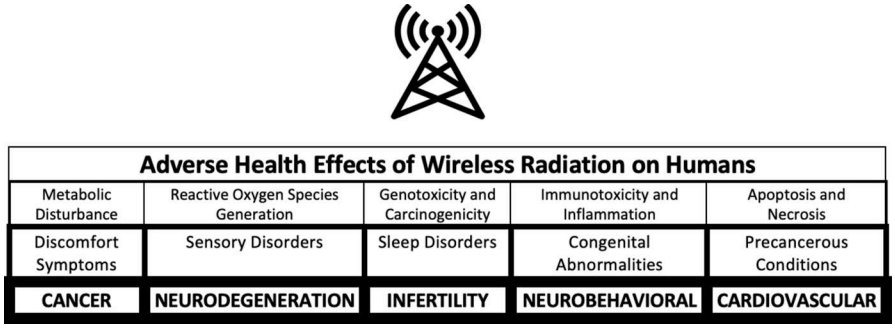
GRAPHICAL ABSTRACT



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ABSTRACT

This article identifies adverse effects of non-ionizing non-visible radiation (hereafter called wireless radiation) reported in the premier biomedical literature. It emphasizes that most of the laboratory experiments conducted to date are not designed to identify the more severe adverse effects reflective of the real-life operating environment in which wireless radiation systems operate. Many experiments do not include pulsing and modulation of the carrier signal. The vast majority do not account for synergistic adverse effects of other toxic stimuli (such as chemical and biological) acting in concert with the wireless radiation. This article also presents evidence that the nascent 5G mobile networking technology will affect not only the skin and eyes, as commonly believed, but will have adverse systemic effects as well.

1. Introduction

Wireless communications have been expanding globally at an exponential rate. The latest imbedded version of mobile networking technology is called 4G (fourth generation), and the next version (called 5G- fifth generation) is in the early implementation stage. Neither 4G

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nor 5G have been tested for safety in credible real-life scenarios. Alarmingly, many of the studies conducted in more benign environments show harmful effects from this radiation. The present article overviews the medical and biological studies that have been performed to date relative to effects from wireless radiation, and shows why these studies are deficient relative to safety. However, even in the absence of the missing real-life components such as toxic chemicals and biotoxins (which tend to exacerbate the adverse effects of the wireless radiation), the literature shows there is much valid reason for concern about potential adverse health effects from both 4G and 5G technology. The studies on wireless radiation health effects reported in the literature should be viewed as extremely conservative, substantially underestimating the adverse impacts of this new technology.

2. Wireless radiation/electromagnetic spectrum

This section overviews the electromagnetic spectrum, and delineates the parts of the spectrum on which this article will focus. The electromagnetic spectrum encompasses the entire span of electromagnetic radiation, including:

- ionizing radiation (gamma rays, x-rays, and the extreme ultraviolet, with wavelengths below $\sim 10^{-7}$ m and frequencies above $\sim 3 \times 10^{15}$ Hz);
- non-ionizing visible radiation (wavelengths from $\sim 4 \times 10^{-7}$ m to $\sim 7 \times 10^{-7}$ m and frequencies between $\sim 4.2 \times 10^{14}$ Hz and $\sim 7.7 \times 10^{14}$ Hz);
- non-ionizing non-visible radiation

short wavelength radio waves and microwaves, with wavelengths between $\sim 10^{-3}$ m and $\sim 10^{5}$ m and frequencies between $\sim 3 \times 10^{11}$ to $\sim 3 \times 10^{3}$ Hz;

long wavelengths, ranging between $\sim 10^{5}$ m and $\sim 10^{8}$ m and frequencies ranging between 3×10^{3} and 3 Hz.

How are these frequencies used in practice?

- The low frequencies (3 Hz – 300 KHz) are used for electrical power line transmission (60 Hz in the U.S.) as well as maritime and submarine navigation and communications.
- Medium frequencies (300 KHz–900 MHz) are used for AM/FM/TV broadcasts in North America.
- Lower microwave frequencies (900 MHz – 5 GHz) are used for telecommunications such as microwave devices/communications, radio astronomy, mobile/cell phones, and wireless LANs.
- Higher microwave frequencies (5 GHz – 300 GHz) are used for radar and proposed for microwave WiFi, and will be used for high-performance 5 G.
- Terahertz frequencies (300 GHz – 3000 GHz) are used increasingly for imaging to supplement X-rays in some medical and security scanning applications (Kostoff and Lau, 2017).

In the present study of wireless radiation health effects, the frequency spectrum ranging from 3 Hz to 300 GHz is covered, with particular emphasis on the high frequency communications component ranging from ~1 GHz to ~300 GHz. Why was this part of the spectrum selected? Previous reviews of wireless radiation health effects found that pulsed electromagnetic fields (PEMF) applied for relatively short periods of time could sometimes be used for therapeutic purposes, whereas chronic exposure to electromagnetic fields (EMF) in the power frequency range (~60 Hz) and microwave frequency range (~1 GHz-tens GHz) tended to result in detrimental health effects (Kostoff and Lau, 2013, 2017). Given present concerns about the rapid expansion of 5G communications systems (which are projected to use mainly the higher microwave frequencies part of the spectrum in the highest performance (aka high-band) mode) in the absence of adequate and rigorous safety testing, more emphasis will be placed on the communications frequencies in this document.

3. Modern wireless radiation exposures

In ancient times, sunlight and its lunar reflections provided the bulk of the visible spectrum for human beings (with fire a distant second and lightning a more distant third). Now, many varieties of artificial light (incandescent, fluorescent, and light emitting diode) have replaced the sun as the main supplier of visible radiation during waking hours. Additionally, EMF radiations from other parts of the non-ionizing non-visible spectrum have become ubiquitous in daily life, such as from wireless computing and telecommunications. In the last two or three decades, the explosive growth in the cellular telephone industry has placed many residences in metropolitan areas within less than a mile of a cell tower. Future implementation of the next generation of mobile networking technology, 5 G, will increase the cell tower densities by an order of magnitude. Health concerns have been raised about wireless radiation from (1) mobile communication devices, (2) occupational exposure, (3) residential exposure, (4) wireless networks in homes, businesses, and schools, (5) automotive radar, and (6) other non-ionizing EMF radiation sources, such as ‘smart meters’ and ‘Internet of Things’.

4. Demonstrated biological and health effects from prior generations of wireless networking technology

There have been two major types of studies performed to ascertain biological and health effects of wireless radiation: laboratory and epidemiology. The laboratory tests performed provided the best scientific understanding of the effects of wireless radiation, but did not reflect the real-life environment in which wireless radiation systems operate (exposure to toxic chemicals, biotoxins, other forms of toxic radiation, etc). There are three main reasons the laboratory tests failed to reflect real-life exposure conditions for human beings.

First, the laboratory tests have been performed mainly on animals, especially rats and mice. Because of physiological differences between small animals and human beings, there have been continual concerns about extrapolating small animal results to human beings. Additionally, while inhaled or ingested substances can be scaled from laboratory experiments on small animals to human beings relatively straight-forwardly, radiation may be more problematic. For non-ionizing radiation, penetration depth is a function of frequency, tissue, and other parameters. Radiation could penetrate much deeper into a small animal’s interior than similar wavelength radiation in humans, because of the much smaller animal size. Different organs and tissues would be affected, with different levels of power density.

Second, the typical incoming EMF signal for many/most laboratory tests performed in the past consisted of single carrier wave frequency; the lower frequency superimposed signal containing the information was not always included. This omission may be important. As Panagopoulos states: “It is important to note that except for the RF/microwave carrier frequency, Extremely Low Frequencies - ELFs (0–3000 Hz) are always present in all telecommunication EMFs in the form of pulsing and modulation. There is significant evidence indicating that the effects of telecommunication EMFs on living organisms are mainly due to the included ELFs…. While ~50 % of the studies employing simulated exposures do not find any effects, studies employing real-life exposures from commercially available devices display an almost 100 % consistency in showing adverse effects” (Panagopoulos, 2019). These effects may be exacerbated further with 5 G: “with every new generation of telecommunication devices…..the amount of information transmitted each moment…..is increased, resulting in higher variability and complexity of the signals with the living cells/ organisms even more unable to adapt” (Panogopoulos, 2019).

Third, these laboratory experiments typically involved one stressor

(toxic stimulus) and were performed under pristine conditions. This contradicts real-life exposures, where humans are exposed to multiple toxic stimuli, in parallel or over time (Tsatsakis et al., 2016, 2017; Docea et al., 2019a). In perhaps five percent of the cases reported in the wireless radiation literature, a second stressor (mainly a biological or chemical toxic stimulus) was added to the wireless radiation stressor, to ascertain whether additive, synergistic, potentiative, or antagonistic effects were generated by the combination (Kostoff and Lau, 2013, 2017; Juutilainen, 2008; Juutilainen et al., 2006).

Combination experiments are extremely important because, when other toxic stimuli are considered in combination either with each other or with wireless radiation, the synergies tend to enhance the adverse effects of each stimulus in isolation. This was shown in several studies that evaluated the cumulative effects of chronic exposure to low doses of xenobiotics in combination (Kostoff et al., 2018; Docea et al., 2018; Tsatsakis et al., 2019a; Docea et al., 2019b; Tsatsakis et al., 2019b, c; Fountoucidou et al., 2019). For those combinations that include wireless radiation, combined exposure to toxic stimuli and wireless radiation translates into much lower levels of tolerance for each toxic stimulus in the combination relative to its exposure levels that produce adverse effects in isolation. Accordingly, the exposure limits for wireless radiation when examined in combination with other potentially toxic stimuli would be far lower for safety purposes than those derived from wireless radiation exposures in isolation.

Thus, almost all of the wireless radiation laboratory experiments that have been performed to date are flawed/limited with respect to showing the full adverse impact of the wireless radiation that would be expected under real-life conditions. Either 1) non-inclusion of signal information or 2) using single stressors only tends to underestimate the seriousness of the adverse effects from wireless radiation. Excluding ***both*** of these phenomena from experiments, as was done in the vast majority of the reported wireless radiation health effects studies, tends to amplify this underestimation substantially. Thus, the results reported in the biomedical literature should be viewed as 1) extremely conservative and 2) the very low 'floor' of the seriousness of the adverse effects from wireless radiation, not the 'ceiling'.

In contrast to the controlled pristine environments that characterize the wireless radiation animal laboratory experiments, the wireless radiation epidemiology studies carried out to date typically involved human beings who had been subjected to myriad known and unknown stressors prior to (and during) the study. The real-life human exposure levels from cell tower studies (reported by Kostoff and Lau (2017)) that showed increased cancer incidence were orders of magnitude lower than those exposure levels generated in the recent highly-funded National Toxicology Program animal laboratory studies (Melnick, 2019). We believe the inclusion of real-world effects in the cell tower studies accounted for the orders of magnitude exposure level decreases that were associated with the increased cancer incidence. The laboratory tests were conducted under controlled conditions not reflective of real-life, while the epidemiology studies were performed in the presence of many stressors, known and unknown, reflective of real-life. The myriad toxic stimuli exposure levels of the epidemiology studies were, for the most part, uncontrolled.

A vast literature published over the past sixty years shows adverse effects from wireless radiation applied in isolation or as part of a combination with other toxic stimuli. Extensive reviews of wireless radiation-induced biological and health effects have been published (Kostoff and Lau, 2013, 2017; Belpomme et al., 2018; Desai et al., 2009; Di Ciaula, 2018; Doyon and Johansson, 2017; Havas, 2017; Kaplan et al., 2016; Lerchl et al., 2015; Levitt and Lai, 2010; Miller et al., 2019; Pall, 2016, 2018; Panagopoulos, 2019; Panagopoulos et al., 2015; Russell, 2018; Sage and Burgio, 2018; van Rongen et al., 2009; Yakymenko et al., 2016; Bioinitiative, 2012). In aggregate, for the high frequency (radiofrequency-RF) part of the spectrum, these reviews show that RF radiation ***below the FCC guidelines*** can result in:

- carcinogenicity (brain tumors/glioma, breast cancer, acoustic neuromas, leukemia, parotid gland tumors),
- genotoxicity (DNA damage, DNA repair inhibition, chromatin structure),
- mutagenicity, teratogenicity,
- neurodegenerative diseases (Alzheimer's Disease, Amyotrophic Lateral Sclerosis),
- neurobehavioral problems, autism, reproductive problems, pregnancy outcomes, excessive reactive oxygen species/oxidative stress, inflammation, apoptosis, blood-brain barrier disruption, pineal gland/melatonin production, sleep disturbance, headache, irritability, fatigue, concentration difficulties, depression, dizziness, tinnitus, burning and flushed skin, digestive disturbance, tremor, cardiac irregularities,
- adverse impacts on the neural, circulatory, immune, endocrine, and skeletal systems.

From this perspective, RF is a highly pervasive cause of disease!

The response from industry has been that no mechanism could explain the biological action of non-thermal and non-ionizing EM fields. Yet, reports of clear perturbations of biological systems at levels near or even below 1000 μW/m^2 (Bioinitiaive, 2019) were explained by perturbations in electron and proton transfers supporting ATP production in mitochondria (Sanders et al., 1980; 1985) exposed to RF or ELF signals (Li and Heroux, 2014).

To obtain another perspective on the full spectrum of adverse effects from wireless radiation, a query was run on Medline to retrieve representative records associated with adverse EMF effects (mainly, but not solely, RF). Over 5400 records were retrieved, and the leading Medical Subject Headings (MeSH) extracted. The categories of adverse impacts from both approaches match quite well. The adverse health effects range from myriad feelings of discomfort to life-threatening diseases.

The full list of MeSH Headings associated with this retrieval is shown in Appendix 1 of (Kostoff, 2019). The interested reader can ascertain what other diseases/symptoms were included. The 5400+ references retrieved are shown in Appendix 2 of (Kostoff, 2019).

5. What types of biological and health effects can be expected from 5G wireless networking technology?

The potential 5G adverse effects derive from the intrinsic nature of the radiation, and its interaction with tissue and target structures. 4G networking technology was associated mainly with carrier frequencies in the range of ~1-2.5 GHz (cell phones, WiFi). The wavelength of 1 GHz radiation is 30 cm, and the penetration depth in human tissue is a few centimeters. In its highest performance (high-band) mode, 5G networking technology is mainly associated with carrier frequencies at least an order of magnitude greater than the 4G frequencies, although, as stated previously, "ELFs (0–3000 Hz) are always present in all telecommunication EMFs in the form of pulsing and modulation". Penetration depths for the carrier frequency component of high-band 5G wireless radiation will be on the order of a few millimeters (Alekseev et al., 2008a, b). At these wavelengths, one can expect resonance phenomena with small-scale human structures (Betzalel et al., 2018). Additionally, numerical simulations of millimeter-wave radiation resonances with insects showed a general increase in absorbed RF power at and above 6 GHz, in comparison to the absorbed RF power below 6 GHz. A shift of 10 % of the incident power density to frequencies above 6 GHz was predicted to lead to an ***increase in absorbed power between 3–***370 % (Thielens et al., 2018).

The common 'wisdom' presented in the literature and media is that, if there are adverse impacts resulting from high-band 5 G, the main impacts will be focused on near-surface phenomena, such as skin cancer, cataracts, and other skin conditions. However, there is evidence that biological responses to millimeter-wave irradiation can be initiated

within the skin, and the subsequent systemic signaling in the skin can result in physiological effects on the nervous system, heart, and immune system (Russell, 2018).

Additionally, consider the following reference (Zalyubovskaya, 1977). This is one of many translations of articles produced in the Former Soviet Union on wireless radiation (also, see reviews of Soviet research on this topic by McRee (1979, 1980), Kositsky et al. (2001), and Glaser and Dodge (1976)). On p. 57 of the pdf link, the article by Zalyubovskaya addresses biological effects of millimeter radiowaves. Zalyubovskaya ran experiments using power fluxes of 10,000,000 μW/square meter (the FCC (Federal Communications Commission) guideline limit for the general public today in the USA), and frequencies on the order of 60 GHz. Not only was skin impacted adversely, but also heart, liver, kidney, spleen tissue as well, and blood and bone marrow properties. These results reinforce the conclusion of Russel (quoted above) that ***systemic results may occur from millimeter-wave radiation.*** To re-emphasize, for Zalyubovskaya's experiments, the incoming signal was unmodulated carrier frequency only, and the experiment was single stressor only. Thus, the expected real-world results (when human beings are impacted, the signals are pulsed and modulated, and there is exposure to many toxic stimuli) would be far more serious and would be initiated at lower (perhaps much lower) wireless radiation power fluxes.

The Zalyubovskaya paper was published in 1977. The referenced version was classified in 1977 by USA authorities and declassified in 2012. What national security concerns caused it (and the other papers in the linked pdf reference) to be classified for 35 years, until declassification in 2012? Other papers on this topic with similar findings were published in the USSR (and the USA) at that time, or even earlier, but many never saw the light of day, both in the USSR and the USA. It appears that the potentially damaging effects of millimeter-wave radiation on the skin (and other major systems in the body) have been recognized for well over forty years, yet today's discourse only revolves around the possibility of modest potential effects on the skin and perhaps cataracts from millimeter-wave wireless radiation.

6. What is the consensus on adverse effects from wireless radiation?

Not all studies of wireless radiation have shown adverse effects. For example, consider potential genotoxic effects of mobile phone radiation. A study investigating "the effect of mobile phone use on genomic instability of the human oral cavity's mucosa cells" concluded "Mobile phone use did not lead to a significantly increased frequency of micronuclei" (Hintzsche and Stopper, 2010).

Conversely, a 2017 study investigated buccal cell preparations for genomic instability, and found "The frequency of micronuclei (13.66x), nuclear buds (2.57x), basal (1.34x), karyorrhectic (1.26x), karyolytic (2.44x), pyknotic (1.77x) and condensed chromatin (2.08x) cells were highly significantly ($p = 0.000$) increased in mobile phone users" (Gandhi et al., 2017). Also, a 2017 study to ascertain the "effect of cell phone emitted radiations on the orofacial structures" concluded that "Cell phone emitted radiation causes nuclear abnormalities of the oral mucosal cells" (Mishra et al., 2017). Further, a 2016 study to "explore the effects of mobile phone radiation on the MN frequency in oral mucosal cells" concluded "The number of micronucleated cells/1000 exfoliated buccal mucosal cells was found to be significantly increased in high mobile phone users group than the low mobile phone users group" (Banerjee et al., 2016). Finally, a study aimed at investigating the health effects of WiFi exposure concluded "long term exposure to WiFi may lead to adverse effects such as neurodegenerative diseases as observed by a ***significant alteration on AChE gene expression*** and some neurobehavioral parameters associated with brain damage" (Obajuluwa et al., 2017).

There are many possible reasons to explain this lack of consensus.

1) There may be 'windows' in parameter space where adverse effects occur, and operation outside these windows would show a) no effects or b) hormetic effects or c) therapeutic effects. For example, if information content of the signal is a strong contributor to adverse health effects (Panagopoulus, 2019), then experiments that involve only the carrier frequencies may be outside the window where adverse health effects occur. Alternatively, in this specific example, the carrier signal and the information signal could be viewed as a combination of potentially toxic stimuli, where the adverse effects of each component are enabled because of the synergistic effects of the combination.

As another example, an adverse health impact on one strain of rodent was shown for a combination of 50 Hz EMF and DMBA, while no adverse health impact was shown on another rodent strain for the same toxic stimuli combination (Fedrowitz et al., 2004). From a higher-order combination perspective, if genetic abnormalities/differences are viewed conceptually as potentially equivalent to a toxic stimulus for combination purposes, then a synergistic three-constituent combination of 50 Hz EMF, DMBA, and genetics was required to produce adverse health impacts in the above experiment. If these results can be extrapolated across species, then human beings could exhibit different responses to the same electromagnetic stimuli based on their unique genetic predispositions (Caccamo et al., 2013; De Luca et al., 2014).

1) Research quality could be poor, and adverse effects were overlooked.
2) Or, the research team could have had a preconceived agenda, where finding no adverse effects from wireless radiation was **THE** objective of the study. For example, studies have shown that industry-funded research of wireless radiation adverse health effects is far more likely to show no effects than funding from non-industry sources (Huss et al., 2007; Slesin, 2006; Carpenter, 2019). Studies in disciplines other than wireless radiation have shown that, for products of high military, commercial, and political sensitivity, 'researchers'/organizations are hired to publish articles that conflict with the credible science, and therefore create doubt as to whether the product of interest is harmful (Michaels, 2008; Oreskes and Conway, 2011). Unfortunately, given the strong dependence of the civilian and military economies on wireless radiation, incentives for identifying adverse health effects from wireless radiation are minimal and disincentives are many. These perverse incentives apply not only to the sponsors of research and development, but to the performers as well.

Even the Gold Standard for research credibility - **independent replication of research results** - is questionable in politically, commercially, and militarily sensitive areas like wireless radiation safety, where the accelerated implementation goals of most wireless radiation research sponsors (government and industry) are aligned. It is imperative that highly objective evaluators with minimal conflicts of interest play a central role ensuring that rigorous safety standards for wireless radiation systems are met before widescale implementation is allowed.

7. Conclusions

Wireless radiation offers the promise of improved remote sensing, improved communications and data transfer, and improved connectivity. Unfortunately, there is a large body of data from laboratory and epidemiological studies showing that previous and present generations of wireless networking technology have significant adverse health impacts. Much of this data was obtained under conditions not reflective of real-life. When real-life considerations are added, such as 1) including the information content of signals along with 2) the carrier frequencies, and 3) including other toxic stimuli in combination with

the wireless radiation, the adverse effects associated with wireless radiation are increased substantially. Superimposing 5G radiation on an already imbedded toxic wireless radiation environment will exacerbate the adverse health effects shown to exist. Far more research and testing of potential 5G health effects under real-life conditions is required before further rollout can be justified.

Transparency document

The Transparency document associated with this article can be found in the online version.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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The International Commission on Non-Ionizing Radiation Protection: Conflicts of interest , corporate capture and the push for 5G

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and

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The International Commission on Non-Ionizing Radiation Protection: Conflicts of interest, corporate interests and the push for 5G

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Foreword by Klaus Buchner and Michèle Rivasi

This report deals with an issue of which the importance cannot be overrated: the possible health effects of Radiofrequency Radiation (RfR) or electro magnetic fields (EMF); It deals more specifically with how the scientific debate has been hijacked by corporate interests from the Telecom industry.

After having read the reports of a journalistic collective called Investigate Europe, the many articles from Microwave News as well as all the publications from independent scientists from around the world, who for years have all been ringing alarm bells on adverse health effects from the use of mobile phones and EMF, we decided that we needed to dig deeper into this strange, unknown to the public but powerful scientific NGO based in Germany called the 'International Commission on Non-Ionizing Radiation Protection' (ICNIRP).

The findings of this report ('The International Commission on Non-Ionizing Radiation Protection: Conflicts of interest and the push for 5G') give us an uncomfortable déjà-vu: many facts and processes that lead to the actual situation whereby European authorities – from the European Commission to most of the member states – simply close their eyes for real scientific facts and early warnings. We have seen exactly the same scenario in the debate on Tobacco, asbestos, climate change and pesticides.

Also in it's latest guidelines from March this year, ICNIRP assures the world that there is no scientific evidence of adverse health effects from the radiation that comes with the new communication technologies, within the limits it proposes. But at the same time a growing number of scientists and also citizens are worried that EMFs do cause health problems. ICNIRP pretends to be scientifically neutral, and free from vested interests of the Telecom industry. We show with this study that this is 'playing with the truth' or simply a lie.

Already in 2011 Dr. Jacqueline McGlade, Executive Director of the European Environment Agency said on mobile phones and the potential head cancer risk for EMF: "The European Parliament has responded (resolution of April 2009) to this public concern with a resolution on EMF in 2009 which, among other things, called for lowering exposure to electromagnetic fields and for lower exposure limits that would better protect the public from health hazards. We share these recommendations."

McGlade pleaded interim actions to protect public health, particularly for children on the basis of the precautionary principle, as central to public policymaking where there is scientific uncertainty and high health, environmental and economic costs in acting, or not acting, when faced with conflicting evidence of potentially serious harm. "This is precisely the situation that characterises EMF at this point in its history. Waiting for high levels of proof before taking action to prevent well known risks can lead to very high health and economic costs, as we have seen with asbestos, leaded petrol and smoking," said McGlade.

The EEA plea for a precautionary approach to policy making in this area, is based on an evaluation of the existing evidence and on the lessons from earlier hazards, analysed in the EEA "Late Lessons from Early Warnings" project. David Gee, EEA Senior Advisor on Science, Policy and Emerging Issue and on the drivers of this project said: "Mobile phones have numerous social, economic and even environmental benefits", said. "However, there is significant disagreement in the scientific community about whether mobile phone use increases the risk of head cancers. We recommend using the precautionary principle to guide policy decisions in cases like this. This means that although our understanding is incomplete, this should not prevent policy makers from taking preventative action".

In a recent discussion Gee stated that there are "several striking similarities" between 5G/radiofrequency radiation and many of the technologies or substances that featured in the "Late Lessons" case studies. Gee pointed to "a lot of hubristic hype surrounded the introduction of the new technology". Gee rightfully points to a "marketing hype which is widespread" on 5G and "a failure to systematically and independently scrutinise the claimed benefits and costs of the new technology". He sees a "gross imbalance between research on developing and promoting the technology and on anticipating and reducing potential harm to people and environments" as well as a "failure to ensure independent research into health and environmental effects that can help combat manufactured doubt".

Gee was tough for the scientific community because scientists fail to acknowledge what they do not know and "to properly understand and embrace knowledge from other relevant disciplines". Gee also sees "a failure of scientists to be transparent about the paradigms, assumptions, judgements and values used in academic science and in their evaluations of scientific evidence in regulatory science. A failure of scientists and policymakers to appreciate complex and variable realities; multi-causality; and the likelihood of inconsistent scientific results. A failure by policymakers to understand the difference between the high strength of evidence needed to establish robust scientific knowledge and the case specific appropriate strength of evidence needed to justify timely preventive action."

Late lessons from early warnings, is indeed also a clear pattern that rises from this report. And there have been more and more warnings (but unfortunately so far no lessons learned).

Also the Council of Europe adopted in May 2011 a strong resolution on "the potential dangers of electromagnetic fields and their effect on the environment" in which it called upon governments to take all reasonable measures to reduce exposure to electromagnetic fields and said about ICNIRP: "It is most curious, to say the least, that the applicable official threshold values for limiting the health impact of extremely low frequency electromagnetic fields and high frequency waves were drawn up and proposed to international political institutions (WHO, European Commission, governments) by the ICNIRP, an NGO whose origin and structure are none too clear and which is furthermore suspected of having rather close links with the industries whose expansion is shaped by recommendations for maximum threshold values for the different frequencies of electromagnetic fields".

In an article, 'Planetary electromagnetic pollution: it is time to assess its impact', published in *The Lancet (*December 2018) scientists from the Australian research group ORSAA state that out of 2266 studies on EMFs, no less than 68 percent found "significant biological effects or health effects". Significant biological effects do not necessarily mean that human health will be harmed, but is an important indicator for risk assessment and then for risk evaluation by regulators. To us the atgument that that there is insufficient scientiifc evidence for regulators to act is factual not corect and simply not true.

The International Agency for Research on Cancer (IARC), a global authority on cancer, concluded in 2011 that radiation from mobile phones is a 'possible' head cancer risk. And recently an Advisory Group has recommended that IARC should reassess the cancer risks associated with non-ionizing radiofrequency radiation with high priority. According to the panel's report, published in The Lancet, the group suggests that the new evaluation should take place between 2022 and 2024.

In 2012 a group of 29 independent scientists and health experts from around the world warned in an update of their Bio Initiative 2007 Report, about "possible risks from wireless technologies and electromagnetic fields". However, they acknowledge that "sometimes, science does not keep pace with new environmental exposures that are by-products of useful things we want to buy and use in

society. So, the deployment runs ahead of knowledge of health risks. It is an old story. This is the case for EMF (electric and magnetic fields) and RFR (Radiofrequency radiation)."

The Bio Initiative report underscores the "critical need to face difficult questions, make mid-course corrections, and try to repair the damage already done in this generation, and to think about protecting future generations".

And they state that the existing public safety limits as formulated by the US regulator FCC and by ICNIRP do not sufficiently protect public health against chronic exposure from very low-intensity exposures: "If no mid-course corrections are made to existing and outdated safety limits, such delay will magnify the public health impacts with even more applications of wireless-enabled technologies exposing even greater populations around the world in daily life."

In 2017, more than 200 doctors and scientists from various countries launched the, so-called 5G Appeal, that has since received more endorsements and whose mission statement starts with : "We the undersigned scientists and doctors(…), recommend a moratorium on the roll-out of the fifth generation, 5G, for telecommunication until potential hazards for human health and the environment have been fully investigated by scientists independent from industry."

Since then there have been five replies on this Appeal by the European Commission, the last one dating from December 2019. The first reply, the Commission states that 'the Commission is not aware of any conflicts of interests of members of international bodies such as ICNIRP or the members of SCENIHR'. One of the leading figures of the appeal professor Lennart Hardell stated that this «does not represent the scientific evidence of inherent conflicts of interest both in ICNIRP and SCENIHR. The European Commission seems to be ill-informed or even misinformed, as the EU seems to take information mainly from these two fraudulent organisations, but not from independent researchers. The EU does not seem to rely on sound science and thereby downplays the RF-related risks."

It is clear from this report that ICNIRP itself does not have a sharp definition of conflicts of interest (CoI's), nor does it have a well-developed policy to avoid these kinds of conflicts. It is a crying shame that under the pretext of 'scientific uncertainty' ICNIRP, but especially the European Commission and member states keep on failing to protect their citizens.

We very much agree with the title and content of the latest publication on Microwave News, which reads " The Lies Must Stop, Disband ICNIRP - Facts Matter, Now More Than Ever" . There are two major casualties in this polarised debate: the truth and public health. Both are too important not to protect with all that we have. That is what we consider as our responsibility as elected politicians .

By MEP's Michèle Rivasi (Europe Écologie) and Dr. Klaus Buchner (Ökologisch-Demokratische Partei)

Introduction & Scope

In the last few decades, since the introduction, and rapid expansion, of new communication technologies, there has been a proliferation of electromagnetic fields worldwide. A lot of countries are now about to roll out 5G networks. The International Commission on Non-Ionizing Radiation Protection (ICNIRP) assures the world that this can be done safely and that there is no scientific evidence of adverse health effects within the limits it proposes. But at the same time a growing number of scientists and also citizens are worried that EMFs do cause health problems.

It is therefore high time to look into the workings of ICNIRP. If the European Commission and national governments keep relying on this commission, as is currently the case, we must be completely sure that it functions wholly independently and that there is no evidence of its members being in situations of conflicts of interest.

ICNIRP is a non-governmental organisation (NGO) or association, registered in Munich, specialising in non-ionizing radiation protection. One of the organisation's tasks is to determine exposure limits for electromagnetic fields used by devices such as cellular phones. On its website, ICNIRP states that it is a non-profit organisation with a scientific mission, and that it is "formally recognised as an official collaborating non-state actor by the World Health Organisation (WHO) and the International Labour Organisation (ILO). ICNIRP is consulted by the European Commission and is linked to many organisations engaged in non-ionizing radiation (NIR) protection worldwide through diverse collaborative projects".

ICNIRP states that its "aim is to protect people and the environment against adverse effects of NIR." To this end, it "develops and disseminates science-based advice on limiting exposure to non-ionizing radiation." ICNIRP works with experts from all over the world, from a wide variety of disciplines, including biology, epidemiology, medicine, physics, and chemistry. ICNIRP's also states that its protection advice is based on current scientific knowledge about the biological effects, and the action mechanisms, of radiation for the whole NIR frequency range.

To a large extent, the European Commission, as well as the WHO, depend on the "exposure guidance" and safety advice given by ICNIRP. Furthermore, many EU member states look to the EC and WHO for (European) advice on this issue. Therefore, it goes without saying that ICNIRP has a significant role to play in ensuring the general public is protected against any possible health risks related to electromagnetic fields (EMF).

In March 2019, in a comprehensive report, *How much is Safe?, by Investigate Europe*, a collective of investigative journalists from all over Europe, ICNIRP is described as follows:

''ICNIRP is a particularly influential group, as it not only evaluates radiation and health risk research, but also provides guidelines for radiation safety limits that most countries use. It is a private, German-registered organisation located outside Munich, behind a yellow door on the premises of the German Federal office for radiation protection. Decisions on who to invite in, are taken by ICNIRP itself."

The report highlighted the close links that exist between ICNIRP and other important organisations in the field of health protection.

Most European governments and radiation protection authorities rely mainly on these four scientific bodies for advice on non-ionizing radiation protection:

- The international commission on non-ionizing radiation protection, ICNIRP.
- The EU Scientific Committee on Health, Environment and Emerging Risk, SCENIHR / SCHEER.
- The World Health Organisation WHO's International EMF Project.
- The WHO Cancer Unit IARC, International Agency for Research on Cancer.

Investigate Europe showed the close links between especially the first three bodies. "The groups, however, are to a remarkable degree, staffed by the same experts," it stated. "Of 13 ICNIRP scientists, six are members of at least one other committee. In the WHO group, this applies for six out of seven (members)." The SCENIHR Working Group on EMF also counts two ICNIRP-members.

In view of the rapid expansion of EMF's, in particular in the context of the planned deployment of 5G networks in which telecom and media operators have huge financial and economic vested interests, and given the evidence of closed circles of experts involved in determining health guidelines in this field, critical scrutiny on the functioning of ICNIRP is important and necessary.

New guidelines

In March 2020, ICNIRP published its latest 'Guidelines on Limiting Exposure to Electromagnetic Fields', designed for "the protection of humans exposed to radiofrequency electromagnetic fields (RF) in the range 100 kHz to 300 GHz. The guidelines cover many applications such as 5G technologies, Wi-Fi, Bluetooth, mobile phones, and base stations."

This publication replaces and supersedes earlier publications from 1998 and 2010. In a press release from March 11th 2020, the then ICNIRP Chairman, Dr Eric van Rongen (now co-chair) said: "The new electromagnetic field guidelines have taken seven years to develop and are more appropriate than the 1998 guidelines for the higher frequencies that will be used for 5G in the future. We know parts of the community are concerned about the safety of 5G and we hope the updated guidelines will help put people at ease. When we revised the guidelines, we looked at the adequacy of the ones we published in 1998. We found that the previous ones were conservative in most cases, and they would still provide adequate protection for current technologies."

Van Rongen's main message was that when the new ICNIRP guidelines are followed 5G is absolutely safe. He stated: "The new guidelines provide better and more detailed exposure guidance, in particular for the higher frequency range, above 6 GHz, which is of importance to 5G, and future technologies using these higher frequencies. The most important thing for people to remember is that 5G technologies will not be able to cause harm when these new guidelines are adhered to."

So, this is how ICNIRP presents itself: an independent organisation that gives sound scientific advice on safety guidelines with respect to non-ionizing radiation and that ensures citizens remain safe.

However, this description raises doubts on two levels: Firstly, is ICNIRP really independent and also, are its assurances that non-ionizing radiation is absolutely safe when their guidelines are applied correct? Our report will focus on the question of ICNIRP's independence, but first, we will briefly outline the current debate around the safety guidelines.

The health debate

The possible adverse health effects of non-ionizing radiation, mainly microwave radiation form mobile phones and other wireless devices/infrastructure, is a highly sensitive and polarising issue. In some countries citizens and scientists plead for the application of the 'pre-cautionary principle' in relation to the rolling out of 5G networks, whilst associations such as ICNIRP maintain that "the most important thing for people to remember is that 5G technologies will not be able to cause harm when these new guidelines are adhered to."

In 2012 a group of 29 independent scientists and health experts from around the world published an update of their Bio Initiative 2007 Report, about "possible risks from wireless technologies and electromagnetic fields". The scientists, of which ten holding a medical degree, still update their "rationale for Biologically-based Public Exposure Standards for Electromagnetic Fields (Extremely low frequency, ELF and radiofrequency, RF)" by assessing the latest scientific research and reporting on it. However, they acknowledge that "sometimes, science does not keep pace with new environmental exposures that are by-products of useful things we want to buy and use in society. So, the deployment runs ahead of knowledge of health risks. It is an old story. This is the case for EMF (electric and magnetic fields) and RFR (Radiofrequency radiation)."

The Bio Initiative report underscores the "critical need to face difficult questions, make mid-course corrections, and try to repair the damage already done in this generation, and to think about protecting future generations".

And they state that the existing public safety limits as formulated by the US regulator FCC and by ICNIRP do not sufficiently protect public health against chronic exposure from very low-intensity exposures: "If no mid-course corrections are made to existing and outdated safety limits, such delay will magnify the public health impacts with even more applications of wireless-enabled technologies exposing even greater populations around the world in daily life."

In an article, 'Planetary electromagnetic pollution: it is time to assess its impact', published in *The Lancet Planetary Health* in December 2018, scientists (from the Oceania Radiofrequency Scientific Advisory Association, ORSAA, and the Institute for Health and the Environment, of the University at Albany) state that out of 2266 studies on EMFs, no less than 68 percent found "significant biological effects or health effects". Significant biological effects do not necessarily mean that human health will be harmed, but is an important indicator for risk assessment and then for risk evaluation by regulators.

The authors stated that it is high time for a wide-ranging debate on the rapid global proliferation of artificial electromagnetic fields. “The most notable is the blanket of radiofrequency electromagnetic radiation, largely microwave radiation generated for wireless communication and surveillance technologies, as mounting scientific evidence suggests that prolonged exposure to radiofrequency electromagnetic radiation has serious biological and health effects.”

Unfortunately, this mounting evidence did not result in policy changes, the authors from ORSAA observe. “However, public exposure regulations in most countries continue to be based on the guidelines of the *International Commission on Non-Ionizing Radiation Protection* and Institute of Electrical and Electronics Engineers, which were established in the 1990s on the belief that only acute thermal effects are hazardous. Prevention of tissue heating by radiofrequency electromagnetic radiation is now proven to be ineffective in preventing biochemical and physiological interference”.

“For example, acute non-thermal exposure has been shown by NIH scientists, to alter human brain metabolism, electrical activity in the brain and systemic immune responses. Chronic exposure has been associated with increased oxidative stress and DNA damage, and cancer risk. Laboratory studies, including large rodent studies by the US National Toxicology Program and Ramazzini Institute of Italy, confirm these biological and health effects in vivo. As we address the threats to human health from the changing environmental conditions due to human activity, the increasing exposure to artificial electromagnetic radiation needs to be included in this discussion.”

The results of the National Toxicology Programme (NTP) the mentioned Lancet-authors referred to, were presented at the end of 2018. The U.S. Food and Drug Administration (FDA) nominated radio frequency radiation (RFR) used by cell phones for an NTP study because of the widespread public use of cell phones and the limited knowledge about potential health effects from long-term exposure. The study found that high exposure to RFR (900 MHz) used by cell phones was associated with:

- Clear evidence of tumours in the hearts of male rats. The tumours were malignant schwannomas.
- Some evidence of tumours in the brains of male rats. The tumours were malignant gliomas.
- Some evidence of tumours in the adrenal glands of male rats. The tumours were benign, malignant, or complex combined pheochromocytoma.

However, ICNIRP criticised the NTP-study, saying that it did not prove a link between Radio Frequency, Electro Magnetic Fields and carcinogenesis. But according to scientists like Lennart Hardell, an oncologist, professor and researcher at the University hospital in Örebro in Sweden, the ICNIRP rebuttal of the NTP-study was unfounded. The NTP-study leading scientist Ronald Melnick recently also published a comment on the ICNIRP-note in which he criticizes ICNIRP’s ”incorrect statements” and “false claims”.

James Lin, professor at the University of Illinois in Chicago and also editor of the online journal, *Bioelectromagnetics*, published a remarkable and nuanced review of the NTP-study in late 2019. The review is remarkable because, from 2004 to 2016, James Lin was himself a member of ICNIRP. As stated above, ICNIRP basically dismisses the NTP-study. However,

basing his conclusions partly on the NTP-study, Lin now questions if the existing safety guidelines are still adequate: “An outstanding question persists on the adequacy of these guidelines for safe long-term exposure to RF radiation at or below 1.6 or 2.0 W/kg. Perhaps, the time has come to judiciously reassess, revise, and update these guidelines.”

Lin’s review is nuanced in so much as he uses the peer-review process to analyse the conception and all possible methodological ‘problems’ of the NTP-study in depth: “This project is the largest NTP animal cancer study ever. It was nominated by the Food and Drug Administration (FDA) in 1999. The supposedly 5-year project was sole sourced in 2004 to an industrial research firm as the project’s principal investigator. The work began in 2005. However, the project had been protracted for more than a dozen years with huge budget overruns, and an estimated eventual price tag of $25 million.”

Somewhat surprisingly, at the end of his review, Lin advocates for wireless radiation to “get a more stringent cancer risk class": “Now that the NTP review panel has concluded that there is clear evidence of carcinogenicity from long-term RF exposure in rats, is it conceivable that IARC would upgrade its epidemiology-based classification of RF exposure to the next higher levels of carcinogenicity to humans?” Lin seems to suggest that IARC should put cell phone radiation in WHO-hazard class 1 (carcinogenic), instead of today’s 2B (possibly carcinogenic).

Worldwide, there is rapidly growing concern and a proliferation of publications about EMF, specifically concerning the out-roll of new generation 5G. On this subject, we will only cite a 2019 in-depth report called “5G Deployment: State of Play in Europe, USA, and Asia”[1]. It reads: “Increased exposure may result, not only from the use of much higher frequencies in 5G, but also from the potential for the aggregation of different signals, their dynamic nature, and the complex interference effects that may result, especially in dense urban areas. (…) The 5G radio emission fields are quite different to those of previous generations because of their complex beam-formed transmissions in both directions – from base station to handset and for the return.”

The authors state that with 5G we are entering unknown territory. “Although fields are highly focused by beams, they vary rapidly with time and movement and so are unpredictable, as the signal levels and patterns interact as a closed loop system. This has yet to be mapped reliably for real situations, outside the laboratory. (..) The problem is that currently it is not possible to accurately simulate or measure 5G emissions in the real world.”

The debate on the safety of non-ionizing radiation is fascinating, heated and important, and has been on-going for at least 30 years. This paper however does *not* go further into the scientific debate on the possible levels of harm to public health caused by non-ionizing radiation, mainly from mobile phones. We will focus on the independence of ICNIRP and the possible existence of conflicts of interest of its members.

[1] A study requested by the ITRE committee of the European Parliament, published in 2019 by the Policy Department for Economic, Scientific and Quality of Life Policies - Directorate-General for Internal Policies.

The importance of funding

ICNIRP claims it is “free of vested interests”. ICNIRP's funding relies on grants from public bodies. Additionally, ICNIRP members and ICNIRP SEG members may not be employed by industry.

But not being “employed by industry” is not, in itself, sufficient to avoid conflicts of interest. It is also important to ascertain to what extent ICNIRP research activities may be funded by industry.

It is a well-established fact that the source of funding for scientific research can have an influence on the outcomes of research. A clear and precise explanation of how this is may occur can be found on the website of UC Berkeley:

“In a perfect world, money wouldn't matter — all scientific studies (regardless of funding source) would be completely objective. But of course, in the real world, funding may introduce biases — for example, when the backer has a stake in the study's outcome. A pharmaceutical company paying for a study of a new depression medication, for example, might influence the study's design or interpretation in ways that subtly favour the drug that they'd like to market. There is evidence that some biases like this do occur. Drug research sponsored by the pharmaceutical industry is more likely to end up favouring the drug under consideration than studies sponsored by government grants or charitable organisations. Similarly, nutrition research sponsored by the food industry is more likely to end up favouring the food under consideration than independently funded research.”

“This does not lead to the conclusion that we should ignore any research funded by companies or special interest groups”, Berkeley says. But it is a reason for the need “to scrutinize studies funded by industry or special interest groups with extra care. *So, don't, for example, brush off a study of cell phone safety just because it was funded by a cell phone manufacturer — but do ask some careful questions about the research before jumping on the bandwagon*. Are the results consistent with other independently funded studies? Does the study seem fairly designed? What do other scientists have to say about this research? A little scrutiny can go a long way towards identifying bias associated with funding source.”

“A little scrutiny” is perhaps an understatement. In the 2013, the ‘Late lessons from early warnings’ report produced by the European Environment Agency (EEA), a chapter written by Lisa A. Bero, describes the various opinions on how to deal with private funding of scientific research without compromising an independent non-biased outcome and/or publication of that research.

For example, various researchers argue that it is logical for industry to fund research, in so much as it is about their products that concerns exist. Former ICNIRP scientist Norbert Leitgeb, professor at the Institute of Health Care Engineering at the Graz University of Technology in Austria, told *Investigate Europe* that what is crucial is the putting in place of effective firewalls to ensure that “private partners cannot interfere with researchers and influence scientific outcomes or conclusions”.

That the source of funding has an important influence, is also something various ICNIRP-researchers acknowledge. For example, in 2009 two scientists who are now members of the ICNIRP-commission – Anke Huss and Martin Röösli – where co-authors of a systematic

review that showed that "industry-sponsored studies were least likely to report results suggesting effects". They concluded that the correlation between the "source of funding and conflicts of interest are important in this field of research."

in his evaluation of the NTP-study, another former ICNIRP-member, professor James Lin, also pointed to the dominance of the telecom industry in the research: "The FDA should be applauded for nominating, and NIEHS/NTP should be lauded for having sponsored the research and conducted the Cell Phone Radio Frequency Radiation (RFR) Studies. It's important for the U.S. government to step in to conduct such a research program, and not leave the matter entirely to the cell phone industry. The wireless industry has had nearly free reign to develop and roll out cellular mobile phones and related RF devices as they see fit. (…)". Lin goes on to quote figures from the 'systematic review': "A systematic review of 59 published studies of controlled exposure to RF radiation with health-related outcomes [10] showed that public agencies or charities funded 11 (19%), the wireless communications industry funded 12 (20%), mixed sources (including industry) funded 14 (24%), and in 22 (37%) the source of funding was not reported."

This specific debate has been ongoing for many years, as *Investigate Europe* reports: "At least three studies over the years have documented that there is often a link between conclusions of studies and the source of the money that paid for the research. Science funded by industry is less likely to find health risks than studies paid for by institutions or authorities."

In 'How much is safe?' by *Investigate Europe*, Lennart Hardell, an oncologist, professor and researcher at the University hospital in Örebro in Sweden, a critical EMF researcher, warns that although funding for research often goes to universities with "firewalls" put in place between the individual scientist and the funder, the problem is, that researchers can come to depend on this private funding to safeguard the future of their research.

Hardell carries out research on the possible links between long-term mobile use and brain cancer and has published results that indicate that there are correlations between the two. Hardell was a member of the IARC committee that researched EMF-effects, but is not a member of (any) other committees concerned with the effects of non-ionizing radiation. *Investigate Europe*: "According to Hardell, his research is funded through his salary from the hospital, as well as by funds raised by local cancer foundations and national organisations. "Of course, I have also worked a lot on my free time", he says."

There are some ICNIRP-researchers who acknowledge that it is possible for the source of funding to influence conclusions, but they say that they are very aware of this and cautious to avoid it. For example, Gunnhild Oftedal, - associate professor at the Norwegian University of Science and Technology, who specialises in research on the effects of electromagnetic fields on humans, and is a member of ICNIRP and therefore part of "the small international network that determines what science to trust" said to *Investigate Europe* that "today we are concerned about it. I have the impression that scientists are much more cautious about receiving support from the industry – at least direct support."

What about the direct funding received by ICNIRP itself? ICNIRP states that its "funding stems from subsidies granted by national and international public institutions such as the German Federal Ministry for the Environment, Nature Conservation, and Nuclear Safety

(BMU), the European Union Programme for Employment and Social Innovation (EaSI) 2014-2020 (EC - Directorate General Social Affairs), and the International Radiation Protection Association (IRPA)."

"Occasionally, ICNIRP also receives support to organise meetings or workshops from national ministries or radiation protection agencies, such as the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA), and the Turkish Ministry of Health (MoH). Funding is reported yearly in the ICNIRP annual reports". ICNIRP also acknowledges that it receives funding from national or international public organisations and via private donations. But ICNIRP claims that in order to safeguard its independence, "only donations from private individuals or from businesses not related in any way to the field of non-ionizing radiations can be accepted. For reasons of transparency, donations cannot be anonymous and are listed in an ICNIRP donors' report."

According to the ICNIRP 2018 annual report, it received € 132,150 in subsidies. The Australian research group ORSAA points out that these kinds of funding sources are not always as neutral as they may seem: "ICNIRP funding partly comes from government regulatory bodies, such as, for example, the Australian Radiation Protection & Nuclear Safety Agency (ARPANSA). What is actually going on is best described as 'money laundering' by the Telecom industry through government (ARPANSA) and onto WHO's International EMF Project and ICNIRP."

In Australia, as is the case for many countries worldwide, the government issues spectrum licences to Telecom operators for large sums of money – often in the billions. In Australia, this licensing is the remit of the industry regulator ACMA, the Australian Media Communications Authority. ORSAA explains that ACMA also collects a separate levy, or tax, from the wireless industry, money that is earmarked for scientific research on RF-EMR health effects: "This has remained a set amount of $1M per annum since 1997, despite the massive increases in wireless industry revenues."

According to ORSAA, ACMA then diverts $300,000 to another government body, ARPANSA (Australian Radiation Protection & Nuclear Safety Agency) for its public information campaign, and $700,000 to the National Health & Medical Research Council (NHMRC). From the $300,000 received annually by ARPANSA, a portion goes to the WHO's IEMFP (some years ago this was around $50,000 a year), and finally, it appears that a portion goes to ICNIRP. So, after a long trajectory, money from the Telecom industry does end up with ICNIRP, which is contrary to the statement on the ICNIRP website: "Only donations from private individuals or from businesses not related in any way to the field of non-ionizing radiations can be accepted."

Still according to ORSAA, "the money that the Australian NHMRC receives in order to provide grants for medical research has mostly gone to industry-friendly researchers who have direct links with the wireless industry. For example, the largest recipient of these NHMRC research funds is Prof. Rodney Croft, a psychology researcher at the University of Wollongong, who held the role of Director of the Australian Centre for Electromagnetic Bio-effects Research (ACEBR) for many years[2]. Rodney Croft has essentially been the head of RF-EMR health research in Australia, despite his questionable qualifications for this health research role.

[2] See also portrqit of Rodney Croft on pqge 50 of this report.

Notably, he has led ICNIRP's RF-EMR exposure guidelines development team and now he has been elected as the next Chairman of ICNIRP as from May 2020. Prof. Croft has received ample direct industry funding in addition to his lucrative NHMRC grants, which should be termed indirect industry funding."

Finally, ICNIRP states on its website that all its experts "are required to comply with the ICNIRP policy of independence and declare their personal interests. (…) These are key elements to ICNIRP's commitment to independence and transparency, which ICNIRP believes is fundamental to carrying out its scientific mission."

Whether those declarations of interests are really checked is something that the Italian 'Vallisoletana Association of people affected by mobile phone antennas' (AVAATE) questioned in their public statement from July 2015, attacking ICNIRP: "It is hard to understand whether ICNIRP investigates the Declarations filed by appointed members of the ICNIRP Commission and Scientific Expert Committee, since in some cases these members report that they work or have worked for these organisations but do not specify what they have done or whether they are paid. It is also hard to understand how ICNIRP controls the content of the declarations by the appointed members of their Expert Committees, when in most cases the most contentious aspects of the biographical statement are not reported in these statements."

The citizens behind AVAATE also ask "how ICNIRP controls the content of the declarations by the appointed members of their Expert Committees when, at least in five cases, the persons concerned have not signed their statements".

Corporate capture

In the debate on EMF and possible health effects, terms like 'corporate capture' of scientific research and 'war game science' are often used, and references to the tactics of the tobacco industry are often made. According to several authors, these tactics also influence organisations like ICNIRP and WHO's International EMF Project.

In the 2013 'Late lessons from early warnings' report produced by the European Environment Agency (EEA), in collaboration with a broad range of external authors and peer reviewers, these tactics are described in detail in the chapter entitled 'Tobacco industry manipulation of research'. The focus is on "the strategies used by the tobacco industry to deny, downplay, distort and dismiss the growing evidence that, like active smoking, ETS causes lung cancer and other effects in non-smokers." Author Lisa A. Bero concentrated "on the 'argumentation' that was used to accept, or reject, the growing scientific evidence of harm. Who generated and financed the science used to refute data on adverse health effects? What were the motivations? What kind of science and information, tools and assumptions were used to refute data on the adverse health of tobacco?"

Bero says: "The release of millions of internal tobacco industry documents due to law suits in the US has given insights into the inner workings of the tobacco industry and revealed their previously hidden involvement in manipulating research. However, this insight is not available for most corporate sectors."

Bero also discusses the possibilities of 'full disclosure' of funding sources and special interests in research and risk assessment in order to secure independence and prevent bias

towards particular viewpoints. She states that “while smoking bans are now being introduced in more and more countries, other industries are drawing inspiration from tobacco company strategies, seeking to maintain doubt about harm in order to keep hazardous products in the marketplace.”

With respect to the EMF-debate, according to Bero, public institutions or authorities should adhere to the following: “when data on risk appear to be controversial, users of the data investigate the sources of the controversy. Does the controversy exist only because the findings of interest group-funded research are contrary to data collected by others? Is the controversy supported primarily by evidence published in interest group-supported publications? (…) Policymakers should apply these questions to all situations in which a company has an interest in creating controversy about the risks of its products.”

According to Bero, the tobacco industry's methods for influencing the design, conduct and publication of research are similar to those of other corporate interests.

One of the leading researchers in the US who defends the viewpoint that the same tactics are being used by Telecom companies is Theodora Scarato, Executive Director of the US based Environmental Health Trust (EHT). As a policy analyst, Scarato manages and updates the comprehensive EHT database on international policy that documents the 20+ nations that have protective policies in place to reduce public exposure to cell phone and wireless radiation.

Scarato and EHT claim that “Just as the Tobacco Industry created a ‘Playbook’ to defend cigarettes and manufacture doubt about the health effects of cigarettes, the Wireless Industry seems to have a fine-tuned the “Playbook” of advertising, public relations and industry-funded science to defend wireless products and falsely reassure the public that cell phones and wireless products are safe.”

“Key to this public relations effort are industry created resources, websites and materials that communicate the myth of no proof of harm from wireless products. These are all part of the Playbook to manufacture doubt that a problem exists. Examples of such propaganda range from glossy brochures, Questions and Answers on Hot Topics such as “children and cell phones”, websites on EMF and Health and research forums.”

And according to Scarato, “these materials are paid for, designed and prepared by ‘non-profit’ organisations that are created by telecom and wireless companies pooling money together. When citizens raise concerns about a particular product or when research comes out indicating a health risk, companies can simply pull from these materials to respond as if there are no concerns”.

These kind of tactics, used to influence science and risk assessment, also have their repercussions for standard-setting bodies like ICNIRP and WHO’s International EMF Project, according to scientific researcher Don Maisch (in his PhD thesis ‘An examination of the manipulation of telecommunications standards by political, military, and industrial vested interests at the expense of public health protection’): “In an ever increasingly globalised world the reliance on international organisations to set standards to protect public health seems inevitable. Proposed internationalised standards such as ICNIRP’s recommendations act as an aid to economic development by not hindering trade that might conflict with

stricter national standards (such as the Russian Federation, the Czech Republic's former standard and China for example). In the delicate trade-off between economic benefits and adequate health protection, international organisations should ideally be "eternally vigilant" to ensure that their tasks are not co-opted by vested interest groups that are the producers of risks to be regulated."

This appears to be a global issue. US researcher, Norm Alster, in his report 'Captured Agency' describes what this kind of corporate capture can lead to by referring to the workings of the FCC (Federal Communications Commission), which is the main official US institution that deals with Telecom issues, and is sometimes mentioned in critiques of ICNIRP: "That is a term that comes up time and time again with the FCC. Captured agencies are essentially controlled by the industries they are supposed to regulate. A detailed look at FCC actions—and non-actions—shows that over the years the FCC has granted the wireless industry pretty much what it has wanted".

"As a result, consumer safety, health, and privacy, along with consumer wallets, have all been overlooked, sacrificed, or raided due to unchecked industry influence. (…) Most insidious of all, the wireless industry has been allowed to grow unchecked and virtually unregulated, with fundamental questions on public health impact routinely ignored. (…) Industry control, in the case of wireless health issues, extends beyond Congress and regulators to basic scientific research. And in an obvious echo of the hardball tactics of the tobacco industry, the wireless industry has backed up its economic and political power by stonewalling on public relations and bullying potential threats into submission with its huge standing army of lawyers. (…) Industry behaviour also includes self-serving public relations and hyper aggressive legal action. It can also involve undermining the credibility of, and cutting off funding for, researchers who do not endorse cellular safety. It is these hardball tactics that recall 20th century Big Tobacco tactics."

Conflicts of Interest

In 2017, almost 200 doctors and scientists from various countries launched the, so-called 5G Appeal, that has since received more endorsements and whose mission statement starts with : *"We the undersigned scientists and doctors(…), recommend a moratorium on the roll-out of the fifth generation, 5G, for telecommunication until potential hazards for human health and the environment have been fully investigated by scientists independent from industry."*
Since then, as professor Hardell describes in his article "Appeals that matter or not on a moratorium on the deployment of the fifth generation, 5G, for microwave radiation" published in January 2020, there have been five replies on this Appeal by the European Commission, the last one dating from December 2019. The first reply, by the Commission (from October 13, 2017 by the Directorate-General Health and Food Safety) states that *'the Commission is not aware of any conflicts of interests of members of international bodies such as ICNIRP or the members of SCENIHR'*.

However, according to Hardell, "that does not represent the scientific evidence of inherent conflicts of interest both in ICNIRP and SCENIHR. The European Commission seems to be ill-informed or even misinformed, as the EU seems to take information mainly from these two

fraudulent organisations, but not from independent researchers. The EU does not seem to rely on sound science and thereby downplays the RF-related risks."

Given the important effects of funding on research outcomes described above, there can be no doubt that it is extremely important for ICNIRP to ensure it avoids any possibility of conflicts of interests in the way that it, or any of its members, function. In its statutes, it writes: 'No member of the Commission shall hold a position of employment that, in the opinion of the Commission, will compromise its scientific independence.'

The crucial words here are '*in the opinion of the Commission*'. The Commission evaluates itself about possible conflicts of interest. There are no clear rules by which the Commission judges if any of its members interests compromise its scientific independence. In its statement on the declarations of interests ICNIRP writes:

"The evaluation of personal integrity is very complex and might never be achievable in a perfect way. It is the duty of the ICNIRP Commission to carefully consider and decide if the declared interests potentially constitute a conflict of interest."

It is clear from this that ICNIRP itself does not have a sharp definition of conflicts of interest (CoI's), nor does it have a well-developed policy to avoid these kinds of conflicts.

It is useful to refer to a recent study requested by the European Parliament's Petitions (PETI) committee which, as a key message, said that "EU institutions and agencies lack a consistent definition of conflicts of interest and common rules on transparency'. This same study also stated that "a coherent policy should be developed for the required length of time between working in the industry and being called to a committee among agencies with a similar function, i.e. risk assessment".

In the online newsletter, *Politico*, the Greek MEP Alexis Georgoulis said: "There is a legal inconsistency between the definitions of the conflicts of interest that should clearly cover any conflicts between public and private functions, but also public functions with other public functions," The report recommends clear clarifications on whether conflicts of interest are potential or also perceived.

So, we will have to look at other, similar, organisations that have more stringent policies in this field. The European Food and Safety Authority (EFSA) seems to be a good candidate. In June 2017, EFSA, after a long history of accusations of CoI's, sharpened its definition and its policy to avoid CoI's.

EFSA defines a conflict of interest as "any situation where an individual has an interest that may compromise or be reasonably perceived to compromise his or her capacity to act independently and in the public interest in relation to the subject of the work performed at EFSA".

This definition is also somewhat broad and vague. EFSA's solution was to set clear rules to which its experts have to comply. For example: Research funding from the private sector benefiting EFSA's experts should not exceed 25% of the total research budget.

The EFSA-rules are minimum requirements. According to *Corporate Europe Observatory* they are not strict enough to completely avoid conflicts of interest. So, it is reasonable to say that

ICNIRP, that presents itself as an independent, scientific advisory board, should, at the very least, comply with the EFSA rules.

In this paper, we will therefore:

* Give an overview of the history and all existing knowledge on the independence of, and the conflicts of interest within, ICNIRP. These chapters provide the context in which we have a closer look at the ICNIRP-members.

* Try to identify all the potential sources of conflicts of interest of ICNIRP-members. Such as: research funding from the private sector; financial investments in, and employment by, telecom business operators; consultancy work for the telecom industry.

* Try to find out if the ICNIRP-members comply to the EFSA-rules on conflicts of interest and give an assessment on the independence of ICNIRP.

These are the ICNIRP experts whose professional backgrounds we will research (see the portraits of each member in Part V):

As from December 2019, the composition of the ICNIRP Commission for the term of office 2020-2024 is as below. The new term of office starts in May 2020.

MEMBERS OF THE ICNIRP COMMISSION:

GUNDE ZIEGELBERGER (SCIENTIFIC SECRETARY), GERMANY
RODNEY CROFT (CHAIR), AUSTRALIA
ERIC VAN RONGEN (VICE-CHAIR) , THE NETHERLANDS

TANIA CESTARI, BRAZIL
NIGEL CRIDLAND, UNITED KINGDOM
GUGLIELMO D'INZEO, ITALY
AKIMASA HIRATA, JAPAN
ANKE HUSS, NETHERLANDS
KEN KARIPIDIS, AUSTRALIA
CARMELA MARINO, ITALY
SHARON MILLER, USA
GUNNHILD OFTEDAL, NORWAY
TSUTOMU OKUNO, JAPAN
MARTIN RÖÖSLI, SWITZERLAND
SOICHI WATANABE, JAPAN

MEMBERS WHO HAVE LEFT THE ICNIRP COMMISSION IN MAY 2020
Maria Feychting
Adèle Green
Zenon Sienkiewicz

MEMBERS OF THE SCIENTIFIC EXPERT GROUP (SEG):

JACQUES ABRAMOWICZ - PG COSMETICS, PG ULTRASOUND
ANSSI AUVINEN - PG DATA GAPS
CHRISTIAN CAJOCHEN - PG SHORT WAVE LIGHT
JOSE GOMEZ-TAMES - PG HF DOSIMETRY REVIEW
PENNY GOWLAND - PG DATA GAPS
JOHN HANIFIN - PG SHORT WAVE LIGHT
JUKKA JUUTILAINEN - PG DATA GAPS
KEN KARIPIDIS - PG COSMETICS, PG DATA GAPS
MASAMI KOJIMA - PG LASER POINTERS
ILKKA LAAKSO - PG HF DOSIMETRY
ISABELLE LAGROYE - PG DATA GAPS
SARAH LOUGHRAN - PG SHORT WAVE LIGHT, PG HF GUIDELINES
JACK LUND - PG LASER GUIDELINES
SIMON MANN - PG HF DOSIMETRY
RÜDIGER MATTHES - PG HF DOSIMETRY
JOHN O'HAGAN - PG LASER GDL, PG LASER POINTERS, PG LED, PG SHORT WAVE
CHIYOJI OHKUBO - PG DATA GAPS
MARGARETHUS PAULIDES - PG HF DOSIMETRY
KENSUKE SASAKI - PG HF DOSIMETRY REVIEW
DAVID SAVITZ - PG ULTRASOUND
KARL SCHULMEISTER - PG DATA GAPS, PG LED, PG LASER GDL, PG POINTERS
DAVID H. SLINEY - PG LASER GDL, PG LASER POINTERS, PG LED, PG SHORT WAVE LIGHT
RIANNE STAM - PG COSMETICS
BRUCE STUCK - PG HF GDL, PG DATA GAPS, PG LED, PG LASER POINTERS, PG LASER GDL
JOHN TATTERSALL - PG HF GUIDELINES
TIM TOIVO - PG COSMETICS
ANDREW WOOD - PG DATA GAPS, PG HF DOSIMETRY
TONGNING WU

I- Historic overview of ICNIRP and accusations of COI

In this chapter, we give an overview of the history of ICNIRP as an organisation and examples of accusations of Conflicts of Interests (COI) and other controversies concerning the organisation's work. The authors do not want to suggest that this overview is, by any means, complete or comprehensive.

About ICNIRP's history, on its website, the organisation simply states that its beginnings go back to 1973 "when, during the 3rd International Congress of the International Radiation Protection Association (IRPA), for the first time, a session on non-ionizing radiation protection was organized. In 1977 the International Non-Ionizing Radiation Committee (INIRC) was created. This Committee was the immediate forerunner of ICNIRP that was chartered as an independent Commission in 1992 during the IRPA 7th International Congress."

In a speech in Rio de Janeiro, in 2008, Paolo Vecchia, the Italian former ICNIRP-chair (2004-2012), explained in more detail: "In June 1974, IRPA President, Italian Carlo Polvani (1973-1977), proposed "a possible role of IRPA in establishing criteria and standards in the field of health protection against non-ionizing radiations" and the IRPA Executive Council decided to set up a Working Group to review the health protection problems arising from different non-ionizing radiation (NIR)."

One could argue that IRPA itself, and then much later it's spin-off ICNIRP, came into existence as a "fall-out" of the first US atomic bomb testing. On its website, on the subject of its historical background, IRPA states: "Before the Second World War, radiation protection had been a largely secondary concern of radiologists and radiological physicists. With the concentration of effort under the Manhattan Project it was soon realised that this would involve working with quantities and types of radiation and radioactive materials that had not previously been envisaged. As a result, a distinct group of scientists within the project were assigned full time to what was termed "Health Physics"."

In an article from 2017 on the history of of ICNIRP, at the occasion of it's 25th anniversary founder Mike Repacholi wrote: "Concern about health risks from exposure to non-ionizing radiation (NIR) commenced in the 1950s after tracking radars were first introduced during the Second World War. Soon after, research on possible biological effects of microwave radiation in the former Soviet Union and the U.S. led to public and worker exposure limits being much lower in Eastern European than in Western countries, mainly because of different protection philosophies." As we will see further in this chapter this divide between Russia and the West on safety measures on non-ionizing radiation exists till today.

At the end of its conference in 1955, the US Atomic Energy Commission voted overwhelmingly to form a professional Health Physics Society and the first IRPA Congress was held in Rome between 5-10 September 1966. It is interesting to see that many of the 12 Executive Council Members of IRPA in 1966 remained in position for many years; a fact that echoes like a prelude to criticism that ICNIRP functions like an 'old-boys network'.

In 1974, IRPA President Polvani insisted that "a separate and independent International Commission on NIR Protection (later ICNIRP) should be established…The ICNIRP would look

to IRPA as the sponsoring international scientific organization in a similar way that ICRP looks to the International Congress of Radiology…. And "IRPA should consider broadening its institutional authority to include NIR".

So Carlo Polvani got what he wanted: the General Assembly amended the Constitution of IRPA so that it could "also apply its objectives and purposes in the field of non-ionizing radiation protection". Then the General Assembly created an International NIR Committee […] "with the objective of developing background documents and internationally accepted recommendations". This became INIRC, set up in 1977, that went on to become ICNIRP, in 1992. Already four years earlier, Mike Repacholi (more on him later), a member of IRPA, had begun writing the charter for ICNIRP which was signed in 1992.

But why elaborate so much on IRPA, before turning to ICNIRP itself? Critics often ask from where ICNIRP got its self-acclaimed international and institutional authority? Well, partly from IRPA, which still plays a role in the actual composition of ICNIRP. The IRPA Charter for the creation of ICNIRP, from 1992, says: "The election of the members of the Commission shall be made by the Commission from current members of the Commission and from nominations submitted by the Commission itself, the Executive Council of IRPA and the IRPA Associate Societies, with regard to an appropriate balance of expertise. Attention shall be paid to geographical representation."

At the end of the 15th International Congress of IRPA, planned for 11-15 May 2020, in Seoul, Korea, the new term of office of the new ICNIRP commission (2020-2024) would officially start. This occurred, despite the international congress in South-Korea being postponed until 2021 due to the corona-crisis. This international congress counts telecom companies of all kinds among its sponsors (platinum, silver, bronze as well as others). Since ICNIRP was born from IRPA, and that, like any parent, IRPA still exerts a strong influence over ICNIRP, and considering ICNIRP claims to function free of any vested interests, it seems important to us to look more closely at IRPA.

And maybe also because of the actual role that IRPA wants to play in the ongoing debate around safety and health in relation to EMF. Current IRPA-president, Roger Coates, writes that "a lot of effort over recent times has gone into preparing the IRPA Guidance for Engagement with the Public on Radiation and Risk". This seems to be the typical type of response given by bodies like IRPA, ICNIRP and others concerning public worries about possible health effects: *let's explain things better, because the public doesn't understand (…that everything is safe)*. It is the same kind of response given in the past by the nuclear sector when people started to become worried about nuclear safety issues (for example after Chernobyl).

Some governments – at various levels – try to put into practice a guiding principle of radiation safety, called "ALARA", which stands for "As Low As Reasonably Achievable". This principle means that even when being subjected to a small dose, if receiving that dose has no direct, practical or medical benefit, you should try to avoid it. IRPA-boss Roger Coates states that "the interpretation of what is 'Reasonable' in the implementation of optimisation of radiation protection is one of the key issues for our profession and is one of IRPA's current key themes. It is central to practical protection and is the dominant factor controlling exposures in any well-developed system of protection. But what does 'reasonable' mean?

There are growing concerns within our profession that we are giving more emphasis to 'as low as' and 'minimisation' rather than truly being 'reasonable'."

On the subject of safety: before Roger Coates became IRPA-president he had a life-long career in the British nuclear industry: he started working in 1975 at the Health Physics and Safety Department at the Sellafield site of *British Nuclear Fuels plc (*BNFL) and did so for over 30 years, "holding radiation protection roles covering operations, environmental protection and emergency planning. His responsibilities broadened to encompass nuclear safety, together with conventional safety and environmental issues. He completed his industry career as Director of Environment, Health and Safety for both BNFL and its British Nuclear Group subsidiary." Over the years, BNFL has had to face up to quite some issues in the field of safety and was the subject of a "damning report into the falsification of safety data at the Sellafield reprocessing plant" at the start of this century.

This year, on its website, IRPA published the first new safety guidelines of ICNIRP since 1998, of which ICNIRP-chair Van Rongen said, as we mentioned earlier: " The new guidelines provide better and more detailed exposure guidance in particular for the higher frequency range, above 6 GHz, which is of importance to 5G and future technologies using these higher frequencies. The most important thing for people to remember is that 5G technologies will not be able to cause harm when these new guidelines are adhered to."

Self-declared legitimacy

Since the signing of IRPA-charter in 1992, ICNIRP is based in Munich, Germany and registered as a self-governed NGO (non-governmental organisation) that was formally recognized as "an official collaborating non-state actor by the World Health Organization (WHO) and the International Labour Organization (ILO)." ICNIRP is consulted by the European Commission and is linked to many organizations engaged in NIR protection worldwide through diverse collaborative projects.

As mentioned in the introduction of this report, extensive reporting by *Investigate Europe*, in March 2019 (updated on June 10th 2020), showed that there are many close links between ICNIRP and other leading organisations in the field of health protection. Many ICNIRP-members are, or were, also members of one of these three scientific bodies (from which most radiation safety authorities in Europe and governments, seek their advice) and it is important to mention them again, because these are the bodies that guide government policies in most countries:

- The EU Scientific Committee on Health, Environment and Emerging Risk, SCENIHR / SCHEER.
- The World Health Organization (WHO) International EMF Project (IEMFP).
- The WHO Cancer Unit IARC, International Agency for Research on Cancer.

It is worth underlining, however, that IARC does not really fit into this "gang of four" because it has a much more critical and independent approach. IARC published a report in May 2011 which concluded that radiofrequency (RF) radiation is "possibly carcinogenic" to humans.

The IARC cancer classification includes all sources of RF radiation, of which the long-term exposure can come from mobile phone base stations, Wi-Fi access points, smart phones, laptops and tablets.

However, IARC may now have a solid reputation as independent scientific body, some years ago, IARC also got into trouble. Anders Ahlbom, senior professor of Epidemiology at the Karolinska Institute in Stockholm, and a long standing, influential member of ICNIRP (Commission Member and ICNIRP SCI working group (Epidemiology)), and ICNIRP Chairman from 1996 until 2008, was also part of the IARC panel of experts in 2011. Ahlbom was, until very recently, doing assessments of environmental health risks as chair of the Swedish Radiation Safety Authority's (SSM), the scientific council on electromagnetic fields, as a member of ICNIRP and of the EU advisory body SCENHIR.

But he was asked to step down from IARC after a journalist exposed him as being on the board of his brother's consulting firm in Brussels, which helps clients on telecoms issues. He had not made IARC aware of this. As the Swedish investigative reporter, Mona Nielsson, wrote: "Furthermore, Anders Ahlbom's brother, Gunnar Ahlbom, was for a long time a lobbyist for Swedish telecom giant Telia (previously TeliaSonera) in Brussels. At the same time Anders Ahlbom served as an "independent expert" on several important expert panels, in Sweden as well as at the WHO and EU. At a meeting organized by the European Commission in cooperation with GSM Association and Mobile Manufacturers Forum in Brussels in 2004, Anders Ahlbom was an invited expert to speak on health effects, while his brother Gunnar Ahlbom sat in the audience representing TeliaSonera."

There was, and is, more controversy and division on this topic within the WHO. In a 2017 article, "A hard nut to crack" , professor Lennart Hardell draws attention to a Fact Sheet issued by WHO in June 2011, only two months after the IARC's report adapting the cancer classification of RF radiation, which stated that "to date, no adverse health effects have been established as being caused by mobile phone use". According to Hardell, this statement was "not based on scientific evidence at that time on a carcinogenic effect from RF radiation. And it was certainly a remarkable conclusion by WHO since IARC is a part of WHO, although seemingly independent". And he goes on to conclude: "Considering the WHO statement of 'no adverse health effects' the aim might have been to undermine the IARC decision and give the telecom industry a 'clean bill' of health."

One of the main reasons for this schizophrenic approach within the WHO is to be found in the figure of ICNIRP-founder, Mike Repacholi, and the WHO's International EMF Project, IEMFP) (see more below). At least four ICNIRP-members were, or are, also members of the WHO-EMF Group.

In January 2019, in the German newspaper Der Tagesspiegel, investigative journalists described ICNIRP as "a Cartel", that systematically refutes all studies that show possible harm: "And no radiation protection agency, no EU commissioner and no minister, contradicts this. For European governments and their authorities, the 13 members of the self-appointed Commission act as a kind of force majeure. But why? Why are all the warners, even prominent figures like the panel of experts for the US Health and Safety Executive, not heard?"

The Investigative journalists describe an “astonishing phenomenon: the members of ICNIRP are simultaneously active in all the relevant institutions and thus have control over the official discourse.” They then go on to note that, legally speaking, ICNIRP is an association that auto-controls itself and thus avoids dissenting opinions, but in the first instance, the connection with the German state begins with the chosen address of ICNIRP which is the same as the German Federal Office for Radiation Protection (BfS).

Is it just a strange coincidence that ICNIRP’s secretariat is located in the building of the BfS in Munich. The scientific coordination for/of/within? ICNIRP has, for the last few years, been the responsibility of a BfS official: Gunde Ziegelberger. “Her predecessor even chaired the club until 2016. At the same time, the German government supports the NGO of scientists with about 100,000 euro a year. The spokesperson rejects the impression that the private organization is almost part of the German authority as "not applicable". The office only supports the international network of research, she said. Moreover, the ICNIRP is officially recognised by the WHO, which gives it legitimacy.”

We have asked Mrs Ziegelberger via email if she would agree to answer our questions on ICNIRP in writing, but we have, to this date, received no response (the ten questions can be found in Annex I)

This self-declared sense of legitimacy was carefully created by the Australian scientist, Michael Repacholi, who co-founded ICNIRP and also, a few years later, in 1996, the EMF Project of the WHO (officially the WHO’s International EMF Project, IEMFP) of which he became the head. The WHO’s International EMF Project (IEMFP) basically based itself on ICNIRP’s guidelines and by doing so gave itself a “quality label”.

ICNIRP under Michael Repacholi’s chairmanship

Since 1978, the Australian biophysicist, Repacholi, has been a member of the International Non-Ionizing Radiation Committee (INIRC), a part of the International Radiation Protection Association (IRPA), and between 1988—1992 he was chairman of INIRC, which then became into ICNIRP. Between 1996 and 2006, Repacholi called the shots at the WHO by creating, and then leading, the WHO EMF Project, to study the health effects of electric- and magnetic-field radiation (EMF).

So, almost simultaneously with his leadership of ICNIRP, Repacholi was able to set up the EMF Project of the WHO (officially the WHO’s International EMF Project, IEMFP) in 1996, and became its head (see more below) until 2006. From the very beginning, the WHO EMF Project and ICNIRP have been intertwined, as Louis Slesin wrote in *Microwave News*. Given the central role of Repacholi, it might explain why, from very early on, ICNIRP was officially recognized by the WHO. From 1996 until today, Repacholi has been “Member Emeritus” of ICNIRP and today, still has access to the organisation he founded.

As early as 1992, ICNIRP adopted Repacholi’s 1984 IRPA proposal that the only health issue to address in standard setting was the short-term effects due to the absorption of RF/MW energy of sufficient power to be converted to heat, based on the IEEE’s (Institute for Electrical and Electronic Engineers) Radiofrequency standard philosophy. Since then it seems to be carved in stone that ICNIRP only recognises the ‘thermal effects’ of radiation as a

serious concern. This is a crucial element to understand the position of ICNIRP, it was built on the logics and thinking of electrical and electronic engineers and completely lacking biomedical expertise.

In 1998, ICNIRP published its first "Guidelines on limits of exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz)", still largely produced under the chairmanship of Repacholi.

A fierce and long-standing critic of the first ICNIRP guidelines was Dr Neil Cherry, Associate Professor of Environmental Health. In November 1999, Dr Cherry was invited by the Ministry of Health/Ministry for the Environment of New Zealand to carry out a peer-review of the proposal to adopt the ICNIRP guidelines for cell sites in New Zealand.

Cherry: "The ICNIRP guidelines were covered by a published assessment in 1998. This review shows that the assessment had ignored all published studies showing chromosome damage. It was highly selective, biased and very dismissive of the genotoxic evidence and the epidemiological evidence of cancer effects and reproductive effects. The assessment gives the strong impression of being predetermined in the belief that the only effects were from high exposures that cause electric shocks and acute exposures that cause tissue heating. For, example, they cite two studies saying that they do not show any significant increased effects of Brain/CNS cancer from microwave exposures when the actual published papers, Grayson (1996) and Beall et al. (1996), both do show significant increases of Brain/CNS cancer."

In September 2000, he presented evidence of Health Effects of Electromagnetic Radiation to the Australian Senate Inquiry into Electromagnetic Radiation. The Inquiry Chairperson, Senator Lyn Allison, described Cherry's evidence as the only independent professional evidence with no relation to industry. The conclusions from this evidence are strongly contrasted with the position of Dr Michael Repacholi, the WHO, ICNIRP, the Australian Radiation Laboratory and many other organisations around the world.

Twenty years ago, Cherry said: "This issue has been so politicized. There are two major casualties, the truth and public health. On these matters, I have no respect for the position of ICNIRP, nor that of the WHO. The WHO position is taken solely by Dr Repacholi. ICNIRP is a small self-appointed, self-promoted group that claims standing by having WHO recognition. In other words, a body formed in part and led by Dr Repacholi, claims its standing by being recognized by Dr Repacholi."

Cherry used harsh words for INCIRP under Repacholi's chairmanship. "They consistently misquote and misrepresent the published research results. They reject all epidemiological evidence because every single epidemiological study occurs with mean exposure levels and orders of magnitude below their thermally-based standard. They are highly selective, using only a small proportion of the available studies in order to construct and defend their own case. They prefer author's conclusions that there are no effects, even when the data and analysis in the paper clash with this and contradict it. They dismiss large, reliable and well-defined studies as ill-defined and unreliable. They state that studies don't show significant increases in CNS cancers when they actually do, even when the papers include significant dose-response relationships. Both the WHO and ICNIRP, under Dr Repacholi's leadership, have maintained the thermal view to the present, despite the large and ever-growing body of scientific research that firmly and conclusively challenges this."

He also accused Repacholi of maintaining close links with industry. “He not only appeared in New Zealand in two court cases for industrial clients, in Vienna he was taken to an industry sponsored press conference where he stated that there was no evidence that GSM cell phones were hazardous to health. At the conference, he presented his paper on the Telstra (**Telstra** is Australia's largest mobile network operator and telecom company) funded project that showed that GSM cell phone radiation at quite low non-thermal levels, doubled the cancer in mice. When challenged by the conference chairman, Dr Michael Kundi, Dr Repacholi said that a study is not evidence until it is replicated. The conference rejected this. A study is evidence. Replication provides confirmation and establishment.”

The fact is that Repacholi has followed a remarkable career path, from member of IRPA and working in an Australian hospital, to holding a dominant position in the international debate on EMF risks. He also developed as a scientist, from publishing a study in 1997 on lymphoma incidence in mice exposed to RF radiation, to becoming a consultant for telecom and power companies ten years later.

In 2017, he published ‘A History of the International Commission on Non-Ionizing Radiation Protection (ICNIRP)’ in the scientific review *Health Physics*, in which he stated: “ICNIRP’s guidelines have been incorporated into legislation or adopted as standards in many countries. While ICNIRP has been subjected to criticism and close scrutiny by the public, media, and activists, it has continued to issue well-received, independent, science-based protection advice. This paper summarizes events leading to the formation of ICNIRP, its key activities up to 2017, ICNIRP’s 25th anniversary year, and its future challenges.”

It is quite revealing that Repacholi writes, “ICNIRP has been subjected to criticism and close scrutiny by the public, media, and activists”, and yet, forgets to mention, *and also by scientists*. Because, since the first publication of guidelines by ICNIRP in 1998, there has been a never-ending stream of critical academics publishing harsh analysis on the scientific work of ICNIRP. The issue is that Repacholi has not only been a dominant figure, but also a very divisive figure, in the international EMF-debate and he has been able to make sure that independent scientists who do not agree with the ICNIRP-dogma of ‘thermal effects only” have not become part of ICNIRP nor of the WHO EMF Project.

The fact that, in his article for the 25th anniversary of ICNIRP, Repacholi makes no mention of the criticism and close scrutiny by scientists is quite telling. Because basically, the story of ICNIRP and the ongoing controversy and ever deeper divisions within the scientific community in the EMF-debate, started around the persona of Michael Repacholi himself.

‘Good science’ and the EMF Project (IEMFP)

As we have stated above, Repacholi was not only ICNIRP chairman, but also the leader of the WHO EMF Project. In his own words: “The WHO established the International EMF Project to provide a mechanism for resolving the many and complex issues related to possible health effects of EMF exposure. The Project assesses health and environmental effects of exposure to static and time varying electric and magnetic fields in the frequency range 0 - 300 GHz, with a view to the development of international guidelines on exposure limits.”

In 1999, Repacholi published the Proceedings of an International Seminar on EMF Risk Perception and Communication that took place in Canada. The event was not only sponsored by the WHO, some government ministries and the Faculty of Medicine at the University of Ottawa, but also by the Cellular Telephone Industry Association, the Canadian Wireless Telecommunications Association and some electricity companies. The almost 300-page document published by Repacholi's "International EMF Project" (part of the WHO's Department of Protection of the Human Environment) kicks off with this statement: "Possible health effects of exposure to electromagnetic fields (EMF) have led to concerns among the general public and workers that appear to go well beyond those that are attributed to well-established risks. It is necessary to understand why this occurs and to deal with it through an effective communications programme. People have the right to access reliable, credible and accurate information about any health risks from EMF exposure."

In his review, "A hard nut to crack", professor Hardell writes: "Michael Repacholi immediately set up a close collaboration between WHO and ICNIRP (being head of both organizations) inviting the electric, telecom and military industries to meetings. He also arranged for large part of the WHO EMF project to be financed by the telecommunication industry's lobbying organisations; GSM Association and Mobile Manufacturers Forum, now called Mobile & Wireless Forum (MWF)." Hardell states that Repacholi acted like "a representative for the telecom industry while responsible for the EMF health effects department at the WHO"

An investigative article in US magazine, *The Nation*, stated: "Although Repacholi claimed on disclosure forms that he was "independent" of corporate influence, in fact Motorola had funded his research: While Repacholi was director of the WHO's EMF program, Motorola paid $50,000 a year to his former employer, the Royal Adelaide Hospital, which then transferred the money to the WHO program. When journalists exposed the payments, Repacholi denied that there was anything untoward about them because Motorola had not paid him personally."

According to *The Nation*, "eventually, Motorola's payments were bundled with other industry contributions and funnelled through the Mobile and Wireless Forum, a trade association that gave the WHO's program $150,000 annually. In 1999, Repacholi helped engineer a WHO statement that "EMF exposures below the limits recommended in international guidelines do not appear to have any known consequence on health."

In a *Microwave News* article, Repacholi claims that he always followed the WHO rules on funding and that, "NO funds were EVER sent to me." But the article's author, Louis Slesin goes on to say that "this is financial *legerdemain*. As *Microwave News* has previously reported, Repacholi arranged for the industry money to be sent to the Royal Adelaide Hospital in Australia, where he used to work. The funds were then transferred to the WHO. Seven years ago, Norm Sandler, a Motorola spokesman, told us that, "This is the process for all the supporters of the WHO program." At the time, Motorola was sending Repacholi $50,000 each year. That money is now bundled with other industry contributions and sent to Australia by the Mobile Manufacturers Forum (MMF), which gives the project $150,000 a year."

A scientist who is very critical about the activities of Repacholi is American Professor Andrew A. Marino (who used to work at the departments of Orthopedic Surgery, Neurology, and

Cellular Biology & Anatomy at the LSU Medical School in Louisiana) wrote: "In 1996 the World Health Organization began what it said was a program to assess the scientific evidence of possible health effects of EMFs. But the project was corrupted from the start because it was controlled by the power- and cell-phone companies in the industrialized countries. The companies designated Michael Repacholi as the project head. He had long been a consultant and spokesman for power companies, so it was unrealistic to expect him to conduct an open and honest inquiry, but his performance in office was even more miserable than could have been anticipated based on his known conflict-of-interest."

Marino: "While heading the EMF program at WHO, Repacholi dealt almost exclusively with experts on the payroll of cell-phone and power companies. Scientists who disagreed with the viewpoint of the EMF companies were excluded from the EMF evaluation process. The public was also excluded from participation even though it was a major stakeholder in the EMF debate. Only pro-industry spokesmen were heard in Repacholi's star-chamber processes, which ultimately resulted in reports and evaluations that exonerated the companies from any responsibility for human disease produced by their EMFs."

Marino saw Repacholi at the Annual Meeting of the Bioelectromagnetics Society (BEMS) in Cancun, Mexico, in June, 2006: "The Mobile Manufacturers Forum, a consortium of the world's major cell-phone companies, were "Gold Sponsors" of the BEMS meeting, and the leaders of BEMS, had invited Repacholi to give a talk entitled "Results from 10 Years of WHO's International EMF Project," which he delivered at a plenary session of the meeting. Unsurprisingly, his talk was a paean to his EMF activities at WHO. He was proud of having successfully stemmed the tide of concern regarding the link between environmental EMFs and other human diseases, and of having defended the principle that man-made environmental EMFs were harmless. He touted model legislation that he had drafted, and said that he hoped it would be enacted by various governments so that the fact that environmental fields were safe would be enshrined in law."

In 2006 Repacholi stepped down as director of WHO's EMF Project.

Not much later *Microwave News* announced: "It's Official: Mike Repacholi Is an Industry Consultant And He's Already in Hot Water": "Just months after leaving his post as the head of the EMF project at the World Health Organization (WHO), Mike Repacholi is now in business as an industry consultant. The Connecticut Light and Power Co. (CL&P), a subsidiary of Northeast Utilities, and the United Illuminating Co. (UI) have hired Repacholi to help steer the Connecticut Siting Council away from a strict EMF exposure standard."

To strengthen his testimony on behalf of the two electric utilities, Repacholi cited the findings of an unfinished WHO report —Environmental Health Criteria (EHC)— on EMF risks. Twenty invited experts drafted this report at a meeting in Geneva in October 2019. The final version was expected to be made public months ago but it's still being edited by the WHO staff.

According to Chris Portier, who chaired the expert EHC panel for the WHO, Repacholi has misrepresented the group's conclusions: "The paraphrasing sometimes has gone a bit far and may be misleading". Portier is the associate director for risk assessment at the National Institute of Environmental Health Sciences (NIEHS)." (see below).

Portier cites a couple of examples. For example, in a summary of the WHO report, Repacholi states that the EHC panel concluded that "The epidemiological evidence cannot be used as a basis for standards (exposure limits)". Portier retorts, "Such a statement is absurd, since they obviously can be used."

Repacholi has since also been involved in an industry propaganda video and interviews with GSM Association and Hydro Quebec where he clearly speaks in favour of the telecommunications and the power industries, respectively.

A year later, in 2007, *Microwave News* reported that "Mike Repacholi has now revealed that up to half of the funds raised for his EMF Project came from industry. This admission was made in an interview with *Resource Strategies Inc*. in an effort, he states, to "set the record straight." While Repacholi has acknowledged in the past that he raised funds from industry, the extent of the industry support is much greater than anyone has previously suspected. Repacholi has never disclosed how much money he received nor from whom. He insists that the EMF Project was not "influenced by industry."

According to an e-mail seen by *Microwave News*, Repacholi touts the interview as an example of "where the press finally got it right": "*Resource Strategies*, however, can hardly be considered "the press" in the usual meaning of the term. *Resource Strategies* is a corporate consulting firm that prepares briefing papers for clients, which are almost exclusively in the wireless and electric utility businesses. Among them are *EPRI, FGF, GSM Association* and *MMF*. All of these industry groups supported the EMF Project during Repacholi's tenure. And to bring it all full circle, the WHO is also on *Resource Strategies*' client list."

Some current ICNIRP members, such as the new chair, Rodney Croft, also declare doing work for EPRI.

Researcher Don Maisch wrote that Repacholi harmed the credibility of the WHO: "It is acknowledged that in an ever increasingly globalized world the reliance on international organisations to set standards to protect public health is an irrefutable fact of modern life. It is also a fact that international organizations charged with this task need to be "eternally vigilant" to ensure that their organisations are not co-opted by vested interest groups – as exampled by Big Tobacco and WHO. However, when it comes to non-ionizing radiation issues (in this case for power frequency health risk assessment) the evidence is clear that Michael Repacholi has used his standing in both WHO and ICNIRP to stack the WHO's Environmental Health Criteria Task Group for power frequency exposures with representatives of the power industry in contravention of WHO policy."

Maybe one of the most telling episodes in the professional life of Repacholi is his open fight with his former boss, Gro Harlem Brundtland, who was director-general of the WHO. In interviews and a speech, Brundtland admitted that she is 'electrically sensitive': "I never place a mobile phone next to my head because in one second I would develop a bad headache." Repacholi was not amused. In 2012, several Norwegian newspapers reported that the "Former head of WHO's EMF project and ICNIRP chairman says that Brundtland has created "fear of mobiles" in the population". He offered to examine her, as if she had a psychological problem.

Very seldom were critical voices heard within the WHO. From the minutes of the Sixth International Advisory Committee meeting in May 2001, we read that Russian professor Yuori Grigoriev (the one from the 'angry letter' mentioned below) tabled a document outlining EMF activities in Russia, and the difficulties with standards harmonization "particularly because of the inadequate consideration of non-thermal effects by ICNIRP and other national authorities".

Dr Paolo Vecchia, of the National Institute of Health in Italy, and later ICNIRP chair, reacted to this by saying that "it is important to be able to recognize what good science is. WHO should be a reference point or clearinghouse for good science and good scientific review. It is important to recognize that science and legal measures follow the technology – it is not possible to do a mobile phone epidemiological study before the introduction of the technology! Given the pace of new technological development it is not possible, even now, to envisage the complete set of new research that will be needed."

Vecchia also claimed to be personally very concerned about 'defensive science', speaking of over-cautiousness and an over-emphasis on uncertainties. "Scientists should be more confident 'about the state of art'". He is now doing consultancy work and speaks at Telecom-conferences.

IEEE/ ICES

In 2008, Vecchia wrote: "Guidelines for safe exposure to electromagnetic fields have also been developed by other international organizations, in particular the Institute of Electrical and Electronics Engineers (IEEE). Apart from some differences in terminology and numerical values of the limits, these guidelines are based on the same methodological approach, the same structure, and the same scientific database as ICNIRP."

In his thesis on "an examination of the manipulation of telecommunications standards by political, military, and industrial vested interests at the expense of public health protection" ORSAA-member and scientist, Don Maisch, compares the ICNIRP and IEMFP with the American based IEEE. It is interesting because while ICNIRP claims to be free from the influence of private interests, IEEE/ICES has always openly had members of the military and of the telecom industry among its ranks.

Maisch writes: "On the part of both IEMFP and ICNIRP, a disregard for their own stated principles on independence from industry and following questionable criteria for evaluating science, suggests an agenda to cut off the scientific controversy over EMF human health hazards by less than scientific means. It could be argued that IEEE's openly industry and military dominated standard-setting process is at least more honest than WHO / ICNIRP masquerading as independent scientific voices free of vested interest machinations."

Dariusz Leszczynski, Adjunct Professor at the University of Helsinki, writes about conflicts of interest concerning ICES: "ICES, equivalent of ICNIRP, prepares safety recommendations for the exposures of users by radiation emitted by cell phones. Unlike ICNIRP, anyone can apply for membership of ICES and all members of ICES participate in the decision-making process. Sounds nice… Not a "private club" as ICNIRP where participation is by invitation only and the invitees have to have the same opinion on radiation safety – this helps in reaching unanimous decisions… But ICES has another problem that caused me, member of ICES for a

couple of years, to resign my membership in 2009. The problem is that the ICES membership is clearly dominated by scientists working or consulting for telecoms."

And in another blogpost Leszczynski wrote: "The membership of the IEEE-ICES-TC95 consists predominantly of the industrial scientists and the committee is chaired by C.K. Chou since the time he was employed by Motorola. This means that all safety standards being developed by IEEE-ICES-TC95 are, in practice, developed by the industry scientists for the use by the industry they are employed by. The industry scientists have the majority on the committee and upper-hand in any process involving democratic voting. To me this is clear Conflict of Interests".

In the portraits of ICNIRP chair, Croft, and co-chair, Van Rongen, we describe (from page 50) how they worked on establishing closer relations between ICNIRP and ICES.

From the minutes of a meeting by the IEEE/ICES TC95 working groups at a Motorola headquarters, a few interesting things got clear: In 2017 Repacholi was still a member of the "ICES literature systematic review working group". And ICES-chair Faraone Antonio from 'Motorola Solutions' proudly announced that "ICNIRP has delayed finalizing their conclusions to give full consideration of ICES's recommendations".

Former Motorola employee Chou stated at the same meeting on the interaction with World Health Organization (WHO EMF Project) that "in response to C-K Chou, the WHO has agreed to encourage international harmonization of RF Safety Limits, especially between ICNIRP and ICES"

And concerning the WHO EMF Project, Hardell describes how Repacholi recruited Emilie Van Deventer to the WHO EMF Project in 2000, and to this day, she remains project manager at WHO for the EMF project: "She has been a long time member of the industry dominated organization Institute of Electrical and Electronics Engineers (IEEE). IEEE has prioritized international lobbying efforts for decades especially aimed at the WHO." Hardell states that Van Deventer is an electrical engineer and has no formal or earlier knowledge in medicine, epidemiology or biology, so it is surprising that she was selected for such an important position at the WHO. Hardell: "The very same year she was recruited to the WHO EMF Project, Toronto University Magazine wrote about Emilie van Deventer's work, stating that it was 'invaluable' to industry: 'The software modelling done by teams like van Deventer's is invaluable.' 'The industrial community is very interested in our research capabilities,' says Van Deventer. 'It always needs to be working on the next generation of products, so it turns to universities to get the research done'."

The importance of this work is reflected in the research funding van Deventer and her team received from the Natural Sciences & Engineering Research Council of Canada (NSERC), Communications & Information Technology Ontario (CITO), and their major industrial partner, Nortel. "We are fulfilling a very real need in the industry today, which will only increase as technology creates more opportunity. In the process, consumers will continue to enjoy faster computers, lighter cell phones, smaller electronic organizers and the vast array of other electronic gadgets the high-tech world has to offer."

In 2016, during a seminar at the SSI, concerning health effects of EMF, former Swedish investigative journalist, Mona Nilsson, asked both Emilie van Deventer, Head of the WHO

EMF Project, and Eric van Rongen, the then chair of the ICNIRP, "whom the citizens should believe: them or the opinion of 220 scientists who signed an Appeal submitted to the United Nations and the WHO?". Both Van Rongen and Van Deventer answered the question without defending their position. Apparently, neither Van Rongen or van Deventer are willing to fully defend the reliability of the evaluation of science by ICNIRP, because as Leszczynski points out, neither of them said that ICNIRP evaluation of science is reliable and that the Appeal's conclusions are unreliable. "This clearly demonstrates that there is no scientific consensus on the health effects of radiation emitted by wireless communication devices. This situation should be taken into consideration when the WHO selects expert group for preparation of the final version of the Environmental Health Criteria for RF-EMF. Scientists with diverse scientific opinions should and must be appointed in order to facilitate an unbiased scientific debate."

We have sent questions to Van Deventer, but have, to date, received no answer.

Angry Russian letter

Although ICNIRP was recognised as "an official collaborating non-state actor by the World Health Organization (WHO) and the International Labour Organization (ILO)", from the early days, ICNIRP has also been criticized for industry-bias and indisputable situations of conflict of interest.

Hardell notes that the Ethical Board at the Karolinska Institute in Stockholm, Sweden, concluded, already in 2008, that "being a member of ICNIRP may be a conflict of interest that should be stated officially whenever a member from ICNIRP makes opinions on health risks from EMF."

Nevertheless, for the WHO, this does not appear to pose a problem. After the IARC publication in 2011, the WHO announced a new 'formal risk assessment' in 2012, which was launched in 2014 and was then open for public consultation until the end of 2014.

The WHO stated "the drawing of conclusions from the literature and the drafting of these chapters is the remit of a formal Task Group that will be convened by WHO at a later stage in the process."

Hardell disclosed that "it turned out that of the six members in the WHO Core Group, four are active members of ICNIRP and one is a former member." Indeed, in a research paper from 2016, Sarah J Starkey concludes that "the anticipated WHO Environmental Health Criteria Monograph on Radiofrequency Fields, due in 2017, is being prepared by a core group and additional experts, with 50% of those named, being, or having been, members of AGNIR or ICNIRP (Table 2)."

In another research paper, from 2017, Hardell notes: "It is striking how ICNIRP has infiltrated the WHO Monograph core group, making it less likely that the conclusions in that Monograph will differ from ICNIRP's conclusions." And according to him, only one person seems to be independent of ICNIRP and "several persons also have affiliation(s) to other advisory groups, authorities and/or committees. Six of the 20 additional experts are affiliated with ICNIRP".

In March 2017, professor Oleg A. Grigoriev, Chairman and Head of the Scientific Department of Non-Ionizing Radiation, Federal Medical Biophysical Centre of Federal Medical Biological Agency (RNCNIRP) of Russia wrote an angry letter to Maria Neira, Director of Public Health and Environment at the WHO, in which he openly attacks ICNIRP: "It has just come to our attention that the WHO RF Working group consists mainly from present and past ICNIRP members. In general, the WG is not balanced and does not represent the point of view of the majority of the scientific community studying effects of RF. In particular, the private self-elected organization, ICNIRP, similar as majority of the current WHO RF WG members, does not recognize the non-thermal RF effects, which represent the main concern of widespread exposure to mobile communication and upholding guidelines from 1996, which are based on RF thermal effects only."

The Russian scientist concludes that "the guidelines of ICNIRP are irrelevant to the present situation when majority of population over the world is chronically exposed to non-thermal RF from mobile communication. Based on multiple Russian studies and emerging number of studies coming from other countries, the Russian equivalent of ICNIRP has consistently warned against possible health effects from mobile communication. This point of view of RNCNIRP (Russian radiation protection agency) is supported by hundreds of new publications including well known recent RF studies in human and animals."

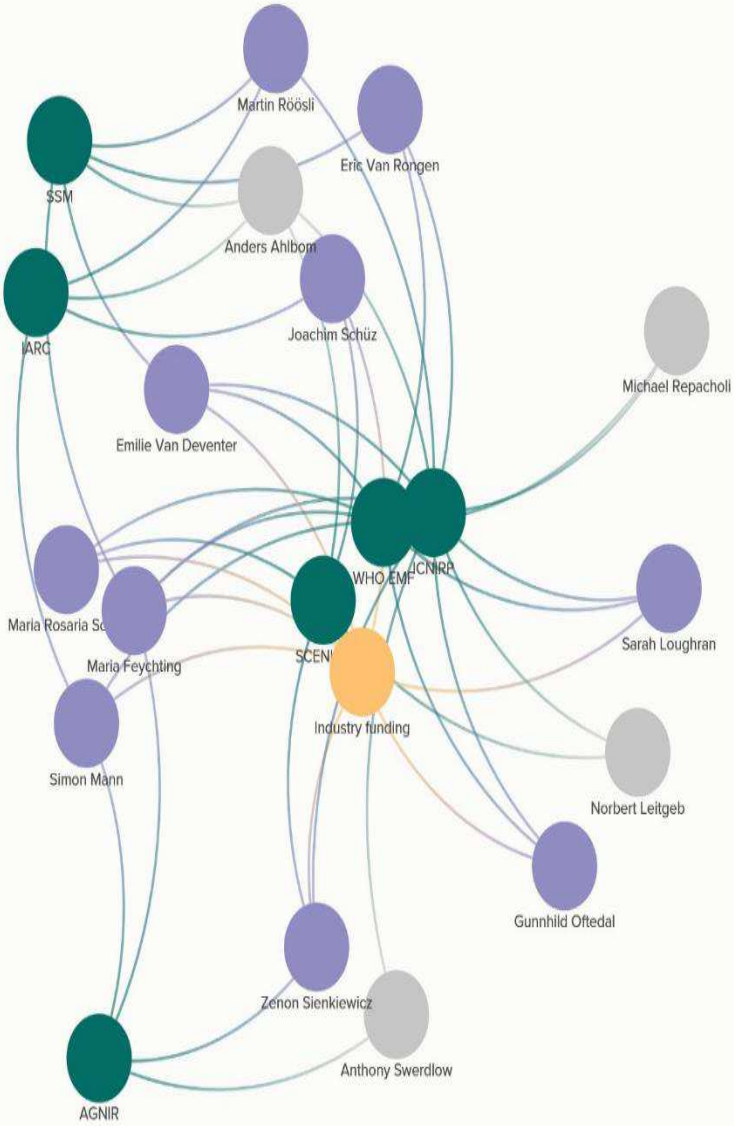
Apparently, this angry Russian letter, in addition to other outcries, did have some effect on the WHO, because it relaunched a Call for Expressions of Interest for systematic reviews (2020) for an 'Environmental Health Criteria Monograph': "The World Health Organization's (WHO) Radiation Programme has an ongoing project to assess potential health effects of exposure to radiofrequency electromagnetic fields in the general and working population. To prioritize potential adverse health outcomes, WHO conducted a broad international survey in 2018. Ten major topics were identified for which WHO will now commission systematic reviews to analyse and synthesize the available evidence."

We wonder if this time the WHO will try to avoid conflicts of interests and whether, for example, there will also be Russian experts and other non-ICNIRP affiliated scientists on the panels of experts.

Investigate Europe wrote that the conflicts in EMF research run deep: "Historically, science in this field has been associated with the telecom sector and the military. ICNIRP's safety limits primarily take into account the needs of the telecom industry, claims Dariusz Leszczynski, former long-time researcher at the Finnish radiation protection agency. In 2011, he sat on the committee of IARC, the cancer body of the World Health Organisation, when it decided that EMF is "possibly carcinogenic" to humans. ICNIRP's goal is to set safety limits that do not kill people, while technology works – so something in between", says Leszczynski."

Dariusz Leszczynski, has written about this many times on his blog and has often referred to an unbalanced expert composition: "ICNIRP can, and should, be considered as a "private club" where, members of the new Main Commission are selected by the members of the outgoing Main Commission. It is a self-perpetuating and self-promoting German NGO that is not accountable for its actions at all. Nobody controls it. Nobody supervises it. Nobody checks it for conflicts of interests. Nobody checks it for the scientific accuracy. In all what and how ICNIRP does, we, the general public, must rely on the self-assurances, from the

ICNIRP, that all is in order. One may ask whether such self-assurances are sufficient when ICNIRP is preparing advisories “enforced” world-wide by the WHO and applied by the numerous governments and by the multi-billion industry.”



The following Graphic – made by *Investigate Europe* shows the interlinkages between renowned ICNIRP-members and other scientific bodies. These groups, are to a large extent staffed by the same experts. “Of 13 ICNIRP scientists, six are members of at least one other committee. In the WHO group, this applies for six out of seven,” *Investigate Europe* writes.

III - Discussion & Controversies

An observation one could make based on what has been discussed above, is that ICNIRP is simultaneously one of the most powerful and one of the least-known non-governmental organisations (NGO's) in the world. Powerful, because for almost three decades, ICNIRP has enjoyed a monopoly in the regulation of exposure to EMFs through their guidelines thanks to the stamp of approval of the WHO. For the past 30 years, and currently, this advice and these guidelines, are to a large extent followed by governments all over the world. In every annual report, by any major telecom company, you will find references to ICNIRP in any discussion or statement on the safety of their mobile phones. ICNIRP garners huge influence worldwide, functioning on a modest yearly budget of around 140.000 euro, and yet ICNIRP is largely unknown by the general public.

ICNIRP presents itself, and is described by the European Commission and in the media, as an independent commission that gives advice based on scientific evidence. Our research shows that there are several reasons to question this (self)-image.

Biased composition

The composition of ICNIRP is very one sided. As one can read in the portraits of the members of the ICNIRP commission and of the Scientific Expert Group (SEG), they all share the same position on the safety issues: non-ionising radiation only poses a health threat at thermal levels.

Prominent ICNIRP-members therefore denounce the findings of the U.S. National Toxicology Program (NTP) that showed rats and mice contracted cancer when exposed to telephone radiation. In a scientific publication, Van Rongen and co-authors state, as we laid out in the portrait of the former chair of the ICNIRP-commission, that "substantial limitations (of the NTP-study) preclude conclusions being drawn concerning RF EMFs and carcinogenesis."

Professor Hans Kromhout of Utrecht University, who is leading a long-term study into the effects of mobile phone use on human health, and who is chairman of a special committee on Electromagnetic Fields of the leading Dutch Health Council, regrets the way INCIRP minimalizes the conclusions of the NTP study. "You can see that certain groups are trying to reason that away. But they are well-executed studies", he said in a Dutch newspaper.

According to Kromhout, a deep controversy divides the scientific community that researches EMF: "Two camps have arisen in science, with the two groups shouting at each other from their trenches. It has become impossible to conduct a normal conversation." This observation is also made by ORSAA-scientists.

And one of these two camps, is not represented at all inside ICNIRP. "It would seem that the Commission is composed only of 'non-believers," Kromhout wrote in an email to us. In the Dutch newspaper, he had earlier stated: "It's a bit of an opaque club. How candidates are elected is not clear. Call it self-indulgent. In that sense, it doesn't really have an independent status."

In more recent exchanges with us, he re-iterates that the use of the word "self-indulgent" is justified. He refers to the sentence in the ICNIRP Charter: "The election of the members of the Commission shall be made by the Commission from current members of the Commission and from nominations submitted by the Commission itself, the Executive Council of IRPA and the IRPA Associate Societies, with regard to an appropriate balance of expertise. Attention shall be paid to geographical representation." The first part – that it is the members of the Commission who elect its new members – puts the Commission at risk of remaining a closed circle made up only like-minded scientists.[3]

The unbalanced composition of ICNIRP is further demonstrated by the lack of expert-members with training and experience in medical and/or biological sciences. As one researcher pointed out, of the outgoing ICNIRP commission only one member was trained in medicine, and only three in biological sciences. Furthermore, the sole medical professional, Adele Green, was not an expert researcher in RF-EMR (with a single original research article back in 2005), but was specialised in UV-radiation and skin cancer. She also left ICNIRP in May 2020. It seems a good thing she has been replaced by Dutch scientist, Anke Huss, assistant professor at the Institute for Risk Assessment Sciences (IRAS) at Utrecht University (NL), who seems to be rather critical. Tania Cestari has replaced Adele Green ICNIRP in May 2020, although, like Green that she has collaborated with, her expertise seems to be on UV radiation in dermatology. Interestingly, a search on the PubMed database showed that she has no publications for radiofrequency or other EMFs so she is not an expert on wireless radiation.

The system of cooptation of ICNIRP and the resulting excessively homogeneous composition clearly favors such biases. In 2013, in his article "Not Entirely Reliable : Private Scientific Organizations and Risk Regulation - The case of Electromagnetic Fields", Gabriel Domenech Pascual, Professor Administrative Law at the University of Valencia, states in his conclusions : "That lack of plurality tends to reduce both the quantity and the quality of the available information that serves the basis of their judgments, to stifle critical dialogue, to exacerbate the common biases and positions of their members and to produce extreme outcomes, polarized in the direction of those biases and points of view."

We can safely say that ICNIRP has been, and is still lacking people with a relevant medical background and over represented by physical scientists, which may not be the wisest composition when your remit is to offer advice on human health and safety to governments around the world.

Dr. Chris Portier, former director of the National Center for Environmental Health and international expert in the design, analysis, and interpretation of environmental health data with a focus on carcinogenicity, writes to us that the ICNIRP Council and SEG "appear to have a very wide balance of experience". However, what they are lacking, according to Portier, "is representation by scientists who have a history of working in risk assessment for chemicals. This leads to their having different risk assessment approaches than the rest of the area."

[3] For a better understanding of IRPA and functioning of ICNIRP, we refer you to the historical section of this report

Portier argues that risk assessment for chemicals is “well-established and has been used for many, many years”. This standard of assessing risks of chemical substances, governs how to judge the quality of various types of scientific studies and how to incorporate them into the final risk assessment decisions.

Portier: “I have long felt that experts from EMF-research have been incorrectly arguing that this exposure is different and must be handled separately. But ionizing radiation is handled the same was as chemicals in risk assessment, why not EMF?” Portier states that ICNIRP could “expand their expertise in epidemiology and toxicology and experts who understand the challenges of biomedical study design and interpretation in a general sense.

And Portier states that “it would also be good to have a few scientists who are more outspoken about potential risks.” Portier writes that these improvements “would” challenge ICNIRP to “be exact about their dismissal of some of the positive findings” in research on health effect of EMF, that do exist.

The composition of ICNIRP is also one sided in another sense: there is a lack of representatives from the Middle East, Russia, China and India who have outstanding research contributions in the RF research and also (in many cases) have more stringent standards.

For Gabriel Domenech Pascual "this lack of plurality is not fortuitous at all, but caused by the system used to elect the members of the ICNIRP. As everybody knows, cooptation tends to produce homogeneous, conservative, immobile and not sufficiently innovative groups."

"This stands in sharp contrast with the principles underlying current European Union Law", Domenech Pascual adds. "As stated in the Communication from the Commission on the collection and use of expertise, pluralism is a determinant of the quality of the scientific advice. Therefore, “wherever possible, a diversity of viewpoints should be assembled. This diversity may result from differences in scientific approach, different types of expertise, different institutional affiliations, or contrasting opinions over the fundamental assumptions underlying the issue. Depending on the issue and the stage in the policy cycle, pluralism also entails taking account of multi-disciplinary and multi-sectorial expertise, minority and non-conformist views".

Various EMF-experts have pointed out on many occasions in the past years that ICNIRP is wrongfully dismissing certain scientific studies showing adverse health effects and sticking, in an almost dogmatic way, to the conviction that “non-ionising radiation poses no health threats and the only effects it has are “thermal”. Two leading experts, Kromhout and Portier confirm to us that ICNIRP is a closed, non-accountable and one-sided organisation. As concluded earlier, “a closed circle of like-minded scientists” has turned ICNIRP into a self-indulgent science club, with a lack of biomedical expertise as well as a lack of scientific expertise in risk assessment and risk management philosophies (similar to those used for ionizing radiation and for chemicals), which might lead to “tunnel-vision”.

Will world safety standards really be safe?

Several ICNIRP-members are, or were, also members of the International Committee on Electromagnetic Safety (ICES) of the IEEE. This is an organisation in which many people from the media and telecom industry and from the military are actively and openly involved. The former chair of the ICNIRP-commission was a member of an ICES-committee. As we mentioned in his portrait, ICES thanked Van Rongen for improving the relationship between ICES and ICNIRP and for his willingness to discuss the harmonisation of ICNIRP-guidelines and IEEE-exposure limits. In its latest published annual report (2016), ICES states: "ICES will maintain its collaborative relationship with ICNIRP with the goal of setting internationally harmonized safety limits for exposure to electromagnetic fields at frequencies below 300 GHz. This interaction with ICNIRP is considered a major step forward."

In 2016 Van Rongen invited members of ICES to give their comment on the new guidelines for HF Fields. And ICNIRP took these comments very seriously. In 2017 during the annual meeting of ICES it was stated that "ICNIRP has delayed finalizing their conclusions to give full consideration of ICES's recommendations".

The new chair of the ICNIRP-commission Croft was also member of ICES until December 2015. Seven other ICNIRP-scientists - Guglielmo d'Inzeo, Akimasa Hirata, Jose Gomez-Tames, Ilkka Laakso, Kensuke Sasaki, John Tattersall and Tongning Wu – were or are also members of an ICES-committee.

This clearly shows that ICNIRP has been working very closely with IEEE/ICES on the creation of the new RF safety guidelines that were published this year. And this implies that large telecom-companies as Motorola and others, as well as US military, had a direct influence on the ICNIRP guidelines, which are still the basis for EU-policies in this domain.

Kromhout comments that he was unaware of the fact that several ICNIRP-members also participate in ICES/IEEE. ICES/IEEE is not one of the organisations that is mentioned as a collaboration partner on the ICNIRP-website. On the subject of the IEEE, the Dutch professor writes that "this is not really an independent organisation when it comes to electromagnetic fields and health."

Portier sees the membership of ICES as a potential conflict of interest. He indicates as an example that the declarations of interests of some ICNIRP-members mention membership in ICES, but no mention of the travel costs associated with that membership being covered by ICES: "This has two consequences. Travel cost reimbursement is a perk and it could be removed if the member fails to give the right answer, hence a potential Conflict of Interests. Secondly, being a member in ICES gives industry access to the ICNIRP member which would not be available to the general public and can thus bias opinions."

A membership of and close cooperation of ICNIRP-members with ICES, which for several years held its annual meetings at a Motorola's branch, can be considered as a possible conflict of interest. As described, during the current leadership of ICNIRP, these ties got even closer "with the goal of setting internationally harmonized safety limits for exposure to electromagnetic fields".

Ties that bind

A lot of ICNIRP-scientists have also participated in research work that was funded, or partly funded, by the telecom industry.

The International Agency for Research on Cancer (IARC) has a strict policy when it comes to inviting scientists to assist it in the writing of the famous monographs – like the one from 2011, that classified radiofrequency electromagnetic fields as, "possibly carcinogenic to humans (Group2B), based on an increased risk for glioma, a malignant type of brain cancer associated with wireless phone use." In the final Monograph 2012 report, it is stated that each scientist must disclose pertinent research, employment, and financial interests during the past 3 years, unless that a grant from for example a company does not exceed more than 5% of total research budget: "All grants that support the expert's research or position and all consulting or speaking on behalf of an interested party on matters before a court or government agency are listed as significant pertinent interests."

In our introduction, we wrote that the European Food and Safety Authority (EFSA) has slightly less stringent member-selection criteria: "Research funding from the private sector benefiting EFSA's experts should not exceed 25% of their total research budget."

It seems that this percentage is not exceeded by most of the members of the ICNIRP-commission and Scientific Expert Group, insofar as we can trust their Declarations of Personal Interest. But these declarations are often not complete. Anssi Auvinen, for example, mentions that he received € 100,000 from the Mobile Manufacturers Forum for the Finnish section of the COSMOS-study. But he does not mention what percentage of his total research budget that amount constitutes. And Maria Feychting, former vice-chair of the ICNIRP-commission, did not mention any research support received from commercial entities in her Declaration of Personal interest, although a lot of her research actually was, as we showed in her portrait, funded by industry. Some of the member's DOI's are also somewhat out of date. For example, the last DOI available for Isabelle Lagroye, published on the ICNIRP-website, is dated October 2015.

The majority of ICNIRP-scientists did perform research partly funded by industry. But is this important information? As we argue in the introduction, we believe it is. Scientific publications, co-authored by two ICNIRP-scientists – Anke Huss and Martin Röösli, confirm the importance of funding. In 2006 and 2009 they did a systematic review of the effect of the source of funding in experimental studies of mobile phone use on health, and their conclusion was that, "industry-sponsored studies were least likely to report results suggesting (adverse health) effects".

And theirs is not the only study that showed this kind of bias. Portier agrees in writing to us that this is a problem: "There have been numerous studies of the differences in reporting from industry-funded research versus publicly-funded research that suggest a strong bias."

David O. Carpenter, professor of Environmental Health Sciences at the University at Albany, explains the mechanism behind this claim in the preface of the book Corporate Ties That Bind - An Examination of Corporate Manipulation and Vested Interest in Public Health: "One of the greatest problems in scientific discovery," he writes, "is the perversion that can result due to conflicts of interest. While there are other possible bases for conflicts of interest,

most are financial. Individual scientists may have financial conflicts of interest that influence the design of the studies they perform so that they obtain a result similar to that which they, or their funders, want. When funding for scientists comes from an organization or corporation with desires to present a clean bill of health to the public, there is strong motivation to give the funder what they want, if only to continue receipt of funding."

The Australian researcher, Don Maisch, claimed in his PhD-thesis, *The Procrustean Approach: Setting Exposure Standards for Telecommunications Frequency Electromagnetic Radiation* (2010), that the dismissal by ICNIRP of all studies that show health effects of non-ionizing radiation shows the influence industry exercises on ICNIRP: "Such dismissal may, on the surface, appear to be objective expert opinion, but an examination of ICNIRP's risk assessment processes finds, however, that power industry influence is endemic to the process. This influence appears to be aimed at ensuring economic protection for the industry against the need to spend enormous amounts of money on upgrading distribution networks as well as the risks of litigation if more restrictive limits were ever put in force.**"**

According to Maisch, the essence is that the thermal limitations of the IEEE standards and the ICNIRP RF Guidelines "can be said to be little more than an outdated artefact from a half-century ago, maintained by a scientific elite who have long staked their scientific credibility on maintaining that viewpoint. From their perspective, to retreat from that paradigm would be to admit that they had it wrong after all."

Ten years after Maisch' publication and many other similar criticisms, ICNIRP still adheres to the paradigm that the only proven effects are thermal. "ICNIRP appears to take into account only the warming of tissue and uncontrolled muscle contractions, although they claim in the most recent advice, that they also evaluated other mechanisms", writes Kromhout.

As many scientists and critical observers have pointed out, it seems as if ICNIRP members are either oblivious or ignoring scientific studies that find possible adverse health effects where there is an absence of heating. Even when some ICNIRP-members themselves acknowledge that industry-funding of scientific research tends to have less positive findings, and publicly funded studies – like the NTP-study – does find significant links between EMF and adverse health effects, this does not seem to influence one iota the views of ICNIRP-members.

A mixed bag of responsibilities

In an e-mail we received from Lloyd Morgan, Senior Research Fellow of the Environmental Health Trust and Director of the Central Brain Tumor Registry of the United States, is very critical of both ICNIRP and governments: "Who are ICNIRP? The International Committee on Non-Ionising Radiation Protection (ICNIRP) are a private, self-appointed body or NGO who together with the Advisory Group on Non-ionising Radiation (AGNIR) and Public Health England (PHE), have somehow ended up effectively setting microwave radiation exposure 'safety' standards for the populations of large parts of the world since the 1990s," he writes. "What amazes me, and simultaneously sickens me, is how did ICNIRP convince a large number of "independent" nations to adopt ICNIRP's so called "standards"?

Morgan suspects that high-level persons in the government's administration was "able to have the legislation passed because almost no-one in the government understood what was happening."

ICNIRP only publishes guidelines. It is then up to national governments to decide if they pass these guidelines into law. According to Lloyd Morgan, "that places the burden on each national government, should its citizens file a lawsuit".

Clearly, the Telecom sector as a whole, and the auctioning off of bandwidth and selling of Telecommunication licenses, are an important source of cash income for governments. The analogy with the Tobacco sector has often been made by scholars who study 'regulatory capture', but there is also an important similarity between the tobacco and telecom sectors in terms of their importance for State budgets.

The auctioning off of Radio frequency spectrums brings in billions of euros for European countries. Telecom companies also earn billions of euros thanks to these spectrum acquisitions, since 'owning the right' to use a specific radio frequency spectrum is an essential resource for telecommunication services such as mobile telephones, TV and radio broadcasting, satellite and broadband communications.

The *European 5G Observatory* notes that, "Germany's Federal Network Agency announced that the 5G auction, which started in March 2019, ended with 6.55 billion euros offered in total by the four bidders. *Deutsche Telekom* and *Vodafone* Germany criticized high prices of the country's auction". In the 5G Action Plan as adopted by the EU in 2016 it says:, "from September 2016, member states will be required to authorise the 700 MHz-band by 2020, unless there are justified reasons for delaying it until mid-2022 at the latest", reports the *European 5G Observatory*. The Observatory also stated, in April 2020, that "exceptional circumstances caused by the Covid-19 epidemic have forced some countries in Europe to postpone 5G auctions scheduled in the first months of 2020. Four EU countries, Austria, France, Spain and Portugal have postponed spectrum auctions for 5G due to the Covid-19 epidemic so far."

The European Commission selected the consultancy firm, Idate-digiworld to carry out the *European 5G Observatory,* to monitor the rolling out of the 5 G Action Plan. IDATE-DIGIWORLD is a smart-looking consultancy company and self-declared "European think-tank for members, policy-makers and players of the digital transformation", with some of the largest telecom operators and producers as its clients.

One of those clients, isn't a Telecom giant, but a governmental regulator, Ofcom in the UK. *European 5G Observatory* reports that 'Ofcom opened a consultation on human exposure to Electromagnetic Field Emissions (EMF) in the UK. The consultation started on February 21th 2020 and ended on May 15th 2020: "The regulator proposes to include a specific condition in telecom licences requiring licensees to comply with ICNIRP guidelines. {…) At the same time, Ofcom released the results collected close to 16 5G base stations in 10 cities across the UK and to 60 GHz fixed wireless equipment in Liverpool. In all cases, the measured EMF levels from 5G base stations were far below the ICNIRP Guidelines (the highest level was approximately 1.5% of the relevant level); the 5G share of the total emissions level observed was currently very low."

To the question, "Is ICNIRP responsible?", Paolo Vecchia, former Chairman for ICNIRP (2004-2012) answered very clearly at a conference in September 2008 that "the ICNIRP guidelines are neither mandatory prescriptions for safety, the "last word" on the issue, nor are they defensive walls for Industry or others." This statement makes it clear that the decision to adopt these guidelines into national legislation as "sufficient to protect public health" is

political. The possible misuse by governments of ICNIRP and its guidelines seems to be another key question, that still needs looking into and answering.

On the other hand, ICNIRP presents itself as the provider of scientific truth. For example, in a report for the Irish government, under the heading, "Recommendations International Guidelines" it states that "there should be strict compliance with ICNIRP guidelines: The ICNIRP guidelines on exposure limits have been recommended by the European Commission to its Member States, and they provide science-based exposure limits that are applicable to both public and occupational exposure from RF and ELF fields. They also provide sound guidance on limiting exposure from mobile phones and masts, as well as for power-line fields. The ICNIRP guidelines provide adequate protection for the public from any EMF sources. While the guidelines were published in 1998, they are constantly under review and still have appropriately protective limits. The guidelines are based on a weight of evidence review from all peer-reviewed scientific literature and not on the conclusions of any single scientific paper."

Even as ICNIRP has been positioning itself during the last 25 years as the sole scientific truth when it comes to possible relation between EMF and adverse health effects, it would not be correct to hold this scientific NGO accountable if one day it would be undisputed that EMF causes health problems. National governments have their own responsibility to protect their citizens, just as the European Commission has, which after all is the 'Guardian of the Treaty' and therefore should also take the legally binding 'precautionary principle' into account.

The telecommunication industry applauds ICNIRP

In most policy fields, industry keeps reiterating that the limits scientific advisory committees propose are too strict. But in the case of the exposure limits for non-ionizing radiation the telecom industry seems very content with the norms ICNIRP proposes. In many reports over the past twenty years, the Telecoms lobby in Europe has always referred to the safety assurances published by ICNIRP.

In its Environmental Report of 2005, the European Telecommunications Networks Operators' Association (ETNO) wrote: "Concerning the European Union's legislative and policy framework on EMF, ETNO has been in direct contact with EU institutions. The association has provided a steady stream of facts and advice to legislative bodies in order for the EU to base its Directive concerning 'minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (electromagnetic fields)' on a sound scientific basis as provided by the International Commission on Non-Ionising Radiation Protection (ICNIRP)."

Thirteen years later, the Boston Consulting Group, in a report with the ominous title, 'A playbook for accelerating 5G in Europe', pleads for the harmonized limits ICNIRP (and also IRPA and the WHO EMF project) proposes, and criticizes governments that apply stricter limits. Exactly the same point was made by ETNO in a public consultation by the European Commission. ETNO was in favour of the "harmonised ICNIRP limits".

The same word, *harmonised*, comes back in a plea for "a harmonised EU approach to 5G security" that ETNO launched on 29 January 2020. "We therefore welcome today's publication of the "5G Security Toolbox", presented by EU Member States with ENISA and the European Commission. Europe's decision-making on 5G should continue being based on

facts, it should be proportionate to threats and build on a solid understanding of technology reality. In this context, we invite National Governments to avoid disproportionate actions that negatively impact the investment climate, and which could in turn harm both Europe's competitiveness and its strategic position in 5G development."

ETNO argues that rules and regulations should not hamper but support European investment and innovation, because "regulatory pressure still risks holding back European investment and innovation on many fronts"…"The speed of 5G rollout is significantly slowed by excessive spectrum prices and challenging license conditions."

ETNO continues to explain the policy-wish list: "The opportunity of fully unleashing fibre deployment awaits a pro-investment implementation of the European Electronic Communications Code. Regulatory asymmetries, especially in the field of data, still hold back European innovation. Market fragmentation still affects Europe's full potential in network investment. European institutions and national governments both have a major role to play in removing such barriers."

Yet again, ETNO does not lobby for lowering the ICNIRP standards, these are not seen as part of the "regulatory pressure" that hampers technological development. On the contrary: the norms ICNIRP proposes are the "harmonised limits" that ETNO welcomes.

All in all, the telecom-sector seems to be quite pleased with ICNIRP's positioning. This is deviating from the standard procedure in EU-policy making where a specific industry concerned will on essential aspects always try to influence laws and regulations in their favour through various ways of lobbying. Apparently in case of ICNIRP there is simply no need to do so.

The Telecom Lobby

In order to promote a continuation of favourable policy-making, European telecom companies have many lobby-meetings with the European Commission, and no doubt also at national political levels. According to the EU transparency Register, ETNO has a budget of over one million euros for lobbying and representing Europe's telecom companies. With at least seven registered lobbyists, ETNO had 70 registered lobby meetings with the European Commission (EC) in 2019. "ETNO's primary purpose is to develop top-level policy papers and support members in promoting a positive policy environment allowing the EU telecommunications sector to deliver best quality services to consumers and businesses. We also organize some of the main European events for discussing telecom and digital policy."

But of course, the individual telecom companies also have lobbying budgets and lobbyists representing them at the European institutions in Brussels. Ericsson had a lobby budget of 700.000 euros and five accredited lobbyist in 2019, Telefonica had a lobbying budget of 1,8 million euros and 6 lobbyists who covered no less than 83 meetings with the EC, Deutsche Telekom had a 1,5 million lobbying budget, with 5 lobbyists and a total of 110 lobby meetings with the EC.

In early December 2019, a large delegation of CEOs from ETNO met with Margrethe Vestager, Executive Vice-President of the European Commission responsible for "Europe fit for the Digital Age". The delegation included: Tim Hoettges from *Deutsche Telekom*,

Stephane Richard from *Orange*; Thomas Arnolder from *Telekom Austria*, Salvatore Rossi from *TIM*, Alexandre Fonseca from *Altice Portugal*, as well as the Chairman of ETNO, Steven Tas, the Director General of ETNO, Lise Fuhr, and senior representatives from *Telefonica* and *Telenor*.

At the end of January 2020, an important event was held, the European 5G conference. It welcomed more than 250 delegates, who discussed "the necessary next steps to ensure the success of 5G in Europe". Eric Van Rongen, at the time still ICNIRP-Chair, was among the speakers who provided "the audience with insightful views on their areas of expertise." The purpose, apparently, was not to discuss the sagacity and safety of rolling out 5G, but rather to ensure the success of 5G deployment.

It is important to note that the efforts of the telecom industry to influence regulatory agencies often take illegal forms. Telecommunications companies are high on the list of the companies that were penalised in the U.S. for corrupt practices. European companies like *Ericsson, Alstom* and *Telia* are in the top ten.

Also significant, is the fact that more and more world leading insurance companies are backtracking from insuring telecom companies concerning the risks around EMF. In March 2019, in its "SONAR Emerging risk insights" report, one of the world's largest insurance companies, *Swiss Reinsurance Company* (Swiss Re), classified "unforeseen consequences of electromagnetic fields" into the highest risk class, together with endocrine disrupting chemicals. "The ubiquity of electromagnetic fields (EMF) raises concerns about potential implications for human health, in particular with regard to the use of mobile phones, power lines or antennas for broadcasting. Over the last decade, the spread of wireless devices has accelerated enormously. The convergence of mobile phones with computer technology has led to the proliferation of new and emerging technologies. This development has increased exposure to electromagnetic fields, the health impacts of which remain unknown."

The lobby power of the telecom-industry in Brussels, the decision-making heart of the EU, is enormous. Yet the corporations involved do not have to lobby the guidelines and health advice related to their technology, because ICNIRP has been providing the "safety certification" for over 25 years. At the same time the insurance sector is not very assured and does not want to pay possible litigation costs once telecom companies would get sued, which is happening more and more frequently.

The call for more independent scientific assessment in this area

Almost ten years ago, in May 2011, the Council of Europe adopted a report from Mr Jean Huss on "The potential dangers of electromagnetic fields and their effect on the environment". It stated that the findings of scientific research on the possible risks of electromagnetic fields were inconclusive and contradictory. In the light of the correlation between origin of funding and the findings it called for "genuine independence on the part of the expert appraisal agencies and for independent, multidisciplinary and properly balanced expert input. There must no longer be situations where whistle blowers are discriminated against and renowned scientists with critical opinions are excluded when

experts are selected to sit on expert committees or no longer receive funding for their research."

In the meantime, not a lot seems to have changed. In a letter, published this year in *Bioelectromagnetics,* three researchers - Steven Weller , Victor Leach and Murray May - of the Australian "Oceania Radiofrequency Scientific Advisory Association" (ORSAA) write: "Half a century of scientific research into the safety of EMFs (from static to 300GHz) has not resulted in any substantial policy advice changes. The question that we believe needs to be asked is as follows: Is the continuing unchanged policy advice on EMFs occurring because those who are trying to advocate change have no voice in the process and because the process is dominated by groups with self-interests in maintaining the status quo?"

The three researchers point to the fact that radiofrequency electromagnetic radiation is "a booming multi-trillion-dollar industry globally, and changing current prescribed safety levels to more stringent standards would bring about unfavourable financial consequences and affect industrial and military functions. In some countries, such as Australia, the regulator, which has a health protection responsibility, also sells RF spectrum licenses, which represents a clear conflict of interest. The very same agencies with responsibility for providing safety advice to the public are also considered by some to have been captured by the industry."

The huge financial weight and power of the telecom companies is something the industry itself also stresses. In its report from January 2020, 'The State of Digital Communications 2020', ETNO boasts that "its Telecom members are thriving and business is booming: Telecom is Europe's major technology business, with a €136.9 billion per year value added and training on the rise. (…) Of the 17 Europe-based companies figured in the 2019 Forbes Digital 100 index, 11 are either telecoms operators or telecoms equipment vendors, and more than half of them are ETNO members."

Whether or not ICNIRP is 'captured by industry', a remarkable fact is that the organisation that appears to be the world's most important body responsible for advice on non-ionizing radiation is a private organisation, not a public authority.

"To me it seems wiser if the EU and national governments stop relying only on the advice of ICNIRP. A committee of its own is not an unnecessary luxury," Hans Kromhout writes. When we ask him if it would seem to him more logical that it be a public organisation giving advice on non-ionizing radiation, he answers: "I completely agree."

But this is not what is happening in the heart of the European Union. According to ICNIRP's website there is a contractual partnership between the European Commission, which is the Guardian of the Treaty, and thus also of the legally enshrined precautionary principle. It states: "The European Commission and ICNIRP collaboration over the years, relies on annual or specific contracts, such as the Concerted Action within FP5 - Life Quality, Key action Environment and Heath. ICNIRP also takes part, in consultation together with other stakeholders, on the development of directives and liaises, upon request, with different EC entities, such as the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR). Support to ICNIRP is provided by the European Commission through its Directorate General Health and Safety at Work as part of an EC grant agreement, as laid down in the ICNIRP reports."

Given the experience with ICNIRP of the past 25 years, the growing body of evidence that there are serious concerns on adverse effects of EMF on public health and the huge economic interests involved, it seems not very wise for the European Commission and national governments to base their policies solely on the ICNIRP guidelines and advice.

Chris Portier agrees by saying that “governments have no say in the governance or membership of ICNIRP. In addition, without their own review committees, governments do not have their own experts to advise them about these topics. I believe it would be best if such an entity was run by a trusted organization that has some form of government oversight.”

Portiers adds in writing to us: “I have been in the position of managing, running, chairing and/or being a member of dozens of national and international committees. These were always government committees or WHO-related entities. When run properly, governments can get excellent advice on issues. There is usually a place for interested parties (industry, concerned citizens) to express their opinions to these committee members at public forums. And there are legal consequences to providing false information on Conflict of Interest forms, etc. All of these reasons lead one to believe a government managed Commission would be better.”

We think that the call for more independent scientific assessment in this area is, for all the arguments mentioned in the above, fully justified.

IV - Conclusion

ICNIRP presents itself, and is described by the European Commission and in the media, as an independent international commission that gives advice based on scientific evidence. We believe that there are various reasons to question this (self)-image.

The composition of ICNIRP is very one sided. With only one medically qualified person (but not an expert in wireless radiation) out of a total of 14 scientists in the ICNIRP Commission and also a small minority of members with medical qualifications in the Scientific Expert Group, we can safely say that ICNIRP has been, and is still, dominated by physical scientists. This may not be the wisest composition when your remit is to offer advice on human health and safety to governments around the world.

As one can read in the 45 portraits of the members of the ICNIRP commission and of the Scientific Expert Group (SEG), they all share the same position on the safety issues: non-ionising radiation poses no health threats and the only effects it has are thermal. ICNIRP says "non-ionising radiation poses no health threats if it does not heat the tissue by more than 1 °C", by which it admits that there are possible health effects, but only if exposure levels to strong radiation are too high".

Over the past years, and on many platforms, various EMF-experts have stated that ICNIRP is wrong to continue dismissing certain scientific studies showing adverse health effects – like the American NTP-study - and is mistaken in its almost dogmatic conviction that "non-ionising radiation poses no health threats and the only possible health effects it has are thermal in case of strong radiation".

Even after much criticism from members of the global scientific community, ICNIRP still adheres to the paradigm that the only proven effects (on health) are thermal. "ICNIRP appears to take into account only the warming of tissue and uncontrolled muscle contractions, although they claim in the most recent advice, that they also evaluated other mechanisms", writes Dutch Professor Hans Kromhout, who is currently leading a long-term study (in the Netherlands) into the effects of mobile phone use on human health, and who is chairman of a special committee on Electromagnetic Fields of the leading Dutch Health Council, which advises the Dutch government.

It seems that "a closed circle of like-minded scientists" has turned ICNIRP into a self-indulgent science club, with a lack of bio-medical expertise, as well as a lack of scientific expertise in specific risk assessments. Thereby, creating a situation which might easily lead to "tunnel-vision" in the organisation's scope. Two leading experts, Hans Kromhout and Chris Portier, confirmed to us that ICNIRP is a closed, non-accountable and one-sided organisation.

As many scientists and critical observers have pointed out, it seems that ICNIRP members are either oblivious to, or are ignoring, scientific studies that find possible adverse health effects in the absence of heating. Even though some ICNIRP-members have themselves acknowledged that industry-funded scientific research tends to produce less findings showing adverse health effects of EMF, whereas publicly funded studies – like the NTP-study – do find significant links between EMF and adverse health effects, this does not seem to influence one iota the views of ICNIRP-members.

The majority of ICNIRP-scientists have done, or are doing, research partly funded by industry. Is this important? As we argue in the introduction, we believe it is. Scientific publications, co-authored by two ICNIRP-scientists – Anke Huss and Martin Röösli, confirm the importance of funding. In 2006 and 2009 they did a systematic review of the effects of the source of funding in experimental studies of mobile phone use on health, and their conclusion was that, "industry-sponsored studies were least likely to report results suggesting (adverse health) effects". And theirs is not the only study that showed this, as there have been numerous studies of the differences in reporting from industry-funded research versus publicly-funded research that suggest a strong funding bias on the results.

In addition to the fact that certain members of ICNIRP, are simultaneously members of the International Committee on Electromagnetic Safety (ICES) of the US-registered Institute of Electrical and Electronics Engineers (IEEE), we have seen further evidence of a close cooperation between ICNIRP and ICES, an organisation in which many people from the media and telecom industries, as well as from the military, are actively and structurally involved. During the current leadership of ICNIRP, these ties have become even closer "with the goal of setting internationally harmonized safety limits for exposure to electromagnetic fields". This must surely be considered as a situation in which conflicts of interest are a real possibility.

It is clear from ICES minutes that ICNIRP worked very closely with IEEE/ICES on the creation of the new RF safety guidelines that were published in March 2020. And this implies that large telecom-companies such as Motorola and others, as well as US military, had a direct influence on the ICNIRP guidelines, which are still the basis for EU-policies in this domain.

Although there is a lot of lobby-power by the telecom sector in the European Union (both in Brussels and in the member states), the European Telecommunications Networks Operators' Association (ETNO) does not lobby for lowering the ICNIRP standards, as these are not seen as part of the "regulatory pressure" that hampers technological development. On the contrary: the norms ICNIRP proposes are the "harmonised limits" that ETNO welcomes. All in all, the telecom-sector seems to be quite pleased with ICNIRP's positioning. This deviates from the standard procedure in EU-policy making, where a specific industry concerned will, on essential aspects, always try to influence laws and regulations in its favour through various lobbying strategies. Apparently, in the case of ICNIRP, there is simply no need to do so. At the same time, the insurance sector does not, at present, seem very reassured and does not want to be put in a situation of having to pay potential litigation costs, if and when telecom companies get sued, something that is happening more and more often.

Despite ICNIRP positioning itself, during the last 25 years, as the sole purveyor of scientific truth when it comes to possible relation between EMF and adverse health effects, it would not be right to hold this scientific NGO solely accountable if, one day, it were to become undisputed that EMF do cause health problems. National governments, as well as the European Commission, which is, after all, the 'Guardian of the Treaty', have a duty of care and protection of their citizens, and therefore should also take the legally binding 'precautionary principle' into account.

We think that the call for more independent scientific assessment in this area is, for all the arguments mentioned above and in what follows, fully justified.

That is the most important conclusion of this report: for really independent scientific advice we cannot rely on ICNIRP. The European Commission and national governments, from countries like Germany, should stop funding ICNIRP. It is high time that the European Commission creates a new, public and fully independent advisory council on non-ionizing radiation. The funds currently allocated to ICNIRP could be used to set up this new organisation. And given the overall rise in R&D funding via Horizon Europe, with a foreseen budget (for 2021-2027) of between 75 and 100 billion euros, funding should in no way constitute an insurmountable hurdle to setting up this new, truly independent, body.

V - PORTRAITS OF THE ICNIRP-MEMBERS

ICNIRP COMMISSION:

Gunde Ziegelberger (Scientific Secretary)

Biography

On ICNIRP's website we read that Gunde Ziegelberger holds a PhD in Biology and after a career at the Max-Planck-Institute, she joined the Federal Office for Radiation Protection (BfS) in 2002, where she works on "Non-Ionizing Radiation". Since 2004 she also worked as Scientific Secretary for ICNIRP- she replaced Rüdiger Matthes, who became a commission member - and in that function, she is also a member of the ICNIRP Board together with the Chair (see Croft) and Vice Chair (see Van Rongen). ICNIRP's website clarifies: "The three Board members represents ICNIRP externally and mostly in its relations with the international and national partners and the press. The Scientific Secretariat is in charge with some specific scientific projects mostly related to workshops and with all administrative and operational tasks."

Position

In February 2019 Dr Ziegelberger gave a short interview in which she stated that when the limit values are respected so far scientific findings show that human beings don't run any risk from electromagnetic radiation.

Ziegelberger functions as Scientific Secretary of ICNIRP, she co-authors many scientific publications with ICNIRP-members. In September 2016 for example Ziegelberger was co-author of a publication 'A Closer Look at the Thresholds of Thermal Damage: Workshop Report by an ICNIRP Task Group'. The article concludes the workshop – co-organsied by the WHO and financed by the European Commission, the Turkish Ministry of Health, the International Radiation Protection Association (IRPA), the German Federal Ministry for the Environment (BMUB), and the Finnish Radiation and Nuclear Safety Authority (STUK). The conclusion shows that the workshop "will provide valuable input into the revision of the guidelines being formulated by ICNIRP for limiting human exposure to RF fields." It was also clear that only thermal (adverse) effects were discussed as was the case in the new ICNIRP guidelines from 2020.

She co-authored as BfS -researcher a study within the ARIMMORA risk assessment which concluded that "the relationship between exposure to the agent ELF-MF and risk of childhood leukaemia is considered consistent with "IARC Group 2B" classification of possibly carcinogenic to humans (Fig. 1). This category is the result of limited evidence of carcinogenicity in humans and inadequate evidence of carcinogenicity in experimental animals."

Possible conflicts of interest.

Although Ziegelberger plays an important role for ICNIRP, given her position in the board and the fact that she works in an important department for radiation protection (BfS) of the German government, we could not find any DOI.

Rodney Croft (chair as of May 2020)

Biography

Rodney Croft is a psychology researcher. He works as professor of Health Psychology at the School of Psychology, University of Wollongong, Australia.

He joined the ICNIRP Biology Standing Committee in 2008 and the Main Commission in 2012, to become chair in May 2020.

ICNIRP's website states that his research focuses on the delineation of human brain function, as well as psychiatry more generally. He participates in a variety of national and international scientific and government committees, was Executive Director of the Australian Centre for Radiofrequency Bioeffects Research ((ACRBR 2004-2011) and is currently Director of the Australian Centre for Electromagnetic Bioeffects Research.

In June 2011, Rodney Croft as Executive Director of ACRBR announced that the organisation would cease operations because "it had been unable to secure further funding to continue its research activities". But many of the ACRBR Directors would be able to continue their Rf research but under the banner of the Bioelectromagnetics Research Group, part of the Brain and Psychological Sciences Research Centre (BPsyC) at the Swinburne University of Technology, which has for many years very close ties to Telstra, Australia's biggest Telecom company.

In August 2012 Croft received new funding when Australian Minister for Health, Tanya Plibersek, announced the establishment of a new $2.5 million NHMRC Centre of Excellence: the Australian Centre for Electromagnetic Bioeffects Research (ACEBR) to be based at the University of Wollongong and led by Professor Croft. One of the central university partners of the ACEBR research Swinburne University.

Position

Croft is a typical ICNIRP member and has been defending for years and from different positions the point of view that there are no dangers associated with the use of mobile phones. On the ABC Lateline program (April 4, 2009) Dr. Rodney Croft, then Director of ACRBR, stated: "There really has been a lot of research done to date and the research has very clearly shown that there aren't any effects. With children, I really don't think there is any evidence suggesting that this might be a problem. There isn't anything to suggest that we may have to be a little bit more cautious."

Much earlier in 2003 the Australasian College of Nutritional and Environmental Medicine (ACNEM) published a paper by Don Maisch "that detailed reasons why extra precautions

needed to be taken for children and cell phone use. The paper included a number of statements of concern specific to this issue from scientific and medical organizations internationally and concluded with the question: “Is it worth the risk” to continue to allow unrestricted cell phone use by children.”

In 2008 the Russian National Committee on Non-Ionising Radiation Protection (RNCNIRP) issued official advice that the “health of the present generation of children and future generations is under danger” from cell phone use and therefore the committee has recommended that cell phone use be restricted for people under 18 years of age.

Croft said in 2011: “With children, I really don’t think there is any evidence suggesting that this might be a problem. There isn’t anything to suggest that we may have to be a little bit more cautious” And to visually back up ACRBR’s on children and cell phone use on the ACRBR web site published “an animated image that included images of children happily using cell phones”.

In 2009 a scientific review paper with Van Rongen and Croft as first and second authors respectively stated. “Subjective symptoms over a wide range, including headaches and migraine, fatigue, and skin itches, have been attributed to various RF sources both at home and at work. However, in provocation studies a causal relation between EMF exposure and symptoms has not been demonstrated, suggesting that psychological factors such as the conscious expectation of effect may play an important role in this condition.” The article mentions that “all authors are either current or former members of the Standing Committee on Biology of the ICNIRP” but does not mention anything on funding of the study.

During an International Workshop on RF Measurements, Research Studies & Standards Development in 2018 Croft downplays scientific research that shows effect from EMF by saying that “Counterbalancing is necessary to enable appropriate interpretation of data” and “Conclusions must be based on the scientific literature, not just a data set”.

In 2019, Croft and a researcher (expert in antipsychotics) were awarded 1.2 Million$ for a project entitled "Exposures of mobile phone radiofrequency electromagnetic energy in juveniles: effects on brain development and behaviours." Neither of the two researchers are experts in the area of brain development, developmental psychology or juvenile behaviour.

Within ICNIRP, Rodney Croft was the chair of the Project Group that was tasked with preparing the new ICNIRP Guidelines, published early 2020. According to critics, ICNIRP still dismisses completely: the existence and significance of non-thermal effects, existence of the risk of cancer in long term avid users of mobile phones, IARC’s classification of RF as a possible human carcinogen (the IARC monograph review of science was not included in list of science reviews used by ICNIRP in preparation of the new guidelines).

Possible conflicts of interest

Just like his predecessor Van Rongen, Rodney Croft provides unpaid services to the IEEE ICES SC/4 Standards committees, a US version of ICNIRP, with a broad number of representatives from both military and telecom industry; ICES boasted that they had “at least two members of ICES as members of the new 13 person ICNIRP Project Group (PG) on HF guidelines (up to 300 GHz), of which the PG Chairman (Croft), is now very willing to work with ICES to develop

science based safety standards. This will enhance the possibility of harmonizing international RF safety standards."

Croft also advises the EMF reference group, and a community group managed by the Australian Government organization, ARPANSA. He receives funding from the Electric Power Research Institute EPRI for a project investigating RF effects on EEG and thermoregulation.

To possibly answer this question a brief examination of ACEBR's Science & Wireless 2013 seminar "Health & Future RF Technologies" is an indication. In the seminar acknowledgements, the following was stated: "The ACEBR gratefully acknowledges the financial support of the National Health & Medical Research Council of Australia and Telstra Corporation, which has enabled SW2013 to run".

In Rodney Croft's introduction to the presentation by Mr. Mike Wood from the Australian Mobile Telecommunications Association (AMTA) on "4G telecommunications technologies", he said the following, in part: "Clearly what we see here is a whole lot of new technologies which are going to come about. How do we know what's going to be most relevant to us? Well, in the short term I think that our industry representatives are going to give the best indicator of this"

Croft was appointed in 2014 an Associate Editor of the BEMS journal of the Bioelectromagnetics Society (BEMS); The annual meetings of BEMS are a heavily industry sponsored event. The annual meeting celebrating the Bioelectromagnetics Society (BEMS) and the European Bioelectromagnetics Association (EBEA) was in 2015 in California (USA), had sponsors including companies such as, the Electric Power Research Institute (EPRI), Mobile Manufacturers Forum (MMF), Korean Institute of Electromagnetic Engineering Society (Mobile EMF Consortium) and, GSM-ATM5.

Croft also held talks and expert opinion on behalf of industry and for the Mobile Manufacturers Forum, a consortium of the world's major cell-phone companies. At a 5G Conference in Dubai In December 20, 2019, Croft held a lecture on behalf of ICNIRP alongside ICES Chairman Jafar Keshvari and TC95 Chairman C-K. Chou.

He joined the conference organized by the Telecommunication Regulatory Authority (TRA) of the United Arab Emirates held on December 8-9, 2019 in Dubai. Three presentations were on RF exposure safety limits: "5G RF safety concerns: New IEEE StdC95.1TM-2019" by C-K. Chou; "Scientific Basis of 5G Exposure Limits IEEE C95.1:2019 Standard" by Jafar Keshvari, and "Ensuring 5G Safety with the New ICNIRP Guidelines (100 kHz to 300 GHz)" by Rodney Croft of ICNIRP.

Croft has also actively collaborated in research with Ray McKenzie, who is a manager at the Mobile Carriers Forum (MCF) which is a special division of the Australian Mobile Telecommunications Association (AMTA) dealing with the policy, regulatory, public communications and health and safety aspects of the deployment of mobile networks in Australia.

On his website Croft's disclosure statement says: Rodney Croft has consulted to a range of organisations such as Shelharbour City Council, Department of Defence, Comcare and Optus. According to his ICNIRP declaration of interests he received personal remuneration for

providing data analysis services to Heptares Therapeutics Ltd, a pharmaceutical company. And Croft received personal remuneration for providing advice to Australian Bureau of Statistics (ABS) on effects of RF devices used by field staff on field staff, resulting from a contract between University of Wollongong and ABS. He also received personal remuneration for "providing advice to Victorian Government on conducting bioelectromagnetics research, resulting from a contract between University of Wollongong and Victorian Government".

As explained before in this report the Australian government receives billions from issuing spectrum licences to Telecom operators. In Australia, this licensing is carried out by industry regulator ACMA, the Australian Media Communications Authority. ACMA also collects a separate levy or tax from the wireless industry, money that is earmarked for scientific research on RF-EMR. ACMA then diverts $300,000 to the other government institution ARPANSA (Australian Radiation Protection & Nuclear Safety Agency) for its public information campaign) and $700,000 to the National Health & Medical Research Council (NHMRC).

According to the Australian research group ORSAA "the money that the Australian NHMRC receives in order to provide grants for medical research has mostly gone to industry-friendly researchers who have direct links with the wireless industry. For example, the largest recipient of this NHMRC research funds is Prof. Rodney Croft. He has essentially been the head of RF-EMR health research in Australia, despite his questionable qualifications for this health research role. Prof. Croft has received ample direct industry funding in addition to his lucrative NHMRC grants, which should be termed indirect industry funding." Croft was the only Australian who played a part in determining what NHMRC research on EMR and health should be funded.

He used his international contacts at the WHO to get more Australian funding. This is how it worked behind the scenes: Croft was invited from Australia to the WHO for an expert consultation to determine which areas of medical research was needed; The Australian NHMRC research on EMR then looked to the WHO guidelines (co-influenced by Croft and ICNIRP or hi-jacked as some critics say) in order to decide their funding priorities (the 2010 WHO RF Research Agenda is the basis of funding for NHMRC research grants). Croft's laboratory then received the funding and has continued to get most of the research money over many years.

Croft had good relations with an influential industry man, Dr K. Joyner. Which researchers or research groups have been granted the NHMRC funds has been influenced to a large extent by Joyner, who was Motorola's Director of "Global EME Strategy and Regulatory Affairs" and also represented the Australian Mobile Telecommunications Association, an industry group, on the telecommunications standards committee and the Mobile Manufacturers Forum ; Notwithstanding these ties Joyner was a longstanding member of the Standards Australia TE/7 Committee: Human Exposure to Electromagnetic Fields, and later on he was on the ARPANSA committee that set the current Australian Radiofrequency/Microwave human exposure standard. He was regarded by the cell phone companies as Australia's foremost authority on the industry's position on health issues with EMR and has represented Motorola and the Australian cell phone industry on several international standards-setting

bodies. Joyner also had connections with Burson Marsteller, the PR firm representing the cell phone industry in Australia.

In October 2003 Ken Joyner, the key Motorola representative gave a presentation at the Annual Conference of the Australian Radiation Protection Society called: “A Review of RF Bioeffects Studies Relevant to the Use of Mobile Phones by Children”. Don Maisch writes in an article Motorola’s Micky Mouse Review: “The Motorola review’s conclusions as to a lack of scientific evidence of possible harm to children using mobile phones ignores a large body of expert opinion calling for a precautionary approach when it comes to children and mobile phone use.”

As reported in Microwave News (1999) in Europe there was some discontent with scientists with Motorola’s involvement with the EC research and telling European scientists how to spend research funds. As Don Maisch writes in ‘Corporate ties that bind: An Examination of Corporate Manipulation and Vested Interest in Public Health’ (2017): “In January 2009, Dr. Joyner announced that he was leaving his Director position at Motorola after 12 years and was "looking for new opportunities to work in the telecommunications industry". In that same year, Dr. Joyner was listed on the NHMRC's Peer Review Honour Roll which acknowledged its many peer reviewers and external assessors who had exhibited "excellent track records and wide-ranging expertise in Australian and international health and medical research fields". However, under the section "Administering Institution/Employer" he was listed as simply "consultant" and nothing about possible conflicts of interests. He later was appointed as the sole non-radiation expert on the 14-member Victorian government's Health department's Radiation advisory committee.

ORSAA calls this “pure corruption at a huge cost to public health everywhere. This system of funding and promoting an in-club of industry friendly researchers has kept a small number of people in powerful positions within the WHO, ICNIRP, ARPANSA etc., influencing decision making for most of the world.”

Eric van Rongen (Vice Chair ICNIRP-commission, until May 2020 chair)

Biography

Eric Van Rongen is a biologist. He is a staff member of the Dutch Health Council since 1992, where he focuses on non-ionizing radiation.

Van Rongen is a member of ICNIRP since May 2001. In 2016, he became the chair of the ICNIRP-commission. Since the beginning May 2020 he is no longer chair but vice-chair.

He also a member of the International Advisory Committee WHO EMF Project since 1995.

Van Rongen did not publish original research studies on EMF himself, only opinions or review articles.

Position

Van Rongen systematically, in scientific publications and in press articles, defends for more than twenty years the point of view that there are no dangers associated with the use of mobile phones. According to him, even for children there are no reasons to apply the precautionary principle. In 2004 for example he published an article in which he stated: 'The Health Council therefore sees no reason to recommend limiting the use of mobile phones by children.'

He systematically criticizes all studies that seem to show that non-ionizing radiation poses a problem. Recently the National Toxicologic Program (NTP) study on Cell Phone Radio Frequency concluded that there was clear evidence of tumors in the hearts of male rats But in an ICNIRP-publication Van Rongen and others stated that 'substantial limitations (of the NTP-study) preclude conclusions being drawn concerning RF EMFs and carcinogenesis.'

Possible conflicts of interest

The WHO EMF project was severely criticized in 2007 for being for a large part financed by the telecom industry, for example by the Mobile Manufacturers Forum (now Mobile & Wireless Forum), a lobby organisation of the industry.

Since 2000 Van Rongen is a member of the International Committee on Electromagnetic Safety (ICES) of the IEEE. This committee is dominated by people from industry and military. The ICES chairman Jafar Keshvari works at Intel, the chairman of one of the main committees C.K Chou at Motorola. ICES clearly is an industry lobby and standard setting organisation. Maybe Van Rongen decided for that reason to become a 'non active member' according to his declaration of personal interests 2019.

In previous years there was some competition between ICNIRP and ICES/ IEEE – at the time when the chair of ICES was still Dr. Ralf Bodemann, topshot of Siemens and Dr. B Jon Klauenberg from US Air Force Research Laboratory was the chair of ICES working group TC95. (Klauenberg was the US counterpart of former ICNIRP-chair Repacholi to lead the very start of the WHO EMF in the 90'ies.) According to an annual report of ICES it was thanks to the arrival in 2016 of Van Rongen as chair of ICNIRP that the relations with ICES improved significantly, as they were not so cordial before: "In May 2016, there was a change of leadership and some members of ICNIRP. The new ICNIRP Chairman and one of the new members of the 14-member committee are also ICES members and ICNIRP is now willing to discuss harmonization of the exposure limits found in IEEE Stds C95.1 TM -2005 and C95.6 TM -2002 and the ICNIRP Guidelines."

The ICES annual report further mentions that thanks to the invitation to do so by Van Rongen, ICES has been able to comment on the proposed new guidelines by ICNIRP. ICES workgroup TC95 formed a 19-member task group to draft a document to comment on the ICNIRP proposed guidelines on time. "ICES will maintain its collaborative relationship with ICNIRP with the goal of setting internationally harmonized safety limits for exposure to electromagnetic fields at frequencies below 300 GHz. This interaction with ICNIRP is considered a major step forward."

A year later during the annual meeting of ICES in 2017 it was stated that “ICNIRP has delayed finalizing their conclusions to give full consideration of ICES’s recommendations”. And Van Rongen gave a presentation saying that there is “No evidence that HF-EMF causes such diseases as cancer, no evidence that HF-EMF impairs health beyond effects that are due to established mechanisms of interaction.”

Scientist Dariusz Leszczynski was a member of TC95, but resigned. He explained why on his blog: “My problem was that the membership of the IEEE-ICES-TC95 consists predominantly of the industrial scientists and the committee is chaired by C.K. Chou since the time he was employed by the Motorola. This means that all safety standards being developed by IEEE-ICES-TC95 are, in practice, developed by the industry scientists for the use by the industry they are employed by.” According to Leszczynski this is a clear conflict of interests.

The latest minutes of TC95 that ICES published on its website (August 2019) show that the committee is still dominated by industry scientists.

In October 2019 Van Rongen spoke at the GSMA Europe EMF Forum. The GSM Association is a lobby organisation that defends the interests of mobile operators worldwide. In 2018, he also was a guest at the Forum. Then he defended ideas that GSMA received with pleasure: "The ICNIRP limits provide a high level of protection for all people against known adverse health effects. Dr van Rongen explained that there is no scientifically substantiated evidence that radio signals cause diseases such as cancer and that ICNIRP had considered studies such as that of the American National Toxicology Program."

In November 2019 Van Rongen presented the “ICNIRP RF guidelines revision” at 23rd GLORE (Global Coordination of Research and Health Policy on RF Electromagnetic Fields) conference held on 4th – 6rth of November in Lima, Peru. GLORE is an initiative to coordinate research and policy initiated by Japan and Korea in 1997 and joined by Europe and then by USA, Australia and Canada. Main speakers were also his ICES-colleagues Jafar Keshvari and TC95 Chairman C-K. Chou.

Van Rongen recently assured the Dutch press that there are no conflicts of interest inside ICNIRP right now. He stated: 'In the past certain members maybe received co-funding from the private sector, but currently no member has ties with the telecom sector.'

Of course, it depends on what you consider as a 'tie with industry', but his own involvement in ICES is already shows that it is not true that 'currently no member had ties with the telecom sector'. He also published articles together with researchers who did receive industry funding, for example with Bernard Veyret, who is 'a member of the Scientific Council of the French mobile operator Bouygues Telecom. His laboratory has received research funds from the same operator.' This information can be found in the footnotes of this article.

Tania Cestari

Biography

Tania Cestari received her medical degree from the University of Rio Grande do Sul and completed her medical Residency in Dermatology in Porto Alegre, Brazil and since 1995 she works as Professor of Dermatology at the same university, where she studies predominantly on clinical aspects and skin response. Dr Cestari has authored 112 scientific peer-reviewed publications, 42 book chapters and joined the ICNIRP Commission in May 2020.

Position

Dr Cestari has been doing mainly research into skin allergies and dermatological problems; We could not find any publication linked to EMF.

Possible conflicts of interest

In her 'Declaration of Interests' it is mentioned that she received research grants via the Medical Foundation of her hospital from Pfizer, Abbvie Pharmaceutical and Vichy Laboratoires for drug research.

Nigel Cridland

Biography

Nigel Cridland is Senior Group Leader at Public Health England. He joined what was to become the Public Health England (PHE) already in 1990, where he specialised in non-ionising radiation. He was member of the project team that wrote the European Commission guide to implementation of the Artificial Optical Radiation Directive (2006) and leader of the project team that developed the guide to implementation of the EMF Directive (2013).

He was Scientific Co-ordinator Mobile Telecommunications and Health Research (MTHR) Programme 2001 - 2012. Cridland was a member of the Independent Expert Group on Mobile Phones (2000). On LinkedIn he states that he was also member of the management committee of the European COST 281 action Potential Health Implications from Mobile Communications Systems.

Position

The 2000-report of the Independent Expert Group on Mobile Phones stated that 'the balance of evidence to date suggests that exposures to RF radiation below NRPB and ICNIRP guidelines do not cause adverse health effects to the general population'. But at the same time, it said: "the gaps in knowledge are sufficient to justify a precautionary approach".

The MTHR-programme (2001-2012) of which he was the Scientific Co-ordinator concluded that no association between cancer and mobile phone use was found. We can now be, said

professor David Coggon, the chairman of the MTHR-programme, ‘be much more confident about the safety of modern telecommunications systems.’ Curiously enough the authors stated that: ‘We see no need for need for further research in any of the areas addressed by the research that is summarised in this report.’

Possible conflicts of interest

The MTHR-programme was funded by government and industry together, both for half of it. The final report states that to ensure that any of the funding organisation could not influence the outcome of the Programme an independent Programme Management Committee was set up. But there can be doubts about the independence of its members. From 2001 until 2007 Mike Repacholi (ICNIRP-founder, see the chapter on the history of ICNIRP) was for example member of the committee.

Guglielmo d'Inzeo

Biography

On ICNIRP’s website it reads that Guglielmo d'Inzeo is a Professor of "Bioelectromagnetic Interaction" at "La Sapienza" University of Rome since 1990. He researched active and passive microwave component design and bioelectromagnetism, mainly the interaction of electromagnetic fields with biological tissues, the effects of microwaves and ELF fields on biological samples and humans. He is author or co-author of more than seventy papers in international refereed journals and books.

He became a member of the European Bioelectromagnetics Association EBEA in 1989, and then President from 1993 to 1998. From 1992 to 2000 he was an Italian representative for the COST 244 and 244Bis projects on "Biomedical Effects of Electromagnetic Fields". From 1998 to 2004 he chaired the Italian ICEmB (Inter-University Centre Electromagnetic Fields and Biosystems). From 2001 to 2006 he was an Italian National representative in COST 281 project “Potential Health Effects from Emerging Wireless Communication Systems” and from 2007 in COST BM0704 related project.

Position

He has been active in the IEEE since the 80’ies, served as secretary–treasurer of ‘the IEEE Middle and South chapters’ and was from 2004 to 2009, also a member of the Technical Committee 95 (TC95) of IEEE International Committee on Electromagnetic Safety (ICES), of which Eric Van Rongen and Rodney Croft are also members. He published in the past 20 years a number of studies in IEEE Transactions on Biomedical Engineering and other IEEE publications, in which several times ICNIRP-founder Mike Repacholi was heartily thanked for his help.

In 2005 he was responsible for the Italian chapter of the report “European Information System on Electromagnetic Fields Exposure and Health Impacts” published on behalf of DG SANCO (European Commission), which was coordinated by the Joint research Centre (JRC of the EU); Alongside this project the “JRC developed during 2003-2004 the EIS-EMF project on behalf of DG SANCO with the overall objective of promoting cooperation among policy makers on public health and EMF risk communication issues in the EU”. What these projects basically reflect is the idea that concerns about possible health effect occur because people

do not understand the issue well enough and that the concerns can be taken away by better communication.

Possible conflicts of interest

As we stated before (see Van Rongen and Croft), ICES is dominated by people from industry and military.

His declaration of personal interest 2019 is signed but only partly completed. d'Inzeo did some paid consultancy for an Italian legal office called Trifirò & Partners and for a Environmental Measurement Report Managers & Partners - Actuarial Services S.p.A in Rome. His DOI from 2016 mentions that he has been doing work for the "Marconi Foundation". The Guglielmo Marconi Foundation states to "promote research in the field of telecommunications and carries out activities devoted to the knowledge and diffusion of Guglielmo Marconi's scientific activity". The Marconi Foundation further states that "professional training and teaching play a major role" in its activities and that "their research focuses on two major fields: 1) mobile and personal communication systems, with a special focus on radio access and propagation; and 2) the computer-assisted design of non-linear microwave devices".

What is not declared in his DOI is that d'Inzeo, is a director of the scientific committee of Elettra 2000, a consortium of Marconi and other foundations. The self-declared aim of Elettra 2000 is to "spread knowledge of Bioelectromagnetics and start a dialogue between science, politics, industry and citizens, involving young people and schools." And "Elettra 2000 promotes researches and studies related to specific areas of interest. In particular, the consortium co-finances a number of national and international projects devoted to the study of the effects of electromagnetic fields on human health, in order to provide an authoritative scientific answer, fair and independent to the problem."

Elettra 2000 provides "advice to enterprises" and "owns a modern fleet of instruments for measuring electromagnetic fields in both low and high frequency" which "are available to both institutional and private entities in order to promote the improvement of standards of protection and safety of people and environment."

This paper from 2008 (The Italian national electromagnetic field monitoring network) is an example of the kind of research projects that is financing. The conclusions reads: "The monitoring campaign, combined with the travelling communication campaign contributed to create a different and more constructive approach to the problem by the citizens. This is demonstrated by the analysis of the data press that shows criticality and greater negative involvement in those areas where the monitoring campaign has been less efficient or less intense".

Furthermore, in 2019 an Italian journalist of Investigative Europe wrote the following in *Il Fatto*: "He has done multiplied scientific opinions for companies such as Vodafone, participated in European projects - all funded by industry, such as Interphone, Cosmos, Cefalo, and since the late 90s participates in the Efhran portal, where among the financiers are Deutsche Telecom and the European Association of GSM producers."

Akimasa Hirata

Biography

Akimasa Hirata is professor of Electrical and Electronic Engineering at the Nagoya Institute of Technology and Director of Center of Biomedical Physics and Information Technology.

He also is an Administrative Committee Member and Subcommittee Chairperson (SC6 EMF Dosimetry Modelling) in IEEE International Committee on Electromagnetic Safety (ICES). The latest committee (also called TC95) is the one of which Eric Van Rongen and Rodney Croft were also members.

Position

In November 2019 TC95 once again came to conclusion that the IEEE standards are safe. The authors, among which Hirata, wrote:

"a) The weight-of-evidence provides no credible indication of adverse effects caused by chronic exposures below levels specified in IEEE Std C95.1TM-2019.

b) No biophysical mechanisms have been scientifically validated that would link chronic exposures below levels specified in IEEE Std C95.1TM-2019 to adverse health effects."

Possible conflicts of interest

As we stated before (see Van Rongen and Croft), ICES is dominated by people from industry and military.

Hirata conducted research published in *IEEE Transaction* in 2010 partly funded by KDDI Foundation. KDDI Corporation is a Japanese telecommunications operator.

But according to a recent publication Hirata himself judges that he has no conflicting interests.

Anke Huss

Biography

ICNIRP's website states that Anke Huss is an assistant professor at the Institute for Risk Assessment Sciences (IRAS) at Utrecht University, the Netherlands. "Her research focuses on environmental and occupational exposure assessment to environmental factors including electromagnetic fields and their health".

Huss is also involved in the GERoNiMO project, cancer and neurodegenerative diseases such as Parkinson's disease, Alzheimer's or ALS in the NOCCA (Nordic Occupational Cancer Study) and SNC (Swiss National Cohort) studies and on electromagnetic hypersensitivity. She is a

member of the Dutch Health council, and the Scientific Council for Electromagnetic fields of the Swedish Radiation Safety Authority (SSM).

Position

She is one of the rare members of ICNIRP who seems to be aware of an industry-bias; In the book "Overpowered: The Dangers of Electromagnetic Radiation (EMF) and What You Can do about it" by Martin Blank, Anke Huss is quoted on Industry bias in research to the possible health risks of EMF.

In a scientific paper Huss writes that 82% of the research funded by public agencies or governments and 71% of the research jointly funded by industry and public sources, report health effects from RF exposure. When the research is solely funded by industry only 33% finds such a link.

Later Huss published another study in which she and colleagues examined whether the source of funding of 59 studies of the effects of low-level RF radiation has an effect on the results of studies. "Of these 59 studies, 12 (20%) were funded exclusively by the telecommunications industry, 11 (19%) were funded by public agencies or charities, 14 (24%) had mixed funding (including industry), and in 22 (37%) the source of funding was not reported." Huss et all conclude that "there is widespread concern regarding the possible health effects associated with the use of cellular phones, mobile telephone base stations, or broadcasting transmitters. Most (68%) of the studies assessed here reported biologic effects. At present, it is unclear whether these biologic effects translate into relevant health hazards. Reports from national and international bodies have recently concluded that further research efforts are needed, and dedicated research programs have been set up in the United States, Germany, Denmark, Hungary, Switzerland, and Japan. Our study indicates that the interpretation of the results from existing and future studies of the health effects of radiofrequency radiation should take sponsorship into account."

In 2010, she published a follow up study which confirmed the previous findings: "Of 75 additional studies 12% were industry-funded, 44% had public and 19% mixed funding; funding was unclear in 25%. Previous findings were confirmed: industry-sponsored studies were least likely to report results suggesting effects.

She also published in 2018 a meta-analysis based on among others epidemiologic studies "to examine associations of occupational exposure to extremely-low frequency magnetic fields (ELF-MF)" with amyotrophic lateral sclerosis (ALS).

Possible conflicts of Interests

Her DOI says she gets funding from US based EPRI for a study called TransExpo on leukaemia in children. Ironically, she states that the contract does not mention complete independence from the funder, but she explains clearly why the data will be analysed independently and "that there is no way that the funders can have an influence on what we report to them."

Ken Karipidis

Biography

Ken Karipidis has been working as a scientist at the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) since 2000. He is, states ICNIRP, 'currently the assistant director of the Assessment and Advice Section at ARPANSA where he is heavily involved in the scientific and regulatory aspects of radiation protection from electromagnetic radiation sources.'

He is member of the Scientific Expert Group since August 2015. In May 2020, he became member of the ICNIRP Commission.

Position

In 2017 Karipidis published an article with the conclusion that the exposure to radiofrequency radiation due to Wi-Fi in schools was very low. In a letter to the editor three scientists criticized the study as 'of little practical use' and 'misleading'.

Karapidis and Rodney Croft were part of a subcommittee established by ARPANSA to look at EHS and the research in 2016/17. According to an ORSAA member present in these meetings both Karipidis and Croft ignored clinical/medical evidence "in favour of poorly conducted provocation studies performed by psychologists, some of whom were funded by industry".

At the end of 2018 Karipidis together with among others Rodney Croft published a study that claimed to proof that in Australia there has been no increase in any brain tumour that can be attributed to mobile phones. That study received a lot of criticism because it excluded the group of people above sixty, which is the largest segment of the population with brain tumours.

In August 2019 Karipidis advised 40,000 Australian doctors or general practitioners via an article on the website of Royal Australian College of General Practitioners (RACGP) in which he wanted "GPs and their patients to know there is no evidence to support the concern that 5G technology, which uses radio waves and emits low-level radiofrequency (RF) electromagnetic energy (EME), will cause harms to the public". He stated: "There's been a lot of research into whether radio waves cause adverse health effects, and the only established health effects of radio waves are very high-power levels, where they raise temperature. An everyday example of that is your microwave oven at home; inside the microwave is very powerful radio waves which make the water molecules in the food bounce very fast, heating them up."

Possible conflicts of interest

In the introductory chapter, we wrote about the financial relationship between ARPANSA and the telecom industry. ARPANSA every year has a meeting with the Australian Telecommunications Association (AMTA), a lobby-organisation of the telecom industry. Minutes of this meeting made public after a Freedom of Information Request show that the funding of research was also on the agenda. 'Industry remains supportive of continued funding,' it says.

Carmela Marino

Biography

Carmela Marino studied Biological sciences in Faculty of Sciences of "La Sapienza" University of Rome. According to ICNIRP she is currently Head of the Unit of Radiation Biology and Human Health, at Casaccia Research Center of Italian Agency for New Technologies, Energy and Sustainable Economic Development (ENEA).

On behalf of ENEA she coordinated the research activity Subprogram 3 *Interaction between sources and biosystems* (MURST/ENEA-CNR Italian National Program *"Human and Environmental Protection from Electromagnetic Emissions")* and was involved in *several projects of the 5° and 6°FP, as member of steering Committee and Coordinator of research unit.*

Position

On the one hand Marino seems to agree with the official ICNIRP position; On the other hand In May 2012, during ICNIRP's 7th International NIR Workshop in Edinburgh, Marino held a lecture on the advantages, challenges and limits of experimental studies, in which she said that there is a "large number of studies but with controversial results and also a limited number of studies in relation to particular endpoints." Marino asked her fellow ICNIRP members the rhetorical question, whether these studies "really able to give conclusive information?" ICNIRP's answer to that question was and is no.

Possible conflicts of interest

Her Declaration of Personal Interests does not mention anything. Notaby, not that since April 2020 her university holds a patent based on her research, not mentioned in her DOI 2019, although the worldwide application for this patent was filed years ago.

Sharon Miller

Biography

Sharon Miller works at the Food and Drug Administration (FDA) as optical engineer since 1981. According to ICNIRP she served on numerous committees of the International Commission on Illumination (CIE) and the International Organisation for Standardization (ISO).

Position

Miller publications are mainly in the field of ultraviolet radiation and optical issues. It is difficult to find scientific publications or public statements in which she says anything about the safety of non-ionizing radiation.

Possible conflicts of interest

In her Declaration of Personal Interest Miller does not state any possible conflict of interest and we did not find any.

Gunnhild Oftedal

Biography

Gunnhild Oftedal is associate professor at the Norwegian University of Science and Technology (NTNU). According to ICNIRP she is currently, working as Research Co-ordinator at the Faculty of Information Technology and Electrical Engineering, NTNU. "From the early 1990s, she has been involved in research on health effects of EMF in the ELF and the RF ranges, mainly with experimental human studies and observational studies.

She is member of international organisations in the field of non-ionising radiation and participates in the work of WHO (Environmental Health Criteria project) on the health risk assessment on RF fields."

She was one of the experts on a government-commissioned study, published in 2012, of possible health risks with radiation from mobile phones, base stations and wireless networks in Norway.

Position

In 2004 she answered on the questions if electromagnetic radiation from mobile phones may well affect us in other ways, too "that scientists are skating on thin ice when discussing these issues. They know little about the cause-and-effect mechanisms involved, and hence cannot eliminate the possibility that the effect of electromagnetic fields, however weak in mobile phones, may cause health problems".

But she sticks with the official ICNIRP position and in a study for the Norwegian government she suggests that this approach is the right one: "Only effects for which there was reliable scientific evidence were used (by ICNIRP) as the basis for the exposure restrictions."

In another recent study she concludes that "overall, the evidence points towards no effect of exposure. If physical effects exist, previous findings suggest that they must be very weak or affect only few individuals with IEI-EMF. Given the evidence that the nocebo effect or medical/mental disorders may explain the symptoms in many individuals with IEI-EMF, additional research is required to identify the various factors that may be important for developing IEI-EMF and for provoking the symptoms."

As writes Leszczynski the 'nocebo' hypothesis argues that people first become aware, e.g. from news and social media, of the possible health risks of EMF-emitting devices and then worries about the possible health risk lead to develop symptoms, which they attribute to EMF exposures.

Oftedal denies in an article by IE that the health debate is polarised: "In our field it is easy to put people in two camps, but the landscape is much more nuanced". Also, the closed culture

at ICNIRP is being denied: “People who demonstrate that they are skilled are asked to contribute.”

Possible conflicts of interest

In the study on “Mobile phone headache: a double blind, sham-controlled provocation study” co-financed by The Research Council of Norway, Norwegian Post and Telecommunication Authority, Statnett, Telenor, Norsk tele- og informasjonsbrukerforening (NORTIB), Netcom. The study found no effects.

She is member of Bioelectromagnetics society (BEMS) according to the DOI and also of the European Bioelectromagnetics Association (EBEA)

Tsutomu Okuno

Biography

Tsutomu Okuno worked for the National Institute of Occupational Safety and Health, Japan from 1980 to 2015.

He became a member of the Scientific Expert Group in 2013 and is a member of the ICNIRP Commission since 2016.

Position

Okuno was one of the authors of the ICNIRP note that criticized the NTP-study that showed carcinogenicity in rats. For the rest, his work seems mainly to be on ultraviolet radiation, not on radiofrequency radiation.

Possible conflicts of interest

In his Declaration of Personal Interest there do not seem to be sources of possible conflicts of interest and we did not find information that contradicts this.

Martin Röösli

Martin Röösli is Professor for environmental epidemiology at the Swiss Tropical- and Public Health Institute in Basel and leads the Environmental Exposures and Health Unit. His background is situated in atmospheric physics and environmental epidemiology.

In the field of non-ionizing radiation Röösli did several exposure assessments and epidemiological studies on the health effects of electromagnetic fields “including population based studies dealing with cancer, neurodegenerative diseases and non-specific symptoms of ill health”.

He is the chair of BERENIS, a Swiss expert group advising the government on electromagnetic fields and non-ionising radiation. He is a member of the advisory group of Cohort Study of Mobile Phone Use and Health (COSMOS) and between 2015 and 2018 of the the Scientific

Council of the IARC, specifically SC52. Since 2013 he is also a Member of the Editorial Board of Bioelectromagnetics.

He is still a member of the Expert Group for the Swedish Radiation Safety Authority (SSM), for which he gets 3000 Swiss francs yearly.

Relevant to this report Röösli was part of the Working Group of the IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 102: Non-Ionizing Radiation, Part II: Radiofrequency Electromagnetic Fields.

Position

Röösli has contributed to a study (see portrait of Anke Huss) which show that the funding of scientific research into EMF can influence the findings. Nevertheless, he confirms the general position of ICNIRP that no adverse health effects are proven.

In a study from 2010 ("Systematic review on the health effects of exposure to radiofrequency electromagnetic fields from mobile phone base stations") Röösli concludes: "Our review does not indicate an association between any health outcome and radiofrequency electromagnetic field exposure from MPBSs at levels typically encountered in people's everyday environment."

In a recent 5G report for the Swiss government Röösli et all conclude that "No health effect has been consistently proven," which he repeated in an interview.

In an annual report prepared for the Swedish Radiation Safety Authority (April 2020) by a nine-member panel of experts of which, ICNIRP vice-chair Eric Van Rongen and Röösli, which according to *MicroWave News* is published each year "as an annual update with the past year's most important scientific developments on the health effects of EMFs and RF radiation" states very bluntly that "no new established causal relationships between EMF exposure and health risks have been identified." The annual report simply does not mention the NTP report. "The two ICNIRP members and their seven colleagues made believe that the NTP report does not exist. It's not mentioned, there is no citation. Nothing at all. For the record, the NTP final report was released on November 1, 2018."

Louis Slesin of *MicroWave News* wrote: "There is a discussion of the NTP findings in last year's Swedish update. But that was based on an earlier NTP draft where the staff had opted for a weaker designation, "some evidence" of cancer. Later, after an in-depth public peer review, the NTP strengthened the conclusion to "clear evidence" of cancer. That was the headline news of 2018. "Clear evidence" was a game changer; leaving it out of the annual update is a sure sign of bias. The NTP conclusion was now qualitatively different from the earlier draft —it could well have been the title of the panel's 2018 update. But van Rongen, Röösli and the others ignored it."

On January 7, 2020 prof. Lennart Hardell and supported by 22 scientists researching EMF wrote a remarkably critical, open letter to Mrs. Simonetta Sommaruga, President of the Swiss Confederation, in which they conclude: "It is imperative that the chair and other experts evaluating scientific evidence and assessing health risks from RF radiation do not have such clear conflicts of interests or bias as Martin Röösli has. Indeed, being a member of ICNIRP and being funded by industry directly or through an industry funded foundation, constitutes clear conflicts of interest. Furthermore, it is recommended that the

interpretation of results from studies of health effects of radiofrequency radiation should take sponsorship from telecom industry into account."

The group of scientists also point out to a strange contradiction in the positioning of Röösli: "Surprisingly the IARC classification from 2011 of RF-EMF exposure as class 2B, 'possibly' carcinogenic to humans, was ignored in the background material to the new ICNIRP draft on guidelines. Remarkably one of the ICNIRP commission members, Martin Röösli, was also one of the IARC experts evaluating the scientific RF carcinogenicity in May 2011. Röösli did not abstain from the IARC Group 2B classification and should be well aware of that decision, but seems now to neglect that fact being an ICNIRP member. That may be due to the fact that the IARC classification contradicts the scientific basis for the ICNIRP guidelines."

Hardell et al. suggest to the Swiss government that Mr. Martin Röösli should be released from his duties as a scientist who is not objective and has substantial conflicts of interest. On the letter Röösli reacted by saying: "It's not a scientific letter. It sounds like activists who do not use scientific facts but who just attack people. It would be much more compelling if Lennart responded to my criticism of him in a scientific way instead of derailing the debate".

A recent publication of the COSMOS (October 2019) on the outcomes states reassuringly that "using mobile phones most extensively for making or receiving calls at baseline reported weekly headaches slightly more frequently at follow-up than other users, but this finding largely disappeared after adjustment for confounders and was not related to call-time in GSM with higher RF-EMF exposure. (See also the portrait of Anissi Auvinen)

Possible conflicts of interests

Röösli does "unpaid work" for the COSMOS study, which received considerable funding from telecom companies. In the 2019-publication on this study for example, Nokia and mobile network providers TeliaSonera and Elisa are mentioned in the category 'funding'.

According to his DOI he gets 70,000 Swiss francs a year for the Berenis work, from the Federal Office for the Environment.

He also received 16,000 francs for assisting in the Working Group Mobile Phone and Radiation the Federal Office for the Environment of the Swiss government.

The Swiss Tropical and Public Health Institute in which he plays a leading role, has a lot of corporate clients of which Swisscom, the biggest telecom company in Switzerland, of which the Swiss government holds 51% of the shares. In the annual Report 2019 the institute states that of the total budget of roughly 90 million Swiss francs, 78. 6 % was "competitively acquired" and 21.4 % came from "Core contributions".

Studies selected or self-directed by Röösli, were directly funded by the (Research Foundation for Electricity and Mobile Communication)

of which Martin Röösli is a member since 2011, according to his CV on the website of the Swiss Tropical and Public Health Institute. FSM is "a non-profit-making foundation with the purpose of promoting scientific research into the chances and risks of radio and electric power technologies that produce and use electromagnetic fields". The five founders of the FSM are:

ETH Zurich, Swisscom, Salt, Sunrise, 3G Mobile (liquidated in 2011) and the current main sponsors are Swisscom and Swissgrid. The sponsors are also represented in the FSM Foundation Board with one delegate out of seven.

Soichi Watanabe

Biography

Watanabe is currently Director of the Electromagnetic Compatibility Laboratory of the "National Institute of Information and Communications Technology (NICT).

He was a member of ICNIRP Standing Committee III since 2004 and is a member of the Commission since 2012.

He is a guest lecturer of several universities and at the Central Research Institute of Electric Power Industry.

Position

All publications to which Watanabe contributed as author point in the same direction: no effect. For example, this article about tumorgenenis in rats.

In 2019, he was co-author of an article which stated: 'To date, no adverse health effects of the EMF, linked to these applications, have been established.'

Possible conflicts of interest

As a guest lecturer at the Central Research Institute of Electric Power Industry he receives a small amount (about € 450 for each lecture, 1 or 2 a year).

He was co-author of the article with commission-member Hirata on the research funded partly by KDDI Foundation.

MEMBERS WHO HAVE LEFT THE ICNIRP COMMISSION IN MAY 2020

Maria Feychting

Biography

Maria Feychting is a Professor of Epidemiology at the Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.

She joined the Commission in 2008 and was elected vice chair in 2012. She left the Commission in May 2020.

Position

Feychting was in charge of the Swedish part of the Interphone study which concluded that there was no link between brain tumours and mobile phone use.

Feychting also conducted the Swedish part of the COSMOS-study, which in 2011 came to the conclusion that there was no increase in glioma in the Nordic countries that could be attributed to the use of mobile phones.

She recently repeated this point of view in the media in an article on the risks of 5G, which were none according to her.

According to this source she criticized the NTP-study on false grounds.

Possible conflicts of interest.

In a 2019 study in the context of COSMOS, she declared a declaration of interest as "vice chairman of the ICNIRP".

The telecom industry contributed € 5.5 billion to the funding (total € 19.2 billion) of the Interphone Study.

A 2016 publication on the Interphone Study once again mentioned industry funding by among other the Mobile Manufacturers Forum.

The Swedish part of the COSMOS-study was partly funded by the telecom industry: TeliaSonera, Telenor and Ericsson. In her Declaration of Interests for 2015 she declares that her Institute received a grant from industry sources which constituted "no more than 4% of her unit of epidemiology total income."

A 2011 study was partly funded by the Swiss Research Foundation on Mobile Communication, an organisation which is founded and funded by the telecom industry.

A 2012 study was funded by the Electric Power Research Institute (EPRI), an organisation funded by industry.

She did not mention these sources of funding in her Declarations of Personal Interest.

Adèle Green

Biography

Green is an Australian epidemiological scientist at the Queensland Institute of Medical Research, Australia and is the institute's Head of Cancer and Population Studies Group. She specialised in UV and skin cancer causation, harmful effects of UVR exposure in childhood and the prevention of melanoma. Apart from various Australian research bodies, she was also member of many committees at the International Agency for Research on Cancer (IARC) and contributed to the IARC monograph that led to classification in

Position

Although she focussed mostly on UV radiation, Green seemed to agree with her ICNIRP colleagues on the ICNIRP position, for example in this study from 2005 where Green was first author the research did not find any consistent or biologically relevant effect of specific radiation on cells. And another study from 2009 Epidemiologic Evidence on Mobile Phones and Tumor Risk, concludes by saying that "In the last few years, the epidemiologic evidence on mobile phone use and risk of brain and other tumors of the head has grown considerably. In our opinion, overall the studies published to date do not demonstrate a raised risk within approximately 10 years of use for any tumor of the brain or any other head tumor." And despite certain methodologic shortcomings and limited data on long-term use, "the available data do not suggest a causal association between mobile phone use and fast-growing tumors such as malignant glioma in adults, at least those tumors with short induction periods."

Conflicts of Interests

The declarations of interests of Dr Green have disappeared from the ICNIRP's website. The IARC Monograph mentions that Dr Green received "research funds (not exceeding 5% of total research support) from L'Oréal which makes products intended to reduce the dose from solar radiation."

Zenon Sienkiewicz

Biography

Sienkiewicz worked until his retirement in 2018 for Public Health England. There he led a research group that investigates the effects of ionizing and non-ionizing radiation. Since 2011 he has been a member of ICNIRP. He was also external expert for the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) report on 'Potential health effects of exposure to electromagnetic fields (EMF)', adopted in January 2015.

Position

Sienkiewicz systematically defends the position that there is no proof for any harm caused by non-ionizing radiation. In 2002, he said in the media: "The bottom line is there are no known mechanisms by which mobile phone radiation can increase the risk of cancer." Fifteen years later he still holds exactly the same position. In a 2017-article he stated that all the extensive research done has 'not identified any public health risks with any degree of certainty.' Moreover, it concluded that 'animal studies investigating the carcinogenic potential of exposure to multiple RF frequencies should not be given a high priority for research at this time.'

Possible conflicts of interest

A remarkable fact in his latest Declaration of Personal Interests is that he has shares in telecommunications multinational BT Group, one of the largest telecommunications companies in the world from 2003 to the present day. The gain is very little: about 100

pounds a year. But still: if you want to avoid the impression of conflicts of interest buying shares in a telecom company doesn't seem to be a wise decision.

He himself acknowledges this is a potential conflict of interest. Under an article published in 2017 the 'Statement on the Conflict of Interest' is: The authors declare that this work was conducted in the absence of any commercial or financial relationships that could be constructed as a potential conflict of interest, *except Sienkiewicz declares that he has owned 440 ordinary shares in BT Group, a communication services company.'*

In his 2015 Declarations of Interests he declares to have done since 2012 "Provision of research and scientific advice to UK government and other stakeholders". It is not specified who those other stakeholders were, but it can be assumed those were not civil society groups.

Also since 2009, he has been a consultant to the Rapid Response Group at the Japan EMF Information Center, which is funded by "Japan Electrical Safety & Environment Technology Laboratories, where he conducts reviews and analyses of recently published scientific studies

He was between 2001 and 2012 member of the Mobile Telecommunications Health Research (MTHR)-programme. The programme did not find any association between exposure to mobile telephone communication and an increased risk of developing cancer. In the final report of the programme we read that that the core funding was provided in approximately equal share by government and industry. He systematically defends the point of view that there are no health risks associated with non-ionizing radiation. He was co-author of the 2019 article which criticized the NTP-study.

SCIENTIFIC EXPERT GROUP

Jacques Abramowicz

Biography

Jacques Abramowicz is Professor of Obstetrics and Gynecology and the Director of the Ultrasound Services at the University of Chicago.

He is a member of the Scientific Expert Group since May 2016.

Position

Abramowicz is, says his personal page at Chicago University, "an expert in the use of ultrasound for prenatal diagnosis of foetal anomalies and screening for early detection of ovarian cancer."

As far as we could find out, he did not perform research into the health effects of mobile phone radiation.

Possible Conflicts of Interest

In his declaration of personal interests Abramowicz doesn't mention possible conflicts of interest and we did not find information that contradicts this.

Anssi Auvinen

Biography

Auvinen is currently a professor of Epidemiology at the School of Health Sciences, University of Tampere in Finland. He is a member of ICNIRP's Scientific Expert Group since 2013. He was also external expert for the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) report on 'Potential health effects of exposure to electromagnetic fields (EMF)', adopted in January 2015.

Position

In harmony with all ICNIRP-members Auvinen criticizes research that seems to show an association between health problems and mobile phone use. Although there have been individual reports of associations between MP-use and tumours, this research is not consistent and on balance does not provide evidence of an association,' he and his co-authors wrote in 2008. His own research systematically shows no association between health problems and non-ionizing radiation.

Auvinen participated in the Finish Cohort Study of Mobile Phone Use and Health (COSMOS). A recent publication (October 2019) on the outcomes states reassuringly that "using mobile phones most extensively for making or receiving calls at baseline reported weekly headaches slightly more frequently at follow-up than other users, but this finding largely disappeared after adjustment for confounders and was not related to call-time in GSM with higher RF-EMF exposure. Tinnitus and hearing loss were not associated with amount of call-time." In another publication on the COSMOS-outcomes (April 2020) an association between sleep quality and mobile phone use is also not found.

Possible conflicts of interest

In his Declaration of Interests he submitted to ICNIRP he states that he in 2014 and 2015 received research € 100,000 funding from the Mobile Manufacturers Forum, an international organization founded in 1998 by leading manufacturers of mobile phones and radio equipment, such as Alcatel, Ericsson, Mitsubishi Electric, Motorola, Nokia, Panasonic, Philips, Sagem, Samsung, Siemens and Sony Ericsson.

The funding was for the COSMOS-study. In the 2019-publication on this study Nokia and mobile network providers TeliaSonera and Elisa are mentioned in the category 'funding'.

Another recent article states that Auvinen received 'consulting fees from Epid Research Inc.' According to his Declaration of Interest he received a fee of € 1000 in 2015 and 2017. Not in

his declaration of interest is that he received lecture fees from pharmaceutical companies Glaxo Smith Kline and MSD. Maybe one can argue that these companies do not operate in the field of non-ionizing radiation. But to avoid conflicts of interests it seems wise to be transparent about all fees and funding received from industry.

Christian Cajochen

Biography

ICNIRP's website states that Cajochen studied natural sciences followed by a 3-y postdoctoral stay at the Harvard Medical School in Boston, USA. He leads the Centre for Chronobiology at the University of Basel and focusses on the influence of light on human cognition, circadian rhythms and sleep, circadian related disturbances in psychiatric disorders, and age-related changes in the circadian regulation of sleep and neurobehavioral performance.

He serves as associate editor for established sleep-related scientific journals and is editor in chief for "Clocks&Sleep".

He started as a member of the Scientific Expert Group (SEG) in May 2018.

Position

As stated Cajochen focusses on the influence of lights and far as we could find out, he did not perform research into the health effects of mobile phone radiation.

Possible conflicts of interest

In his DOI it is stated that he studies the "effects of day LED on human performance, melatonin and sleep. Research studies in healthy human volunteers partially sponsored by Toshiba Materials." In the period from 2014-2018 that accounts for 120.000 (we assume euro), whereby Toshiba has the right "to request (i) revisions to the publication, so that no Confidential Information is inadvertently disclosed or a delay of not more than 60 days to allow for protection of any potentially patentable subject matter by filing of a patent application."

Toshiba does not focus on telecommunications, but rather on mainly infrastructure energy and Electronic Devices.

Jose Gomez-Tames

Biography

Gomez-Tames is Research Associate Professor in Nagoya Institute of Technology.

He is also Working Group Chair of the Subcommittee on EMF Dosimetry Modelling of the IEEE International Committee on Electromagnetic Safety from 2017.

Gomez-Tames is member of the Scientific Expert Group since 2018.

Position

Gomez-Tames work is more on the modelling of non-ionizing radiation than on the health effects.

Possible conflicts of interest

See Van Rongen and others on the role of IEEE/ICES.

In his Declaration of Personal Interest Gomez-Tames doesn't mention other sources of possible conflicts of interest and we did not find information that contradicts this.

Penny Gowland

Penny Gowland worked at the University of Nottingham School of Physics and Astronomy until 2016 and is now retired. She did a PhD in Magnetic Resonance Imaging from the Institute of Cancer Research in 1990.

According to ICNIRP's website "her work at high field and on foetal development as led her to take a strong interest in the interactions of EMF with the human body, and safety aspects of MRI."

Penny Gowland is a member of the ICNIRP Scientific Expert Group (SEG) since March 2013.

Position

She declared in her DOI that her "research interests are in MRI: but I am also academically and professionally interested in any biological effects of EMFs."

As stated Gowland focussed mainly on MRI and far as we could find out, she did not perform research into the health effects of mobile phone radiation.

Possible conflicts of interest

According to the organisation AVAATE her previous Declaration of Interests, she reported that she has held many research contracts with Phillips Electronics but without any money involved. Gowland has been part of the MR safety working group of British Institute of Radiology. According to the British Institute of Radiology website, Phillips and Siemens are platinum sponsors.

In 2015 AVAATE also stated that the European Society for Magnetic Resonance in Medicine and Biology (ESMRMB), organization mentions that Gowland was a member of several committees, including the Committee on Security, and has received financial support from companies like Hitachi, Philips, Siemens, Toshiba and General Electric.

John Hanifin

Biography

John Hanifin is laboratory director of the Light Research Program at Thomas Jefferson University.

He is a member of the Scientific Expert Group since May 2018.

Position

Hanifin is specialized in the effects of light. A recent publication he contributed to is for example is about the effect on nurse and patient experience of the overnight use of blue depleted illumination. He did not conduct research on the health effects of mobile communications technologies.

Possible conflicts of interest

The Light Research Program received industry support from among others OSRAM, Philips Lighting and Panasonic.

His PhD-thesis (2015) was also party funded by industry, by Philips Lighting, Apollo Lighting and OSRAM.

Hanifin's Declaration of Personal Interest shows that his laboratory earns about 5% of its yearly income by conducting clinical research for Bios Lighting. It mentions that his laboratory is obliged to submit a manuscript to the sponsor before publication for review and comment, 'however Sponsor shall not exercise editorial control over the publication'. The fact that the sponsor can review and comment the manuscript does not seem to be a strong guarantee of independence.

Jukka Juutilainen

Biography

He is a retired professor emeritus of Radiation Biology and Radiation Epidemiology, and Department Head of the Department of Environmental Science at the University of Eastern Finland. Juutilainen teaches generic courses on environmental health and risk assessment, as well as specific courses on non-ionizing and ionizing radiation

He is an Associate Editor of *Bioelectromagnetics*, effective immediately for which he was nominated by the European Bioelectromagnetics Association (EBEA) a non-profit scientific association with many current and former ICNIRP-members.

He was a member of the ICNIRP Standing Committee on Biology from 2004 until 2012 and became a member of the Scientific Expert Group (SEG) in March 2013.

Position

In 2007, *Microwave News* reported positively about a study published by Juutilainen: "Every now and then a new paper comes along that gives hope that one day we'll make sense of the conflicting results that have become the hallmark of EMF research." The study was financed partly by the cell phone industry —the MMF and the GSMA and although Juutilainen suggested that needed a follow-up it never got one.

Another study from 2007 concluded that "the data did not show any effects of radiofrequency electromagnetic fields on micronucleus frequency in erythrocytes. The findings were consistent in two mouse strains (and in a transgenic variant of the second strain), after 52 or 78 weeks of irradiation, at three SAR levels relevant to human exposure from mobile phones, and for three different mobile phone signals." The study was co-funded by Nokia, Elisa Communications Corporation, Finland Benefon, Finland Sonera.

Juutilainen published this study in 2009, together with Croft and Van Rongen, on the 'Effects of Radiofrequency Electromagnetic Fields on the Human Nervous System'. The conclusion was that "However, in provocation studies a causal relation between EMF exposure and symptoms has never been demonstrated. There are clear indications, however, that psychological factors such as the conscious expectation of effect may play an important role in this condition."

Possible conflicts of interest

In his past ICNIRP Declaration of Interests, he stated that he has received research funding from government organizations and foundations.

In his last non-signed DOI he indicates "The Department of Environmental and Biological Sciences of the University of Eastern Finland (UEF) has received funding from the Electric Power Research Institute (EPRI). Although EPRI is an independent, non-profit research organization (and therefore not reported above in research support received from commercial entities), this funding might be perceived as affecting my independence (Period: 2015-2019)."

According to AVAATE he had "numerous research programs funded by Nokia, Benefon, Sonera, Elisa, FINNET, the GSM Association and the Mobile Manufacturer Forum." For example, the national research programme on possible health effects of mobile phones in Finland (from 1998 to 2003) which was coordinated by Juutilainen was mainly funded by TEKES, National Technology Agency a governmental organisation, and also supported by Nokia, Benefon, Sonera, Elisa, Radiolinja, Finnish 2G, Mobile Manufacturers Forum and the GSM Association.

He has participated in conferences and publications funded in part by organizations with interests in the telecommunications sector.

Masami Kojima

Biography

Masami Kojima is a professor of Kanazawa Medical University. He is specialized in ocular damage due to microwaves.

In the period 2001-2004 he was a consulting member for ICNIRP, since november 2014 he is a member of the Scientific Expert Group.

Position

Kojima's research is mainly on the effects of microwaves on the eye, often of rabbits. In his publications, we found no direct statements about possible effects on the eye within the ICNIRP-norms.

Possible conflicts of interest

He was co-author of the 2010 article partly funded by KDDI Foundation (see Hirata and Watanabe).

His Declaration of Personal Interest does not mention other sources of possible conflicts of interest and we did not find any.

Ilkka Laakso

Biography

He is Professor of Electromagnetics in Health Technologies at Aalto University, Finland and focuses on theoretical and computational bioelectromagnetics at both extremely low and radio frequencies. Laakso has been "combining computational electromagnetics with medical image processing and biological neuron modelling." The purpose of this research is to offer the medical and electrical engineering community new computational methods for individual physical modelling of the human body.

According to ICNIRP's website he is the "secretary of Subcommittee of EMF Dosimetry Modeling (SC6) of the IEEE International Committee on Electromagnetic Safety and a working group chairman since 2015."

Laakso became a member of the Scientific Expert Group (SEG) in 2016.

Position

A study from 2009 (Assessment of the Computational Uncertainty of Temperature Rise and SAR in the Eyes and Brain Under Far-Field Exposure From 1 to 10 GHz') about the specific absorption rate (SAR) seems to suggest that the 'reference levels by ICNIRP and maximum permissible exposure limits by IEEE seemed to be conservative in the sense that at the reference levels the temperature rise in the eyes and brain was always less than 1◦C."

Possible conflicts of interest

For IEEE/ICES see Van Rongen and others.

According to his DOI for ICNIRP he owns stocks of and is a board member of 'Fieldsim Oy', a consulting company in Finland that does computer simulations of electromagnetic fields, including electromagnetic field exposure.

Isabelle Lagroye

Biography

Isabelle Lagroye is a director of studies at the Ecole Pratique des Hautes Etudes (EPHE) and works at Bordeaux University. Her research, states a recent publication, 'deals mainly with the biological and toxicological effects of non-invasive electromagnetic fields.' She is currently member of the Bruxelles-Capitale expert committee on non-ionising radiations.

She was member of an ICNIRP committee in the period 2009-2012 and was elected member of the Scientific Expert Group in March 2013.

Position

In 2018 Lagroye together with two other scientists published an article in *European Scientist* in which she concluded that the NTP-study "consolidates current knowledge and reinforces the fact that when effects of mobile radiofrequency fields can be observed, it is at exposure levels that far exceeds the maximum permissible exposure values. In practice, these limits cannot be reached with commonly used wireless communication technologies (relay antennas, mobile phones, Wi-Fi …)."

This statement seems to be in contradiction with findings from her own research. A recent publication of which Lagroye was co-author concludes: 'However, we found that exposure to GSM-modulated 1800 MHz signals at 2 W/kg decreased the PMA maximal efficacy to activate both RAS and ERK kinases' activity.' So, it influences the signaling between proteins.

This is an effect at 2 W/kg, while according to the new ICNIRP-norms health effects in head and torso are only above 20W/kg and the norm is, with a safety factor of ten, 2W/kg.

Lagroye was also co-author of the final report of the Geronimo-project. In this report, we do find indications for health effects. It says:

"Results suggest that increased RF dose to the brain and longer mobile phone call time may be associated with risk of hyperactivity and conduct problems."

And: "a meta-analysis among four birth cohorts (n=55,507) indicated that maternal cell phone use during pregnancy may be associated with shorter pregnancy duration and increased risk for preterm birth (Tsarna et al., 2019, accepted Am J Epidemiol)."

Interesting is also that research conducted by Lagroye seems to suggest non-thermal effects, while ICNIRP states that thermal effects are the only ones for which there is scientific evidence. In this article the authors write: "Altogether, our experimental findings provide

evidence for dose-dependent effects of RF signals on the bursting rate of neuronal cultures and suggest that part of the mechanism is non-thermal."

In 2009, she co-authored a scientific paper with Van Rongen and Croft which stated on the 'effects of radiofrequency electromagnetic fields on the human nervous system' that "there are clear indications, however, that psychological factors such as the conscious expectation of effect may play an important role in this condition."

Possible conflicts of interest

The latest Declaration of Personal Interest of Lagroye that can be found on the ICNIRP-site dates from 19 October 2015, almost five years ago. At that moment, she stated that she got 2,35% of the income of her research unit from a commercial partner, the Réseau de Transport d'Électricité (RTE).

A study published in 2010 which suggested that exposure to WiFi did not damage the brains of young rats was funded by France Telecom and La Fondation Santé et Radiofréquences, an organisation that is for the half funded by industry.

This organisation also partly funded several other studies to which she contributed, like this one published in 2011 and this one published in 2012.

Another 2012 publication was partly funded by Bouygues Telecom.

Sarah Loughran

Biography

ICNIRP's website states that Loughran is currently a researcher at the University of Wollongong in the Australian Centre for Electromagnetic Bioeffects Research (ACEBR) human neurophysiology research group, an NHMRC Centre of Research. She studied physiology and psychology and got a PhD in cognitive neuroscience/psychophysiology at Swinburne University of Technology, investigating the effects of electromagnetic fields on human sleep, the electroencephalogram (EEG), and melatonin.

To this centre (ACEBR) also ICNIRP-chair Rodney Croft and ICNIRP-member Andrew Wood are connected. Swinburne university and in particular the Radiofrequency Dosimetry Laboratory is part of the ACEBR which has a very close relationship with and is co-funded by Telstra, the biggest Telecom company in Australia. (See also portraits on Woods and Croft)

Loughran is also a member of the current World Health Organisation (WHO) RF Environmental Health Criterion evaluation committee, and is on the board of directors for the Bioelectromagnetics Society (BEMS). She is a member of the ICNIRP Scientific Expert Group (SEG) since March 2013.

Position

A 2005 study by Loughran and Woods on the effects of EMF on human sleep demonstrated that "a short exposure to mobile phone-type radiation has an effect on subsequent sleep EEG, although no conclusions can be made regarding adverse health consequences as the mechanisms of the effects are still unknown."

In 2007 Microwave News reports that "the ability of mobile phone radiation to affect sleep is emerging as a robust low-level effect. A team led by Bengt Arnetz has reported that a three-hour exposure to GSM radiation at 1.4W/Kg an hour before bed can disrupt sleep." This study supported earlier findings of Peter Achermann of the University of Zurich and Loughran at the time working at the Brain Sciences Institute at Swinburne University.

Because later findings of other studies got quite some media attention, Loughran, Peter Achermann & Niels Kuster published a statement to temper the seriousness of the findings.

Loughran worked for some years in Switzerland, where several scientists like Kuster do research on EMF and sleep. *The Nation* reported that Niels Kuster, a Swiss engineer co-authored in *The Lancet Oncology* a summary of the WHO's findings of the Interphone study which was launched by the WHO's International Agency for Cancer Research in 2000 (and to which two wireless trade associations contributed $4.7 million or 20 percent of the $24 million budget). Kuster had filed a conflict-of-interest statement affirming that his research group had taken money from "various governments, scientific institutions and corporations." But after his publication The Lancet "issued a correction expanding on Kuster's conflict-of-interest statement, noting payments from the Mobile Manufacturers Forum, Motorola, Ericsson, Nokia, Samsung, Sony, GSMA, and Deutsche Telekom. Nevertheless, Kuster participated in the entire 10 days of WHO-deliberations."

In general, Loughran (ACEBR) is in agreement with Croft. In an interview with Computerworld: "*There are people that are suffering and yes, it's **not due** to electromagnetic energy exposure, it's more of a **psychosomatic** condition…*"

According to a 2017 study "IEI-EMF provocation case studies: A novel approach to testing sensitive individuals" of which Loughran is the second author "*the present experiment failed to show a relationship between RF-EMF exposure and an IEI-EMF individual's symptoms*". The information on Electro hypersensitivity from the WHO's EMF Project (see also History chapter in this report) to which Loughran is connected has not been updated since 2005.

Possible conflicts of interest

In her DOI she declares for 2015 having received almost 16.000$ from EPRI funding a*nd NPF research Institute, which accounted "approximately for 5% of her lab's income".*

In a 2016 EPRI workshop "Loughran provided an overview of the current state of knowledge in the field of human laboratory studies, an assessment of the critical gaps in knowledge, and recommendations for research priorities. Loughran and the session rapporteur, Rodney Croft, University of Wollongong, led the workshop participants in a discussion of human laboratory studies".

See also portraits on Croft and Wood.

Jack Lund

Biography

Jack Lund was research physicist with the US Army Medical Research Command. There he studied the effect of laser radiation on ocular tissue and the visual system. He retired in 2018.

He was an ICNIRP Consulting Expert from 2002 to 2012. He joined the Scientific Expert Group in 2018.

Position

Jack Lund is an expert in laser safety issues. He did not publish article about the health effects of mobile communication technologies and did not make, as far as we could find out, make public statements about it.

Possible conflicts of interest

Lunds 'Declaration of personal interest' is completely empty. We did not find other information about possible conflicts of interest.

Simon Mann

Biography

According ICNIRP's website Simon Mann is a chartered electrical engineer and heads the Physical Dosimetry Department at Public Health England's Centre for Radiation, Chemical and Environmental Hazards. Man is responsible for programmes of scientific work to develop health-related advice on exposures to electromagnetic fields (EMFs) and optical radiation across the UK.

He was secretary to the independent Advisory Group on Non-ionising Radiation (AGNIR), and member of the IARC Working Group that evaluated the carcinogenic potential of radiofrequency EMFs in 2011. He currently works with WHO EMF Project (see also history part) to develop its Environmental Health Criteria monograph on radiofrequency fields.

He is also active in technical standardisation and is a UK delegate to the CENELEC TC106X Committee.

During a meeting of the WHO EMF Project in 2013 Lindsay Martin from ARPANSA – (Australia) and Simon Mann (PHE - UK) were elected chair and vice chair respectively. In the meeting J. Keshvari from International Committee on Electromagnetic Safety (ICES)and the International Electrotechnical Commission (IEC) TC 106 said that "Maintenance work is in hand on several EMF exposure Standards. Harmonisation and avoiding duplication of effort,

between CENELEC, IEEE and ITU is encouraged where possible." Keshvari also mentioned that IEEE/ICES "has been developing an RF safety Standard for NATO".

He is a member of the ICNIRP Scientific Expert Group since 2015.

Position

Mann is part of the close network of ICNIRP and WHO EMF scientists that claim there are no real immediate health effects from EMF. For more on the WHO Project and EMF IEEE/ICES, see the history part of this report and the portrait of Croft and Van Rongen.

Possible conflicts of interest

We could not find a recent DOI on ICNIRP's website: the link to Mann's DOI on ICNIRP's website is not functioning.

However, he did not mention in his former Declaration of Interests statement submitted to ICNIRP, that he has received research funding from the GSM association, the Mobile Manufacturer Forum and the UK's Mobile Telecommunication and Health Research Program (MTHR), on which he still plays a leading role. According to AVAATE MTHR in the past received funding from the Vodafone, a wireless company.

Since 2009 he has been a member of BEMS and the EBEA22.

Rüdiger Matthes

Biography

Rüdiger Matthes was from 1989 until his retirement in 2016 Head of the group "Non-Ionizing Radiation (Dosimetry)" at the German Federal Office for Radiation Protection. He became the Scientific Secretary of ICNIRP in 1993. He was Chairman of the Standing Committee on Physics and Engineering (SCIII) from 2004 to 2008. He became Vice-Chair in 2008, and Chair again in 2012. Since 2016 he is a member of the Scientific Expert Group.

Position

Matthes defends the position that there are no studies that prove the existence of non-thermal health effects of non-ionizing radiation and that no plausible mechanism has been described whereby these effects could take place. There is no evidence for a link between cancer and the use of mobile phones, he said in 2010.

Matthes was one of the authors of a recent ICNIRP-publication in which ICNIRP explains the principles for health protection on which its guidelines are based.

Possible conflicts of interest

In his Declaration of Personal Interests Matthes does not mention any possible conflict of interest and we did not find any information that contradicts this.

During a meeting of the WHO EMF Project in 2013 Matthes spoke on behalf of both BfS and ICNIRP by stating that: “Exposure recommendations have been developed by several organisations such as ICNIRP and IEEE/ICES, and there is good harmonisation between these on fundamental limits.”

John *O'Hagan*

Biography

On ICNIRP’s website it says that John O’Hagan heads the Laser and Optical Radiation Dosimetry Group at Public Health England. This research group covers all aspects of optical radiation dosimetry, including both the beneficial and detrimental effects of optical radiation on people.

He is Vice-President Standard of the International Commission on Illumination (CIE), Convenor of the International Electrotechnical Commission Technical Committee 76 “Optical Radiation Safety and Laser Equipment” Working Group 9 “Non-coherent sources”, Chairman of the British Standards Committee EPL/76 “Optical Radiation Safety and Laser Equipment” and is a member of a number of other national and international committees.

According to his DOI he was also a member of EU SCENHIR/SCHEER Working Group on Potential risks to human health of Light Emitting Diodes (2016-2018) and is a Member of WHO Core Group on NIR Basic Safety Recommendations.

He joined the ICNIRP Scientific Expert Group (SEG) in March 2013.

Position

In 2017 O’Hagan co-wrote a chapter in Clay's Handbook of Environmental Health in which the general line of ICNIRP, SCENHIR and WHO EMF Project is repeated: no adverse health effects.

Possible conflicts of interest

In his DOI he states under activities “Provision of scientific support and advice to government and other stakeholders”, but fails to mention which stakeholders.

In his statement, he says that he is the President of the Committee EPL/76 Optical radiation safety and laser equipment, of BSI Standards Development (BSI is a company that sets rules to help organizations worldwide achieve excellence). Organizations that work with this committee include the Association of Industrial Laser Users, the Association of Manufacturers of Domestic Appliances, GAMBICA Association Limited (a UK national organisation representing the interests of companies in the instrumentation, control, automation and laboratory technology industry) the Institute of Physics, the Institution of Engineering and Technology, the Institution of Mechanical Engineers, and the Lighting Industry Association.

He also reports that he is the Vice President of the CIE-UK National Illumination Committee of Great Britain. This committee was established by the Illuminating Engineering Society of

Great Britain, the Institute of Electronic and Electrical Engineers, the Institute of Gas Engineers, and the NPL, in collaboration with industry and professional associations, government departments and lighting technicians.

Chiyoji Ohkubo

Biography

Chiyoji Ohkubo is Director of the Japan EMF Information Center (JEIC). This organisation was established in July 2008 'to facilitate communication on EMF issues among government agencies, industry, the media and the general public.'

In the period 2005-2007 he worked for the EMF WHO-project.

He is a member of Scientific Expert Group since March 2013.

Position

All his publications seem to fit into the same category: no effect. See for example this study in which the exposure of rats to RF EMF radiation did not alter their cerebral microcirculation.

Possible conflicts of interest

For criticism of the WHO EMF Project see among others Van Rongen.

The Japan EMF Information Center, writes Okhubo himself, 'has been financed from donations by stakeholders and governmental funds.' An information leaflet of the organisation says: 'The JEIC is founded to present in a neutral way the positions of industry, science and society, and to discuss the risk analysis.' It seems to be no coincidence that industry is mentioned first.

Ohkubo did research funded by the Association of Radio Industries and Businesses (ARIB), Japan.

Margarethus Paulides

Biography

Margarethus ('Maarten') Paulides obtained his MSc in Electrical Engineering at Eindhoven University in 2002 and his PhD in Medical Electromagnetics

He works as Associate Professor, Department of Electrical Engineering, Electromagnetics, at the university of Eindhoven as well as Associate Professor, Erasmus Medical Centre in Rotterdam.

The outcome of his research were novel devices, patient-specific simulation technology and pioneering data and knowledge for improving EMF exposure guidelines.

Since 2015 he is board member of the Dutch National Antenna Research Framework (NARF). From 2017, he serves in the Electromagnetics Committee of the Dutch Health Council that advises the relevant ministers in the Netherlands on EMF related subjects. He also is a Management Committee member and Workgroup leader in COST action CA17115.

He is a Member of the ICNIRP Scientific Expert Group (SEG) since 2017.

Position

Most of his research is focussed on applications in health monitoring, disease diagnosis and therapy. We did not find much research on the health effects of radiofrequency radiation.

He did some research on thermal effects on tissue which resulted in this 2018 study in which the authors basically state that the protection levels of ICNIRP and IEEE are conservative and safe: "To protect against any potential adverse effects to human health from localised exposure to radio frequency (100 kHz-3 GHz) electromagnetic fields (RF EMF), international health organisations have defined basic restrictions on specific absorption rate (SAR) in tissues. These exposure restrictions incorporate safety factors which are generally conservative so that exposures that exceed the basic restrictions are not necessarily harmful."

Possible conflicts of interest

According to the ICNIRP website he "also acts as advisor of start-up companies aimed at providing solutions for computer simulation and image guided interventions".

His DOI further states that he does paid consultancy for a company Sensius.biz, which in fact he co-founded, for an amount of 5000€. He also owns 4,9% in stocks of this company.

The same amount he got from a German company Sennewald Medizin Technic.

He received a 45.000€ research Grant form General Electric Research Centre in Germany.

For the contracting company Phillips he received a STW research grant of 10.000€ in cash and 66.300€ in kind.

Kensuke Sasaki

Biography

Kensuke Sasaki is a Researcher of the National Institute of Information and Communications Technology, Japan.

He is a member of Subcommittee of EMF Dosimetry Modelling of IEEE International Committee on Electromagnetic Safety. He is also an expert for a committee of the International Electrotechnical Commission.

He joined the Scientific Expert Group in November 2018.

Position

Most publications of Sasaki are about how to measure the effects of non-ionizing radiation and about the thermal effects of it on for example the eye. We did not find direct statements about the health effects.

Possible conflicts of interest

For information about IEEE/ICES see Van Rongen.

Together with Hirata and Watanabe (see above) he conducted research published in *IEEE Transaction* in 2010 partly funded by KDDI Foundation.

David Savitz

Biography

Savitz is currently Professor of Epidemiology and Obstetrics and Gynecology, at the American Brown University.

His teachings and research is focussed mainly on epidemiologic methods and, reproductive, environmental, and cancer epidemiology and he authored a book entitled "Interpreting Epidemiologic Evidence".

He was a member of the ICNIRP Standing Committee on Epidemiology from 1997 until 2012 and then became a member of the ICNIRP Scientific Expert Group (SEG) in 2013.

Position

Given the fact that he has been connected to ICNIRP for 23 years we can safely assume that he agrees with the position of this NGO on health effects of EMF.

Possible conflicts of interest

His Declaration of Interests statement to ICNIRP, says that he does paid consultancy but "non-relevant to ICNIRP".

According to AVAATE this is not really true: "He gave expert witness testimony on behalf of the defendants in a January 2012 lawsuit in Federal District court in Portland, Oregon."

A company AHM Wireless sued the Portland Public School System, because it called for the removal of a Wi-Fi system in the schools. The testimony of Savitz was requested to assess the expertise of plaintiffs' claims that the implementation of wireless devices and wireless systems in the schools could possibly cause cancer or other adverse health effects.

In court, he states that the purpose of his contract with Battelle was to investigate relationships between environmental agents and human health and that he had a variety of sponsors, including some federal government agencies and other groups that he does not recall at this time.

Remarkably when he is asked about his ICNIRP membership he said that doesn´t consider himself to be really an active member and that he contributed all those years to just four reports, together with Anders Ahlbom, who coordinated their advice work for ICNIRP and whom had also recruited Savitz to join ICNIRP (in 2011 was asked to step down from IARC panel after he was exposed to be on the board of his brother's consulting firm, which telecom clients). Savitz: "My understanding of the organization is really actually quite limited. My role in it has been much narrower to participate in the evaluation of evidence and the reporting of the results of that evaluation. I have not been involved in what's done with that evaluation."

When the lawyer of the public school asks "So the organization, though, it's involved with the protection of human beings from non-ionizing radiation; is that correct?" Savitz answers: "Again, my understanding is not much deeper than as you described it based on the name of the organization. My understanding is that they evaluate evidence and make recommendations that are intended to be protective of health."

When asked if he is paid to be part of scientific committees, he says that he remembers only travel expenses being reimbursed by ICNIRP. He says he doesn´t even remember how many scientific committees he belongs to. He wasn´t involved with what ICNIRP does in making decisions after it receives the results of the evaluation carried out by the Standing Committee on Epidemiology. He says that he has never read the ICNIRP Statutes, its mission, etc. He maintains that he is hired to help evaluate a particular line of research. Also, when asked if there was any relationship between ICNIRP and the WHO for the work in which he contributed to, he said he did not know.

It almost seems as if Savitz does not want to be remembered too much about ICNIRP and tries to distance himself from the NGO and its position. When the lawyer of the public schools asks "you would agree then that we need protection from non-ionizing radiation; is that correct?", Savitz answers: "Well, that's not something that I get involved in the technical judgment of the sort of guidelines or regulations or decision-making. If you're asking, obviously there are levels of exposure that I'm aware that can be harmful, so that I can understand in a general way that it makes sense that there be consideration of regulation."

AVAATE notes that "when asked whether he has been paid out of funds acquired from companies and/or telecom consultants and law firms that represent these companies, he replied that there are a few cases where he has done research funded by the electric utility industry. However, he emphasized that the funders tried to isolate his work from the source of funding. He says he once had done a study before realizing where the money came from."

Savitz also stated that he has done work sponsored by EPRI, as many ICNIRP members, which is funded by the electrical power industry.

There is no record of these kind of data in the Declaration of interests that he submitted to ICNIRP.

Karl Schulmeister

Biography

Karl Schulmeister is since 1994 head of the Laser, LED and Lamp Safety group at Seibersdorf Laboratories in Austria. On his LinkedIn profile he describes himself as 'Consultant on Laser and Optical Radiation Safety'.

He was a member of the ICNIRP Standing Committee on Optical Radiation in the period 2008-2012 and joined the Scientific Expert Group in March 2013.

Position

Karl Schulmeister is specialized in optical radiation. He did not perform research on the health effects of radiofrequency radiation.

Possible conflicts of interest

Seibersdorf Laboratories is a firm, not an academic institution. Schulmeister's group derives, according to his Declaration of Interest, about 10% of its income from paid consultancy.

Research for an article published in 2015 and a white paper published in 2016 received both the support of the Laser Illuminated Projector Association, which presents itself as "a single industry voice in rationalizing laser regulations".

David H. Sliney

Biography

Sliney serves as chair of the IES Photobiology Committee and holds a Ph.D. in biophysics and medical physics from the University of London, Institute of Ophthalmology. He worked for the US Army Public Health Center for 42 years, serving as Program Manager, Laser/Optical Radiation Program, until retiring in 2007.

Het still acts as Safety Director, American Society for Lasers in Medicine and Surgery; And he remains an associate faculty member of the Johns Hopkins School of Public Health, Department of Environmental Science and Engineering, Baltimore, MD.

He served as member, advisor and chairman of numerous committees that are active in the establishment of safety standards for protection against non-ionizing radiation (ANSI, ISO, ACGIH, IEC, WHO, NCRP).

He has been an ICNIRP Commission Member from the very start in1992 until 2004 and as Chairman of ICNIRP SCIV (optical radiation) from 1998 until 2004. He is a member of the ICNIRP Scientific Expert Group (SEG) since November 2017.

Position

Sliney has been mainly focussing on safety and health issues of laser lights, UV light or other sources, important for safety for medical staff who work with laser application in surgery

and medicine. Also, scientists and military staff are risk groups for laser damage to the eye. We could not find research on the health effects of radiofrequency radiation.

Which does not mean that he is not involved in the scientific debate. In 2013 for example he participated in a webinar by the American Conference of Governmental Industrial Hygienists (ACGIH) on electromagnetic radiation.

In an article from 2017 on the history of of ICNIRP founder Mike Repacholi explicitly gives a special thanks to long-term INIRC and ICNIRP member David Sliney for his help with reviewing the article.

In a book from 2000 in 'the NATO Science Series' by B.Jon Klauenberg (US Air Force Research Laboratory) and also NATO-liaison, Sliney is described as "Dr Dave Sliney and army employee who serves on the ICNIRP". Klauenberg who in the first years led the WHO EMF Project together with Repacholi, is a prominent figure from the US Department of Defense (DOD) and describes it as follows: "Because the US military services operates globally and with many different national partners, uniformity of the RFR exposure standards is a desirable goal." He then describe the various ways that the DOD contributes to "worldwide standards harmonisation". So, the DOD participates in the WHO EMF project for example "through active engagement of US Air Force Research Laboratory as well as US army personnel providing service on the IEEE". And Sliney thus seems to be the US army representative in ICNIRP.

Possible conflicts of interest

His DOI is signed in 2019 but does not mention much.

Rianne Stam

Biography

Rianne Stam is senior scientist at the National Institute for Public Health and the Environment (Bilthoven, the Netherlands) since 2007. There she performs risk assessments and policy research on the biological effects and possible health risks of electromagnetic fields (EMF).

She is a member of the Scientific Expert Group since March 2013.

Position

Stamm made in 2015 and 2019 overview reports of the long term effects of electromagnetic fields on the health of workers. The conclusion: 'Scientific research has not yet proven any links between the exposure of workers and the occurrence of cancer, disorders of the nervous system or other illnesses in the long term.'

Possible conflicts of interest

According to her 'Declaration of Personal Interest' Stam has no possible conflicts of interest and we did not find any information that contradicts this.

Bruce Stuck

Biography

Bruce E. Stuck He is now retired. He was from 1992-2010, the Director of the U.S. Army Medical Research Detachment of the Walter Reed Army Institute of Research, where he had responsibilities for the Army Medical Department's laser and radio frequency radiation biological effects research program. Until 2013 he was the Director of the Ocular Trauma Research Division at the U.S. Army Institute of Surgical Research in San Antonio, Texas.

Since 2012 Stuck is a part-time independent consultant on non-ionizing radiation bioeffects.

He has been a member of ICNIRP SC IV since 1999 and of the Commission from 2004 until 2016. Stuck is now supporting the work of the Project Group as a SEG member.

Position

His research focussed on laser and radio frequency radiation biological effects and "establishes protection strategies (e.g. exposure limits or physical protection products) and develops triage and treatment approaches for ocular injury from non-ionizing radiation and shock wave exposures from blast". During his 32 years-experience in laser hazards research experience he was author/co-author of numerous papers on ocular and cutaneous effects of laser and radio frequency radiation. His primary interests are in the biological effects of visible and infrared laser radiation on the retina and cornea and the assessment of laser-induced eye injuries and their treatment.

Possible conflicts of interest

His DOI states that he is "a consultant to Perfect Lens, LLC on a proprietary project under a signed confidentiality agreement to provide advice and written assessment on biological exposure limits as applied to their repetitively pulsed fem to second laser application for use in medical application in the eye". He delivered oral and written reports on the device hazard assessments. Income was less than 1% of personal income from his retirement annuity in 2018 tax year.

John Tattersall

Biography

John Tattersall **i**s scientist in the Defence Science and Technology Laboratory, a government Agency which provides research and advice for the UK Ministry of Defence and other

government departments. He also is Honorary Senior Lecturer in Clinical Neurosciences at the University of Southampton.

He was a member of the IEEE International Committee on Electromagnetic Standards from 2012 until 2017.

He joined the Scientific Expert Group in March 2013.

Position

Twenty years ago, Tattersall did research that showed effects of RF Radiation on the brain of rats. *New Scientist* wrote: "Last year, fears about mobiles affecting brain function received fresh impetus thanks to work by John Tattersall and his colleagues at the Defence Evaluation and Research Agency's labs at Porton Down in Wiltshire. Tattersall exposed slices of rat brain to microwave radiation. He found that it blunted their electrical activity and weakened their responses to stimulation. Because the brain slices were taken from the hippocampus, a structure with a role in learning, the results were seized upon as further evidence that mobile phones could scramble human memories."

But according to later research these effects were artificial, "may be explained by localised heating produced by interaction of the RF fields with the recording and stimulating electrodes".

Tattersall was involved in the new guidelines that were published in 2020.

Possible conflicts of interest

For IEEE/ICES see Van Rongen and others.

Tim Toivo

Biography

Tim Toivo works as senior inspector for the Radiation and Nuclear Safety Authority STUK in Helsinki, Finland. He is mainly involved in regulatory, research and expert work in the area of safety issues of electromagnetic fields (EMF) and ultrasound.

He studied biomedical engineering at Tampere University of Technology 1996. And started his work at STUK–Radiation and Nuclear Safety Authority in 1998 as a scientist in the unit of non-ionizing radiation.

Part of his work is to inform users of EM fields and communicate with the general public about safety issues. He participated in the preparation of the EU directive (EU 2013/35/EU) as an expert for the Finnish delegation.

He is a member of the ICNIRP Scientific Expert Group (SEG) in February 2017.

Position

Toivo was quoted in the book 'Behind the Screen: Nokia's success story in an industry of navel-gazing executives and crazy frogs': "It is fairly easy to prove that something is hazardous, but it is extremely difficult to prove that something is totally safe under all circumstances. It may take 20-30 years before any meaningful results are available from people who have been exposed to low power radiation."

In 2009 STUK published a position that 'children's mobile phone use should be limited.

A publication in 2006 – 'Epidemiological risk assessment of mobile phones and cancer: Where can we improve?' - together with Anssi Auvinen, concluded that "the major opportunity to improve the quality of evidence is, however, through prospective studies. The major limitation of epidemiological studies addressing the health effects of mobile phone use is related to exposure assessment. These limitations are inherent in case–control studies."

A 2008, in Vitro study of Pulsed 900MHz GSM Radiation on human Spermatozoa showed no effect.

In a 2009 publication – 'Specific absorption rate and electric field measurements In the near field of six mobile phone base station antennas' - Toivo and colleagues seem to suggest that the ICNIRP safety standards are very conservative: "It was also shown that the ICNIRP basic restriction for local exposure could be exceeded before the basic restriction for whole-body exposure if the distance to the antenna is less than 240mm."

With several ICNIRP colleagues he published the 'Progress report: ICNIRP Statement on non-ionizing radiation for cosmetic purposes' for the IEEE. They concluded that "'for cosmetic devices using radiofrequency EMF and optical radiation, there is the potential that occupational exposure limits can be exceeded if adequate protection measures are not applied."

Possible conflicts of interest

Hid DOI states that he gets funds from ministries which go directly to the Radiation and Nuclear Safety Authority STUK.

Andrew Wood

Biography

Wood is Professor in Bioelectromagnetic Research Group at Swinburne University of Technology in Melbourne. He also is a Chief Investigator with the new Australian Centre for Electromagnetic Bioeffects Research (a centre to which Rodney Croft and Sarah Loughran are also connected).

Wood used to work at Telstra Research Labs and is now a leading researcher at Swinburne Radiofrequency Dosimetry Laboratory, which is a part of the Bioelectomagnetic Research

Group. Telstra is Australia's largest telecommunications company. Swinburne university and in particular the Radiofrequency Dosimetry Laboratory has close relationship with and is co-funded by Telstra, the biggest Telecom company in Australia.

The close working relationship between the Swinburne University and Telstra is not new, as Don Maisch pointed out: "In fact the Chancellor of Swinburne University, Mr. Bill Scales (2005-2014) was previously Telstra's Group Managing Director, Regulatory, Corporate and Human Relations, and Chief of Staff at Telstra. He was also Telstra's Director of IBM Global Services Australia Ltd. and a Director of the Telstra Foundation."

Wood was a member of the Radiation Health Committee of the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) for over ten years.

He is a member of the Scientific Expert Group since March 2013.

Position

Wood does not see dangers of 5G and warns for being too cautious: "Wireless technologies bring enormous benefits, and being over-cautious would potentially deny these benefits to needy communities."

In a recent article he stated that studies which show health effects have a poor quality: "There are some comprehensive reviews of these, demonstrating that the quality of the studies is very variable, and that, for example, results claiming to show increased genetic damage or other biological effects are much more common in studies of low quality, whereas higher-quality studies predominantly show no significant effects."

Possible conflicts of interest

In a 2016 publication that gave an overview of the work Wood's group performed he and his co-authors wrote: "Over its 25-year history the Bioelectromagnetics Group has received support from national competitive grants and from industry research support schemes. It has been a node for both the Australian Centre for Radiofrequency Bioeffects Research (ACRBR) and the Australian Centre for Electromagnetic Bioeffects Research (ACEBR—see article in this edition). It has benefitted from industry collaboration and with national regulatory authorities."

The close collaboration with industry we see time and again. Just like the actual chair of the ICNIRP-commission Croft, Wood had actively collaborated with McKenzie, who is a manager at the Mobile Carriers Forum (MCF). See for more information the portrait of Croft.

In 2016, he published an article together with an employee of telecommunications company Telstra.

He has done contract work on the issue of smart meters for the private company EMC Technologies Pty Ltd.

According to his Declaration of Personal Interest he receives research support "from two engineers employed by Telstra Corp and one by the Australian Mobile Telecommunications Association."

Tongning Wu

Biography

According to ICNIRP's website Tongning Wu is a senior engineer in the Chinese Academy of Information and Communications Technology. His research focusses on electromagnetic dosimetry, anatomical modelling and biomedical applications of electromagnetic fields.

He is the member of International Advisory Committee (IAC) on Electromagnetic Fields of WHO. He also participated in the IEC/IEEE workgroups on EMF safety. He is currently the co-rapporteur of ITU-D Q7/2 (Strategies and policies concerning human exposure to electromagnetic fields).

He became a member of the ICNIRP Scientific Expert Group (SEG) in 2019.

Position

Wu agrees with the general ICNIRP assessment that "to date, no adverse health effects of the EMF, linked to these applications, have been established." This was also one of the conclusions of a study 'Electromagnetic fields (EMF) exposure' published in 2019.

In 2012 WU published a study on 'A large-scale measurement of electromagnetic fields near GSM base stations in Guangxi, China for risk communication'. The results were that "in general, the measurement mission promotes the science on EMF exposure among the general public. Risk-related public behaviours have been positively influenced. The mission also facilitates the cooperative conflict resolution. It helps strengthen the effectiveness of risk communication."

Possible conflicts of interest

His DOI gives no information.

See Van Rongen and others on the role of IEEE/ICES.

Annex I

Questions put to ICNIRP's secretariat

1 - When will the ICNIRP Annual report 2019 be published?

2 - Are the 14 members of the Commission being paid for their work for ICNIRP (for "representing ICNIRP externally and mostly in its relations with the international and national partners and the press" as well as for their collaboration on specific Projects?)

3 - Same questions as n° 2 go for the Scientific Expert group and the Project Groups?

4 - If they are not paid, do you consider this as a normal practice that international renowned experts work for free, especially given the importance and influence of the work of ICNIRP?

5 - ICNIRP itself claims it is "free of vested interests". ICNIRP's budget relies on support granted by public bodies; Why is the income not specified in your annual reports? Is it possible to get specifications from which public bodies you get which amounts?

6 Who selects the 14 members of the Commission and how?

7 - ICNIRP's statutes state: 'No member of the Commission shall hold a position of employment that in the opinion of the Commission will compromise its scientific independence'

Do we understand it correctly that basically the Commission evaluates itself about possible conflicts of interest? What are the rules by which the Commission judges if interests of the members compromise the scientific independence?

8- In its statement on the declarations of interests ICNIRP states: "The evaluation of personal integrity is very complex and might never be achievable in a perfect way. It is the duty of the ICNIRP Commission to carefully consider and decide if the declared interests potentially constitute a conflict of interest."

By which criteria or protocol are these considerations and decisions being made?

9- Do you consider the membership of IEEE ICES by some ICNIRP-members as a possible conflict of interests?

10- How do you explain the fact that a private organisation like ICNIRP, which is not accountable in democratic terms to anyone, has the position to de facto "determine" via guidelines the EMF policies of most EU member states?

Several attempts to get a reaction to

these questions remained unanswered'

Annex II

Questions put to emfproject@who.int

On your website, you write: "Because disparities in EMF standards around the world has caused increasing public anxiety about EMF exposures from the introduction of new technologies, WHO commenced a process of harmonization of electromagnetic fields (EMF) standards worldwide. With 54 participating countries and 8 international organizations involved in the International EMF Project, it provides a unique opportunity to bring countries together to develop a framework for harmonization of EMF standards and to encourage the development of exposure limits and other control measures that provide the same level of health protection to all people. "

1 - Is there a time schedule for this process of harmonization of electromagnetic fields (EMF) standards worldwide?

2 - We see on your website that the last EMF -WHO meeting took place in 2018. Are there any new meeting planned and if yes when?

3 - Do you know what IARC is currently working on and if so when will IARC publish an update of the monograph?

https://publications.iarc.fr/Book-And-Report-Series/Iarc-Monographs-On-The-Identification-Of-Carcinogenic-Hazards-To-Humans/Non-ionizing-Radiation-Part-2-Radiofrequency-Electromagnetic-Fields-2013

4 - How do you consider the debate on "conflicts of interests" in this specific research area? Would you agree that there has been and still is a lot of attention for this debate? Has his debate been useful in narrowing the divide in the scientific community? What is in your view the role of the WHO on this?

(see for example this recent letter published in "Bioelectromagnetics": https://onlinelibrary.wiley.com/doi/full/10.1002/bem.22225)

These questions remained unanswered

The International Commission on Non-Ionizing Radiation Protection: Conflicts of interest, corporate capture and the push for 5G

June 2020

www.michele-rivasi.eu

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SUNDHEDSSTYRELSEN

Habilitetserklæring

Opgavetype	
Jeg skal bistå Sundhedsstyrelsen med/i?	Afgørelser, råd, undervisning mv
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Mit speciale	Ikke ioniserende stråling

Personoplysninger	
Navn	Christoffer Johansen
Titel	Professor, overlæge
Arbejdsplads/firma/institution	Onkologisk Klinik, Rigshospitalet
E-mail	christof@cancer.dk

Oplysninger om personlige interesser m.m.	
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Spørgsmål 2.6 Er en person i din nærmeste familie ansat, medejer eller på anden måde tilknyttet virksomheder eller institutioner, der er underlagt Sundhedsstyrelsens myndighedsudøvelse.	Nej
Spørgsmål 2.7 Modtager din arbejdsplads økonomiske bidrag fra virksomheder eller institutioner, der er underlagt Sundhedsstyrelsens myndighedsudøvelse?	Nej
Spørgsmål 2.8 Har du andre tilknytninger eller omstændigheder, der kan være relevante for din habilitet?	Ja

Oplysninger om personlige interesser m.m.

Spørgsmål 2.8 - Hvilke:	Jeg har gennem de sidste 20 år (1999, 2006 og 2010) udført en gennemgang og vurdering af videnskabelig litteratur, der belyser sammenhængen mellem eksponering for elektromagnetiske (EMF) felter og helbreds effekter, for det rådgivende ingeniørfirma COWI. Disse rapporter har jeg udført, når COWI har rådgivet de firmaer, der står for etablering af udvidelser eller vedligehold af de netværk der distribuerer elektricitet i Danmark gennem nye højspændingsledninger, transformer stationer og lignende. Rapporterne er udformet som en gennemgang af den videnskabelige litteratur med fokus på undersøgelsernes kvalitet, fortolkningen af resultaterne og betydning ud fra det evidens niveau, der kan fortolkes på baggrund af de konkrete videnskabelige artikel. Der indgår ingen samfundsøkonomske, planlægningsmæssige eller andre vurderinger i de rapporter jeg har skrevet - det er udelukkende den videnskabelige kvalitet og dermed resultaternes indflydelse på den evidens der er for en sammenhæng mellem eksponering for EMF og helbreds problemer. Rapporterne er offentlig tilgængelige og indgår i det materiale som berørte borgere kan få udleveret i forbindelse med anlæggelse af de ovenfor anførte anlæg. Jeg har i efteråret 2017 indgået en kontrakt med COWI om en opdatering af denne rapport og i den forbindelse igen orienteret Sundhedsstyrelsens Direktion om kontrakten.

Underskriv din erklæring

Jeg har efter min bedste overbevisning ingen yderligere uvedkommende interesser, som kan påvirke mit objektive arbejde for Sundhedsstyrelsen. Hvis der sker ændringer udfylder jeg straks en ny erklæring, hvoraf ændringerne fremgår.	Valgt
Udfyldt den	30-08-2017

ESC European Society of Cardiology

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REVIEW

Electromagnetic interference in cardiac electronic implants caused by novel electrical appliances emitting electromagnetic fields in the intermediate frequency range: a systematic review

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Electromagnetic fields (EMF) in the intermediate frequency (IF) range are generated by many novel electrical appliances, including electric vehicles, radiofrequency identification systems, induction hobs, or energy supply systems, such as wireless charging systems. The aim of this systematic review is to evaluate whether cardiovascular implantable electronic devices (CIEDs) are susceptible to electromagnetic interference (EMI) in the IF range (1 kHz–1 MHz). Additionally, we discuss the advantages and disadvantages of the different types of studies used to investigate EMI. Using the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement, we collected and evaluated studies examining EMI in *in vivo* studies, *in vitro* studies (phantom studies, benchmark tests), and simulation studies. Our analysis revealed that cardiac implants are susceptible to malfunction induced by EMF in the IF range. Electromagnetic interference may in particular be provoked by security systems and induction hobs. The results of the studies evaluated in this systematic review further indicate that the likelihood for EMI is dependent on exposure-related parameters (field strength, frequency, and modulation) and on implant- as well as on lead-related parameters (model, type of implant, implant sensitivity setting, lead configuration, and implantation site). The review shows that the factors influencing EMI are not sufficiently characterized and EMF limit values for CIED patients cannot be derived yet. Future studies should therefore, consider exposure-related parameters as well as implant- and lead-related parameters systematically. Additionally, worst-case scenarios should be considered in all study types where possible.

Keywords Electromagnetic interference • Implantable cardioverter-defibrillator • Cardiac pacemaker • Electric fields • Magnetic fields • Intermediate frequency • Systematic review

Introduction

In recent years, the number of patients that have been fitted with cardiovascular implantable electronic devices (CIEDs) such as cardiac pacemakers (PMs) or implantable cardioverter-defibrillators (ICDs) has strongly increased. In the USA, while 9000 CIEDs were implanted in 1990,[1] its number increased to 368 829 in 2009.[2] Over 4.2 million primary CIED implantations were performed between 1993 and 2008.[3] In Europe, 547 586 PMs and 105 730 ICDs were implanted in 2016.[4] Additionally, novel CIEDs like leadless PMs, subcutaneous ICDs, and heart failure devices are gaining more and more importance.

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At the same time, with the success of the CIED technology during the past decades, exposure to external electric, magnetic, and electromagnetic fields (EMF) has increased, at least in the intermediate frequency (IF) and radiofrequency (RF) range.[5–8] Electromagnetic fields are used e.g. to transmit communication signals or arise along power transmission lines. Other sources of EMF are electrical appliances. Electromagnetic fields are classified according to their wavelength and frequency. For example, power lines or electrical household devices emit EMF with a lower frequency (LF) while mobile phones, Wi-Fi, or microwave ovens produce EMF of a higher frequency. Electromagnetic fields in the IF range are generated by many novel electrical appliances, including electric vehicles, RFID (RF identification) systems, induction hobs, or energy supply systems, such as wireless charging systems.

Cardiovascular implantable electronic devices are known to be susceptible to malfunction in the presence of strong EMF.[9–12] Many researchers have studied electromagnetic interference (EMI), i.e. potential, undesirable effects of EMF on the operation of CIEDs. The EMF-Portal (www.emf-portal.org), the most comprehensive scientific literature database on biological and health-related effects of EMF provided by our institute currently comprises 639 records on EMI (June 2017). The Manufacturer and User Facility Device Experience (MAUDE) database of the American Food and Drug Administration[13] identified 2843 cases of malfunctions of medical devices induced by EMI between January 2010 and March 2017. However, this may be an underestimation of events as reporting of such incidents is not mandatory and some physicians may misjudge EMI episodes e.g. as atrial fibrillation. A survey of physicians in France showed that 16% of them were concerned about patients who reported EMI at least once a year, e.g. oversensing of noise signals due to EMF exposure is a phenomenon regularly seen in daily practice.[14] Napp *et al.*[9] demonstrated the general mechanisms of effects in CIED caused by EMF, e.g. heating of the implant or lead by RF fields or induction of electric currents within the human body by LF fields leading to e.g. disturbance of the sensing capabilities of the implant. Additionally, Beinart and Nazarian[10] showed potential everyday sources of EMI and documented typical effects, e.g. damage to CIED circuitry, PM inhibition, asynchronous pacing, or inappropriate ICD shocks.

Standard organizations have not proposed limit values to EMF exposure for patients with CIEDs. The American National Standards Institute (ANSI),[15,16] the International Commission on Non-Ionizing Radiation Protection (ICNIRP),[17] and the European Union[18,19] did not consider patients with CIEDs in their safety guidelines for the protection of humans exposed to EMF. Consequently, it is often difficult for physicians and patients to identify sources of EMF which pose a risk and to determine appropriate safety distances which should be respected. In some cases, the applied safety measures in occupational environments might result in a ban from workplaces for CIED carriers.

To date, no systematic analysis has been done for EMI in the IF range (1 kHz–1 MHz). The aim of this systematic review is therefore to evaluate whether CIEDs are susceptible to EMI in the IF range. In particular, we consider the results from different types of studies (*in vivo* and phantom studies, benchmark test) and outline their advantages and disadvantages. Additionally, we identify the type of study which is most appropriate to further investigate the various parameters (implant setting, lead configuration, and individual parameters) that influence the likelihood for EMI.

Methods

Literature search strategy and general information

As prescribed by the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) statement,[20] we conducted a systematic literature search to identify relevant studies published from inception to October 2016 using our thematically specialized open-access literature database EMF-Portal (www.emf-portal.org). The EMF-Portal is the most comprehensive scientific literature database on biological and health-related effects of EMF and has been approved by the WHO as a reference database.[21] It has been publicly available for more than 15 years and comprises currently 25 900 publications[22] (January 2018). Our search in the EMF-Portal for the current systematic review was based in a first step on the more general search term 'electromagnetic interference' (for a link to the search string, see Supplementary material online: search strategy). Additionally, we performed a more specific search in the frequency range <10 MHz and in the category 'electromagnetic interference'. The lists of results were corrected for double publications.

Eligibility criteria and study selection

Articles were included when they reported experimental studies on EMI with CIED in the frequency range of 1 kHz–1 MHz. We accepted benchmark tests, studies with phantoms, *in vivo* studies, and numerical simulations. Only articles written in English or German and published in a peer-reviewed journal were considered. There was no restriction regarding the year of publication.

Excluded were studies that focused on CIED-programmer interference, CIED-interference with further (cardiac) implant, EMI with other implants (e.g. neurostimulator), or EMI induced by current application (e.g. by medical devices). Furthermore, studies without specification of the tested frequency range were excluded. Review articles, case studies, editorials, commentaries, and unpublished or clearly not peer-reviewed articles were also excluded.

Two authors independently (S.D. and D.S.) screened the studies for eligibility based on inclusion/exclusion criteria. Articles were screened in two stages. First, titles and abstracts were reviewed to identify potentially relevant articles. For those abstracts which met the inclusion criteria, the full text was retrieved and independently reviewed in the second stage of assessment. The two review authors made a joint decision about inclusion of the articles.

Data extraction

The data from the studies included were extracted independently by two authors (S.D. and D.S.). The extraction protocol was defined and agreed upon before the start of the project. Extracted data included bibliographical data, study type (e.g. phantom, *in vivo* study), exposure parameters (field source, i.e. electrical appliance, frequency, and field strength if provided), number of patients/CIED, CIED characteristics, and outcome (disturbance). Additionally, for *in vivo* studies, implant settings and lead polarities were extracted. Disagreements and uncertainties were discussed and resolved between the two review authors.

Results

The systematic literature search identified 389 articles that matched the search criteria. After screening the title and abstract, 203 articles were excluded for various reasons (e.g. secondary literature, not dealing with EMI). The full text was obtained for the remaining 186

articles to check for eligibility to be included in our analysis. Of these, 146 articles were excluded for the following reasons: frequency range not provided or appropriate ($n = 48$), current application ($n = 35$), case report ($n = 21$), CIED-implant interference ($n = 15$), no CIED ($n = 10$), CIED-programmer interference ($n = 4$), or other reasons ($n = 13$). Forty articles fulfilled the eligibility criteria and were included in this review (see *Figure 1*). Of these, most studies ($n = 15$) used combinations of several methods to investigate EMI (e.g. phantom and benchmark), 13 studies used phantoms only, 10 articles investigated EMI in patients (*in vivo*), and one study used a benchmark test only. Additionally, the search identified one simulation study (see *Figure 2*).

Most of the studies investigated EMI on PMs only ($n = 20$), while four studies considered ICDs only. Ten studies considered different types of CIEDs [PMs, ICDs, and implantable loop recorder (ILR)]. Five studies did not use a CIED but a modified CIED case to measure the induced voltage at its terminals. One study investigated EMI on ILRs only.[23]

In some studies, additional frequency ranges or electrical appliances outside the IF range were investigated, but these data on EMI are not considered in this review—if not stated otherwise.

Table 1 provides detailed information on the most important technical terms which are used in this review.

In vivo studies

In *in vivo* studies, patients with CIEDs are directly exposed to EMF to assess the electromagnetic compatibility of CIEDs. As such, individual interference thresholds of the CIED can be determined for specific exposure conditions.

In the current review, 10 *in vivo* studies were evaluated which exposed patients with CIEDs to EMF (Supplementary material online, *Table S1*). Additional five studies used a combined methods approach, i.e. they conducted also benchmark or phantom tests (Supplementary material online, *Table S3*).

Altogether, potential EMI was investigated in 1084 patients that had been fitted with CIEDs (769 PMs, 313 ICDs, and 2 ILRs). The nine studies providing details on PM and ICD types included 369 single chamber (217 PM, 152 ICD), 433 dual chamber (361 PM, 72 ICD), and 42 resynchronization therapy devices (13 PM, 29 ICD). Eight of the 15 studies included CIED carriers with both unipolar and bipolar leads, whereas three studies[24–26] tested only patients with one lead configuration (unipolar OR bipolar). Four studies did not provide any details on lead configuration.[27–30] Eleven of the 15 studies left the CIED sensitivity unchanged or investigated different sensitivities settings (e.g. maximum, nominal), whereas two studies[29,31] tested under maximum sensitivity only. Two studies did not provide any details on sensitivity.[28,32]

All included studies used real-life electrical appliance exposure such as security systems [electronic article surveillance (EAS) systems or metal detector gates, $n = 8$], medical devices ($n = 4$), induction hobs ($n = 3$), or avalanche transceivers ($n = 1$). One study tested both an EAS system and an induction hob (counted separately in each category).[31] None of the included *in vivo* studies were performed under a standardized exposure set-up, i.e. with e.g. a Helmholtz coil. Furthermore, not all of the studies provided details on the field strengths which actually occurred at the height of the implant (chest area). At least, one study measured the magnetic field strength at a distance from the patient to the security device (1.6–2.7 A/m, 50 cm to the security gates)[33] and another study performed comprehensive field measurements (14–310 A/m), but the field strength required to induce EMI remained unclear.[34]

The data of three studies showed that the acoustomagnetic EAS system (58 kHz) could disturb PMs.[31,32,34] There was no evidence that ICDs could be disturbed. However, only 38 ICD patients were included in these studies compared with 265 PM patients (whereof the PMs of 72 patients were disturbed). Electromagnetic interference with PMs was also found with other security systems operating at higher (120 kHz) or lower (10 kHz) frequencies.[33,35] Importantly, the study by Wilke *et al.*[33] provided evidence that PMs can be negatively affected by security systems below the ICNIRP limits for the general public (i.e. 21 A/m).

Two further studies did not observe EMI following exposure to metal detectors[36] or EAS systems[23]; with the latter being one of the two studies investigating ILRs. In a further study it remained unclear whether EMI was induced by a 100 Hz or 1 kHz metal detector.[24]

Three studies on potential EMI of induction hobs[25,26,31] showed that the patients' safety with CIEDs was guaranteed when minimum distances were respected, i.e. no EMI was observed at distances of 20–35 cm. It was, however, not documented in these studies whether disturbances occurred in closer proximity to the induction hobs.

Other devices such as an electromagnetic articulography device,[29] an ultrasonic dental scaler,[30] a magnetic endoscope imager,[27] or avalanche transceivers[37] did not cause EMI under the used conditions. Some dental devices appeared to have the potential to disturb PMs,[28] however, the frequency was provided only for one device and it is unclear whether the other dental devices emitted EMF in the IF range. The relevance of this data is, however, debatable for today's applications, because the study[28] was published in 1975.

Altogether, EMI in the IF range was revealed in 6 out of the 15 studies resulting in e.g. sensing anomalies (e.g. undersensing or oversensing),[31–34] asynchronous pacing,[33,34] increased pacing rate,[24,34] pacing inhibition,[24,32–35] and mode switch.[32] In McIvor *et al.*[34] EMI was accompanied by symptoms in patients, e.g. palpitations and presyncope.

For a general risk assessment, *in vivo* studies with exposures to a single device, such as avalanche transceivers[37] have only a limited significance due to the lack of a proper dosimetry. Additionally, the applicability of the data to other exposure situations is limited. Studies performed under standardized exposure set-ups, i.e. using e.g. a Helmholtz coil or antenna settings are better suited, because EMF can be homogenously generated and EMF at different frequencies and field strengths can be applied systematically. That way, more general data for various applications can be obtained.

Phantom studies

Phantoms simulate the human body or parts of the human body including different tissue characteristics.[38] Experimental *in vitro* studies using phantoms examine either directly the disturbance (EMI) of CIEDs or they are used to determine the intracorporal voltage induced by external EMF at the terminals of CIEDs. In phantoms, both the response of CIEDs to different EMF and the impact of the lead can be tested.

Potential EMI with CIEDs was investigated in 15 studies and five studies measured the induced voltage. An additional five studies

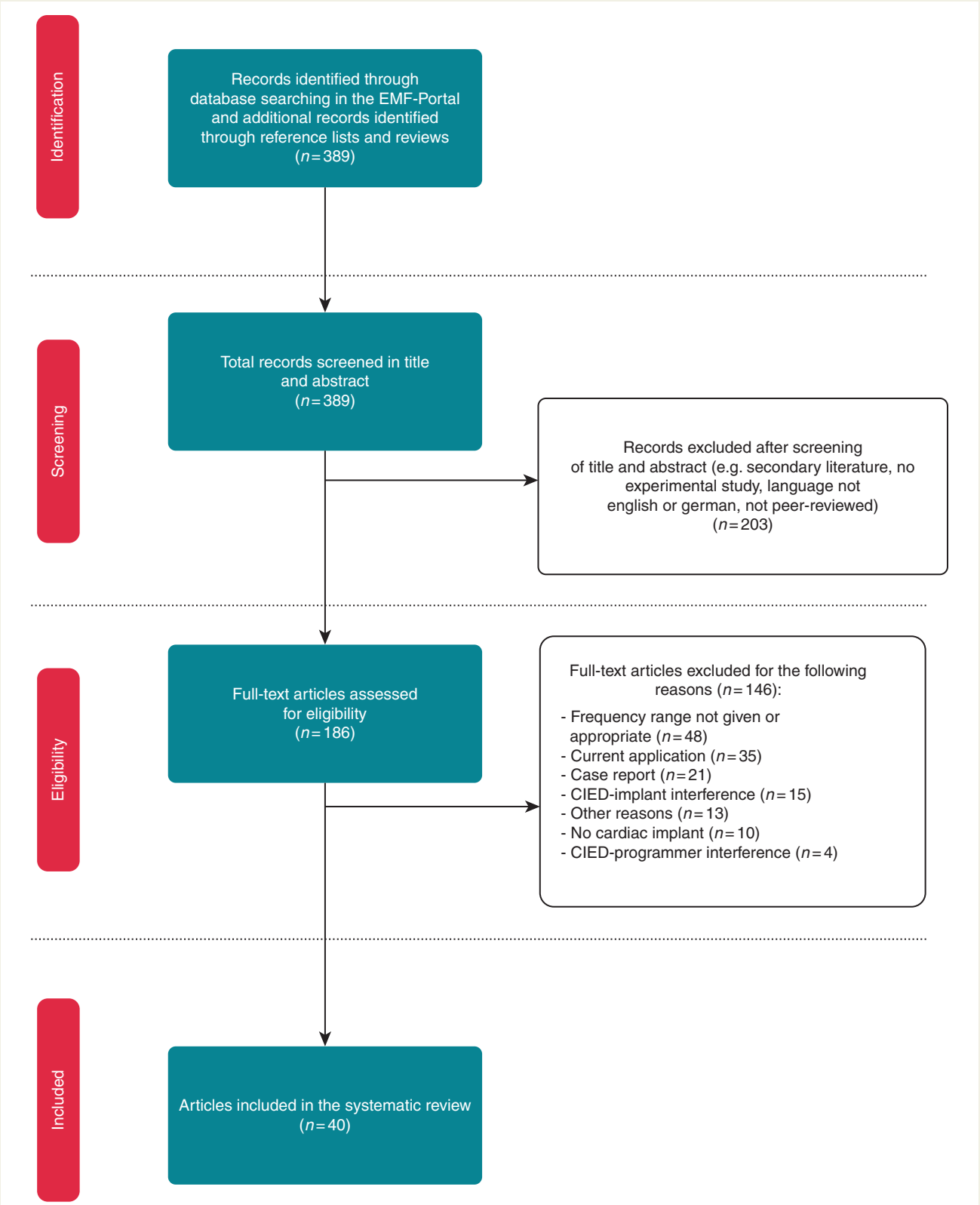

Figure 1 Flow diagram of literature search, eligibility, and inclusion process. Adapted from Moher *et al.*[20] CIED, cardiovascular implantable electronic device; EMF, electromagnetic field.

considered the development of a coupling model (Supplementary material online, *Tables S2* and *S3*). In Babouri *et al.*,[39] the phantom served to validate detection levels recorded in benchmark tests. Therefore, this study is discussed in the 'Benchmark tests and test *in air*' section.

Studies investigating electromagnetic interference

In the 15 studies on potential EMI, altogether, 185 CIEDs were investigated [100 PMs, 60 ICDs, and 25 PM/ICDs (not further specified[40])]. Only 4 out of the 15 studies used a standardized exposure set-up, e.g. a Helmholtz coil setting,[41–44] whereas the remaining 11 studies investigated potential EMI in phantoms upon exposure to real-life electrical appliances [RFID/security systems ($n = 5$), induction hobs ($n = 2$), medical devices ($n = 2$), wireless power transfer (WPT) systems ($n = 1$), or a magnetically levitated linear motor car ($n = 1$)]. Only one study provided precise data on the correlation of EMI and exposure characteristics.[45]

The data of two studies[46,47] on RFID systems showed that the majority of the investigated PMs (67–83%) and ICDs (47–71%) could be disturbed by different 134 kHz RFID systems at a distance of up to 61.3 cm. There was no clear correlation between EMI and lead configuration and no difference between maximum and nominal sensitivity,[46] most likely due to the high intensity field strength of the RFID system. Mattei *et al.*[45] investigated typical exposure patterns of RFID systems and identified EMI from 40 A/m at 125 kHz for a pulsed signal and from 60 A/m for a continuous wave (CW) signal.

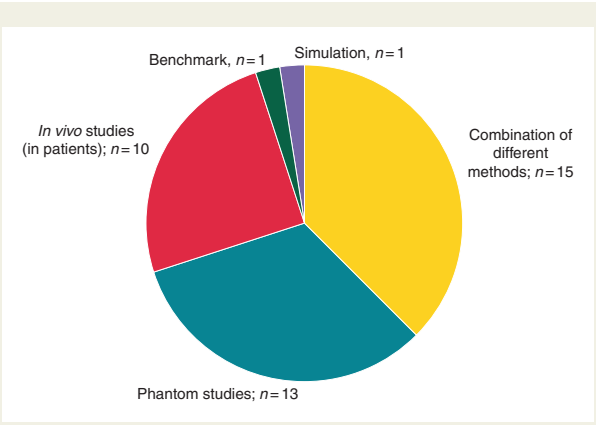

Figure 2 Study types used for EMI investigation in the IF range. EMI, electromagnetic interference; IF, intermediate frequency.

Two further studies on security systems showed that EMI was induced by an anti-theft device of 120 kHz[33] and by different EAS signals (100 Hz–8 kHz, CW or pulsed[48]). Kainz *et al.*[48] found that the interference level of a pulsed signal was lower than that of a CW.

Two studies on induction hobs[25,49] revealed that EMI occurred both as a function of the distance to the induction hob and dependent on the presence/absence of the pot or the position of the pot. However, the exact field strengths at specific distances were not clear from both studies.

The data on security systems or induction hobs showed that some devices in our everyday life may induce EMI in CIEDs and thus confirm the findings of *in vivo* studies.

Hikage *et al.*[40] investigated 14 different WPT systems. Electromagnetic interference occurred in 5 of the 12 WPT systems for mobile application with modulated fields at a maximum distance of ≤2 cm for PMs and at a distance of ≤1 cm for ICDs. The two WPT systems for electric vehicle charging provoked no EMI. However, no field strengths were provided and no details were shown which WPT system caused which kind of EMI.

No EMI was induced under the specific study conditions for a microtron device used for cancer therapy,[50] nor for an electromagnetic navigational bronchoscopy device[51] or for a magnetically levitated linear motor car[52].

The four phantom studies on potential EMI using a standardized exposure set-up[41–44] found that EMI depended on field frequency, CIED type, and programmed sensitivity.

For a general risk assessment, the data of the evaluated phantom studies on potential EMIs are limited as are the data of the discussed *in vivo* studies. Although there are many studies using a comprehensive study design, the applicability of the data to other electrical appliances or comparable exposure scenarios is limited due to the lack of sufficient dosimetric data or a missing correlation of those data to

Table 1 Technical terms used in this review

Terms	Explanation
EMI	Disturbance of CIEDs' operation by induction of intracorporal voltage caused by electric, magnetic, or EMF
External EMF	EMF emitted by an electrical appliance, e.g. magnetic field (measured in T or A/m) or electric field (measured in V/m)
Induced voltage	Intracorporal voltage occurring at the terminals of a CIED induced by external EMF
Interference thresholds	Minimum field strength of an external EMF required to cause EMI
Disturbance/interfering signal	Noise signal that may disturb the regular operation of a CIED
CW, AM, PW, and pulses	Waveforms (CW, AM, PW, and pulses) of an external EMF or a disturbing/interfering signal; the waveform can significantly influence the response of a CIED
Detection level	Voltage of a disturbing/interfering signal which causes disturbance of CIEDs' operation
Performance limits	Minimum detection levels of CIEDs defined in product standards, given in mV

A/m, Ampere/meter; AM, amplitude modulation; CIED, cardiovascular implantable electronic device; CW, continuous wave; EMF, electromagnetic fields; EMI, electromagnetic interference; mV, millivolts; PW, pulsed modulation; T, Tesla; V/m, Volt/meter.

EMI.[25,33,49] Likewise, phantom investigations with exposures to only a single device, such as a magnetically levitated linear motor car[52] only contribute in a limited way to a general risk assessment. The same applies to studies using standardized conditions if EMI is not systematically tested for various frequencies and field strengths.

Determination of induced voltage

Besides the direct measurement of EMI, phantom studies can also serve to determine the voltage induced by external EMF at the terminals of CIEDs. The induced voltage is the critical measurement parameter for electromagnetic compatibility testing of CIEDs and can be compared with international product standards, e.g. Ref.[53] These CIED standards set performance limits up to 3 GHz with the objective of preventing malfunctions induced by EMF. The performance limits increase linearly in the IF range (3 kHz–167 kHz) and vary between unipolar (9–500 mV) and bipolar (0.9–50 mV) testing.[53–55]

The induced voltage was investigated in five studies.

Bassen[56] investigated different iPods but the induced voltage was below the noise level of their measurement instruments. They concluded that no EMI would be expected.

Irnich and Bernstein[57] investigated 11 induction hobs and the induced voltage was between 6 and 800 mV dependent on the distance and the position of the pot. The combination of their phantom study with a benchmark test showed that 14.8% of PMs would be disturbed under worst-case conditions and never at a minimum distance of 35 cm to the thorax. These results confirm the findings of phantom studies on EMI[25,49] and the findings of *in vivo* studies[25,26,31] in that induction hobs appear to be safe at a specific distance.

Seckler *et al.*[58] investigated a WPT system and compared the data with a standardized exposure set-up using Helmholtz coils (111 kHz, both systems). Under the standardized exposure condition the performance limit (i.e. 333 mV for CIEDs with unipolar leads and 33.3 mV for bipolar leads) was already exceeded at 11 μT and thus below the ICNIRP limit[17] of 27 μT; whereas with the WPT system, the limits—even with the WPT system touching the phantom—were not exceeded. This comprehensive study approach demonstrates the significant difference between the voltage induced by a homogenous field of Helmholtz coils and by inhomogenous fields of an electronic device. Moreover, this study highlights the importance to characterize the emitted field patterns (e.g. CW and AM) and dosimetric data of electronic devices.

Mattei *et al.*[59] used an antenna design at 125 kHz to emulate RFID systems and measured a maximum induced voltage for a unipolar lead of 62.2 mV and 19.8 mV for a bipolar lead, thus indicating that the performance limits (i.e. 375 mV for CIEDs with unipolar and 37.5 mV for bipolar leads) were not exceeded under the specific exposure conditions. In a later study by the same authors,[45] however, EMI was detected and thus, the performance limits seemed to be exceeded by exposure of RFID systems (see section 'phantom studies—Studies investigating electromagnetic interference'). Exceeding of the performance levels under consideration of EAS-similar exposure scenarios was also demonstrated by numerical simulations of Leitgeb *et al.*[60] who calculated induced voltages between 3.2- and 13.5-fold above the performance limits in the IF range (60 kHz–5 kHz, respectively) under worst-case conditions (Supplementary material online, *Table S5*).

Gustrau *et al.*[61] identified induced voltages at the PM terminals of 0.126 mV–131 mV (1 kHz–1 MHz, at 1 A/m, i.e. 1.26 μT). Gustrau *et al.*,[61] Seckler *et al.*,[58] and Mattei *et al.*[59] found a dependence of the induced voltage on lead configuration.

Coupling model

A further motivation to perform phantom studies, is the development of a coupling model (transfer function). Transfer functions demonstrate the relationship between the strength of an external EMF and the induced intracorporal voltage at the terminals of a CIED. The transfer functions of the five studies included were determined by numerical or analytical approaches based on data gained in phantom studies or benchmark tests.

Hedjiedj *et al.*[62] developed a transfer function based on a simple phantom and found detection levels from >55/104 mV at 10/25 kHz for a sensitivity level of 0.7 mV. The authors also included benchmark tests and reported detection levels in two out of five PMs of >150/130 mV at 10/25 kHz for a sensitivity level of 1 mV.

In two comprehensive studies, Andretzko *et al.* presented a numerical model for the determination of transfer functions between electric fields[63] or magnetic fields[64] and the induced voltage. The results obtained by numerical simulation were in agreement with experimental data from benchmark tests and phantom studies. The detection levels increased with increasing PM sensitivity values; additionally, the interference thresholds depended on the loop area formed by the CIED with its lead, i.e. for a large (300 cm^2) loop area interference threshold occurred from 20 μT and for a standard loop area (225 cm^2) from 26 μT (unipolar lead).

In a further study by the same research group,[43] the realistic lowest interference thresholds were calculated and given with 33.36 μT (25 kHz) or 79.18 μT (10 kHz), respectively, for unipolar lead settings (200 cm^2). However, the coupling model was not validated and tests *in air* and tests with a phantom yielded different results. The result of this study together with the results of the other three studies suggest that the detection levels of the considered CIEDs were significantly above the performance limits recommended by international product standards,[53] thus indicating compliance with the proposed standards, although the applied magnetic fields (30–85.4 μT) exceeded the limit value recommended by ICNIRP (i.e. 27 μT).[43,64] Whether the transfer function presented by this research group could serve as a solid basis for the calculation of induced voltages from external EMF should be validated by comprehensive realistic data obtained from benchmark tests, phantom, and *in vivo* studies.

Finally, in a study by van Wijk van Brievingh *et al.*,[65] the authors calculated—according to their coupling model—interference thresholds between 0.1 and 100 A/m (i.e. 0.126–126 μT) for 100 Hz and 250 kHz. However, no specific values for induced voltages were provided.

In general, the data of these five studies indicate that interference thresholds depend on the loop area formed by the CIED with its lead, on the frequency of the applied external EMF, the sensitivity setting of the CIED and the device itself and thus confirm the findings of several other phantom studies. Universal transfer function therefore, may provide a helpful tool to estimate induced voltages under specific conditions (e.g. lead type) and in dependence of specific exposure scenarios (frequency and field strength).

Benchmark tests and tests *in air*

Manufactures of CIEDs are obligated to test their implants for compliance with product standards in order to obtain approval for the European market (CE marking) or from the Food and Drug Administration (FDA) for the US market. Regarding electromagnetic compatibility performance limits as well as test methodologies (benchmark tests) are defined in ISO 14117:2012[53] for the US market and in EN 45502-2-1:2004[55] (PMs) and EN 45502-2-2:2008[54] (ICD) for the European market. In benchmark tests disturbance signals are fed directly into the pace/sense channel of the CIED by galvanic coupling in order to analyse the CIED's response and detection levels. The simple methodology is a major advantage of benchmark tests. Cardiovascular implantable electronic devices of different manufacturers and with different settings can be tested and evaluated by any number of various disturbance signals. However, a disadvantage of benchmark tests is that individual parameters of the patient or the lead are not considered and the data cannot be transferred to external EMF of e.g. a certain electrical appliance.

Although benchmark tests are a well-accepted method, many researchers preferred to perform tests *in air*. In such tests, implants are equipped with leads and located within the exposure area of a standardized set-up or close to a specific electrical appliance, comparable to phantom studies but without a phantom. Thus, potential EMI can be correlated with field strengths of an external EMF. In the current review, benchmark tests were used in six studies, whereas tests *in air* were performed in seven studies (Supplementary material online, *Tables S3* and *S4*). Andretzko *et al.*[64] conducted both benchmark tests and tests *in air* (counted in each category separately). Altogether, in the 12 studies included interference was evaluated in >286 PMs (exact number not given in van Wijk van Brievingh *et al.*[65]), in one ICD and in three ILRs. The results of benchmark studies generally indicated compliance with the performance limits set by product standards. Additionally, the results of some benchmark tests and tests *in air* showed the dependence of potential EMI on frequency, the CIED and its sensitivity level.[39,64,66]

In 4 of the 12 studies included, benchmark tests or tests *in air* were combined with other methods in order to establish a transfer function.[62–65] These studies are discussed in detail in the 'Phantom Studies—Coupling Model' section. Nevertheless, due to the data of these studies it can be summarized that the lowest detection levels were found from 55 mV (10 kHz)[62] and 95 mV (25 kHz)[64] depending on the sensitivity level of the PM.[64] Comparable detection levels were also found in an earlier study by the same research group[39] combining benchmark tests and phantom investigations.

A further publication with tests *in air*[43] reported interference thresholds from 1.09 μT at 10 kHz (single-chamber PM) and from 0.54 μT at 25 kHz (dual-chamber PM); however, this was found under artificial loop conditions (90 turns-lead).

Irnich and Bernstein[57] performed benchmark tests in order to investigate the impact of a typical signal of induction hobs (24 kHz) and reported detection levels below the performance limits. It has to be noted, however, that the data was recorded for an older PM model which was released before 1998.

Three out of four studies performing tests *in air* on security systems found that EMI was caused by different EAS systems and EAS signals.[35,48,66] Only the study by de Cock *et al.*[23] did not report EMI. Potential EMI provoked by EAS systems was also found in *in vivo* studies e.g. Refs[31,32,34,35] and in phantom studies e.g. Refs[46,47]. The strengths of external EMF were only provided for EAS systems used in Dodinot *et al.*[35] and for signals used in Kainz *et al.*[48] where interference thresholds were reached from 1.13 mT and approximately 15 $A/m_{peak\text{-}to\text{-}peak}$, respectively. Additionally, the study by Lucas *et al.*[66] indicated that unipolar PMs were affected more often than bipolar PMs.

Finally, Corbett *et al.*[27] investigated a medical magnetic endoscope imager and did not find EMI with different CIEDs. The same result was obtained in their experiment with patients (see *In Vivo* Studies section).

Discussion

The aim of this systematic review was to evaluate whether CIEDs are susceptible to EMI in the IF range generated by many novel electrical appliances, including electric vehicles, induction hobs, or wireless charging systems.

Forty articles fulfilled the eligibility criteria and were included in this review. Most of the studies investigated EMI on PMs only ($n = 20$), while four studies considered ICDs only. Ten studies considered PMs, ICDs, and ILRs. Five studies did not use a CIED but a modified CIED case to measure the induced voltage at its terminals. One study investigated EMI on ILRs only.[23]

There is only limited data on EMI with CIEDs in the IF range and only a few of the evaluated studies correlated the documented EMI with exposure data (e.g. Refs[33,45,58]). Likewise, the studies that did not report EMI, rarely provided detailed data on exposure conditions.[26,29,31,41,42,51]

More than one-third of the studies investigated CIEDs which were exposed to security systems, including EAS, metal detectors, and RFID ($n = 15$), and five studies investigated potential EMI in the proximity of induction hobs. Single studies also investigated other electronic appliances, such as iPods,[56] a magnetically levitated linear motor car,[52] WPT systems,[40,58] or avalanche transceivers.[37]

There is evidence that EMF sources of everyday life such as security systems may induce EMI.[31–34,45,46] Also, induction hobs appear to provoke EMI in close proximity.[25,26,31,49] For other electronic appliances, EMI or exceeding of performance levels was found only for WPT systems.[40,58] However, it cannot be concluded that the other investigated electrical appliances that did not reveal any EMI adhere to safety standards in general, because some studies investigated only a few CIEDs or a few patients (7 ICD[51] or 3 PM, 1 ICD[52]) or the exposure parameters were insufficiently described.[37,56]

We evaluated benchmark tests, simulation, phantom, and *in vivo* studies. As shown in *Table 2*, the studies evaluated in this review used different methods (e.g. phantom and benchmark) to investigate EMI for various electrical appliances. For RFID/EAS systems, EMI was consistently reported in *in vivo* studies, phantom studies, and benchmark tests, whereas for induction hobs and wireless charging systems, EMI was found only in phantom studies.

Clinical relevance

Previous studies have shown that oversensing of noise signals due to EMF exposure occurs in everyday life and physicians caring for CIED patients are regularly confronted with EMI.[14,67,68] In the studies

Table 2 Types of studies used for EMI investigation in different electrical appliances

Electrical appliance	*In vivo* studies	Phantom studies	Benchmark tests and tests *in air*	Simulation
Standardized exposure set-up		X[39,41–44,58,61–65]	O[39,43,62–65]	O[43,61,63,64]
RFID/EAS systems	X[23,31–35]	X[33,45–48,59]	X[23,35,48,66]	O[60]
Metal detector	O[23,24,36]	O[48]	O[23,48]	
Induction hobs	O[25,26,31]	X[25,49,57]	O[57]	
Wireless charging systems		X[40,58]		O[40]
Different medical devices (articulography device, dental devices, microtron device, navigational bronchoscopy device, and magnetic endoscope imager)	O[28–30]	O[50,51]	O[27]	
Different iPods		O[56]		
Avalanche transceivers	O[37]			
Magnetically levitated linear motor car		O[52]		

EAS, electronic article surveillance; EMI, electromagnetic interference; O, no EMI was found in this category; RFID, radiofrequency identification; X, EMI was reported for this study type in at least one study.

included in this review sensing anomalies (e.g. undersensing, oversensing),[31–34] asynchronous pacing,[33,34] increased pacing rate,[24,34] pacing inhibition,[24,32–35] and mode switch[32] were reported.

Oversensing in the atrial channel can be misinterpreted by CIEDs as atrial fibrillation and cause a change in the pacing mode to either VVI(+R) or DDI(+R) mode, which results in atrioventricular dysynchrony. The event may remain unnoticed and has no clinical consequence if the interference is brief. However, in the case of extended atrioventricular dysynchrony, patients with a high ventricular pacing percentage may develop the pacemaker syndrome, including symptoms of palpitations, dizziness, and reduced physical capacity.[69] Additionally, inappropriate mode switch episodes could lead physicians to initiate therapeutic oral anticoagulation if the episodes are not correctly identified as EMI. If atrial oversensing occurs and if mode switch is disabled, inappropriate ventricular pacing may be triggered up to the upper tracking rate.[9]

Oversensing in the ventricular channel may result in pacing inhibition with subsequent severe bradyarrhythmias, (near-)syncope, or asystole in PM-dependent patients.[70] Additionally, sustained ventricular oversensing in ICD patients may lead to inappropriate shock delivery. Inappropriate shocks are not only painful and can result in psychological distress but they can be potentially proarrhythmic and are associated with adverse overall survival.[71] In the case of strong EMF exposure, PMs/ICDs may switch to noise mode with asynchronous pacing (VOO/DOO).[72] In noise mode, the subsequent loss of sensing of the intrinsic signal prevents the detection of the underlying intrinsic rhythm faster than the pacing frequency resulting in a risk of T-wave stimuli as well as the perception of ventricular arrhythmias. Thus, anti-tachycardia therapy of ICDs would be withheld with potential lethal consequences.

Precautionary methods

Cardiologists can reduce the risk of EMI for CIED patients by evaluating and programming the sensitivity settings. The lowest possible sensitivity should be selected, which still ensures an appropriate sensing of intrinsic signals. Features like automatic capture measurement or adaptive sensitivity control may lead to inappropriate automatic reprogramming of the sensitivity and should therefore, be switched off in patients with foreseeable strong EMF exposure or documented EMI. In the case of ICDs, defibrillator testing with ventricular fibrillation induction may be necessary to evaluate appropriate sensing of fibrillation waves with sensitivity settings lower than the manufacturer's recommendations. Prolonged detection intervals and elevation of the VT/VF zones as mentioned in several studies[73–75] may prevent inappropriate shocks without impairing the outcome of the patients.

Programming to VVI mode is an additional option to prevent atrial oversensing which usually occurs before the ventricular channel is affected due to the small intrinsic atrial signals and the corresponding high sensitivity setting (poor signal-to-noise ratio). Therefore, to be able to programme a lower sensitivity it is important to achieve a stable anchoring of the lead with good sensing amplitudes during the implantation procedure.

In a systematic investigation on the CIED's lead location, we found that a medial position and horizontal orientation of a bipolar lead's distal end as well as a short lead's tip-to-ring spacing makes CIEDs less susceptible to EMI.[76] We, therefore, recommend the implantation of true bipolar leads and programming the sensing configurations appropriately in all patients, if possible. When changing the sensing configuration or in case of pre-existing unipolar leads, physicians, and patients should be aware of a higher likelihood of EMI.

Patients with recent EMI events, should first be advised to maintain a greater distance (usually >30 cm) to the source of EMF, followed by a careful evaluation of the technical integrity of the CIED. In addition, an in-depth analysis of the situation of EMI including field measurements, e.g. at the workplace, should be performed and a history of earlier device disturbances should be obtained. Furthermore, remote monitoring of devices may be of great help for early EMI detection.

Research needs

For future studies, we recommend using standardized exposure setups and to conduct different types of studies in order to achieve a comprehensive risk assessment for patients with CIEDs.

Exposure set-up and characterization

Only 11 of the 40 studies included in this review used a standardized exposure set-up, e.g. a Helmholtz coil or an antenna setting, while most of the studies conducted experiments with a single electronic appliance. For a general risk assessment, however, studies with single device exposure have only a limited value, often due to a lack of a proper dosimetry. In contrast, under standardized conditions, EMF of a defined frequency and field strength can be generated and applied. In such a setting, it is possible to determine exactly the exposure parameters for which EMI is likely or unlikely to be induced. When conducting studies with single devices a complete dosimetry should be performed such that the results are applicable to different exposure scenarios, including new technologies. It is important to characterize the field strength and distribution as well as the frequency and modulation (i.e. waveform such as CW vs. pulses) of EMF sources in the vicinity of the patient or implant. This view is supported by McIvor *et al.*[34] and Seidman *et al.*[47] who noted that the field strength, frequency and modulation are the crucial parameters for EMI. From their findings, McIvor *et al.*[34] further concluded that susceptibility to interference was enhanced by the 60 Hz pulsed signal. Additionally, Hikage *et al.*[40] found that EMI were more likely when CIEDs were exposed to pulsed signals with a repetition time close to the physiological heart rhythm.

Different types of studies

Beyond the use of standardized exposure set-ups and the characterization of the exposure parameters, we recommend performing benchmark tests, phantom, and *in vivo* studies. The combination of different types of studies will help to systematically evaluate the influence of CIED-, lead-, and patient-related factors. However, it has to be noted that each type of study has several advantages and disadvantages.

Benchmark tests are highly suited for investigating the influence of different CIED types and sensitivity settings. The disturbance signals are fed directly into the pace/sense channel of the CIED by galvanic coupling in order to analyse the CIED's response and detection levels. Several studies included in this review have shown that the likelihood for disturbance depends on the CIED type (i.e. PM or ICD, model[41,46,47]) and CIED sensitivity settings.[32,66] The simple methodology is a major advantage of benchmark tests. However, a disadvantage is that individual parameters of the patient or the lead cannot be considered.

The influence of the lead can best be examined in phantom studies. Additionally, with phantom studies, the intracorporal voltage induced by external EMF in the CIED-lead system can also be determined. The induced voltage is an important measurement parameter for electromagnetic compatibility testing of CIEDs because it can directly be compared with the performance limits set in international product standards, e.g. Ref.[53] Several studies included in this review have shown that the likelihood for EMI depends on the lead parameters (bipolar and unipolar[42]), lead configuration (i.e. loop area formed by implant housing with its lead wire), and implantation site.[58,59] In clinical practice, the susceptibility of CIEDs to EMI has been reduced by using bipolar instead of unipolar leads. However, bipolar leads are still susceptible to interference in the presence of strong EMF.[76]

Individual parameters, including height or physique, which are also affecting the interference threshold,[77] can however, neither be considered in phantom studies nor in benchmark tests. Therefore, *in vivo* studies should be performed in which patients with CIEDs are directly exposed to EMF and in which individual interference thresholds of the CIED can be determined for specific exposure conditions. Data obtained from *in vivo* studies need no additional validation and can be transferred directly to real-life exposure situations. Therefore, results from benchmark tests and phantom studies should always be validated by *in vivo* studies. A disadvantage of *in vivo* studies is, however, that they are time-consuming and that a large number of patients has to be tested to identify patient-related, CIED-related, and lead-specific predictors.

The combination of benchmark tests, phantom, and *in vivo* studies allows the development of a coupling model (transfer function). Transfer functions demonstrate the relationship between the strength of an external EMF and the induced intracorporal voltage at the terminals of a CIED. A solid transfer function which serves as the basis for the calculation of the induced voltages from various external EMF can be derived by using comprehensive data obtained from the different types of studies. Establishing a transfer function is necessary to define limit values.

It is of great importance to define limit values for patients with CIEDs because the current EMF limit values (e.g. ICNIRP,[17] 27 μT and 21 A/m for 3 kHz–10 MHz) proposed for the general public may be exceeded by everyday electrical appliances emitting EMF in the IF range. According to Leitgeb *et al.*,[60] EAS systems may exceed the limits by a factor of 13 compared with ICNIRP's recommendation published in 2010[17] and even by a factor of 60 with regard to ICNIRP's recommendation published in 1998.[78] Induction hobs may also exceed ICNIRP limit values in close proximity and depending on the position of the pot.[25] This does not suggest that the general public (including people with CIEDs) is automatically at risk but caution is warranted for worst-case exposure scenarios.[60] Vice versa, compliance with ICNIRP does not suggest that the safety of patients with CIEDs is guaranteed, because ICNIRP does not consider people fitted with electronic implants in their recommendation. Furthermore, the results of single studies in the present review provide evidence that EMI may even be induced below the proposed ICNIRP limit values.[33,58] Thus, the establishment of limit values for patients with CIEDs will contribute to estimate which electronic appliances can be considered safe for CIEDs carriers and which distances to various electronic appliances should be respected in order to prevent EMI.

Conclusion

There are several studies investigating EMI of CIEDs by novel electrical appliances emitting EMF in the IF range. However, the current data do not allow a general risk assessment for CIED carriers regarding common or future potential interferers, especially due to the lack of a proper dosimetry in most of the studies or the missing correlation of dosimetric data with EMI. The findings were only consistent for security systems and induction hobs for which EMI in CIEDs could be demonstrated in close proximity to the appliances. The results of the studies evaluated in this systematic review and the results of studies on EMI in other frequency ranges indicate that the likelihood for

EMI is dependent on exposure-related parameters (field strength, frequency, modulation) and on implant- as well as on lead-related parameters (model, type of implant, implant sensitivity setting, lead configuration, and implantation site). To better characterize the factors influencing EMI, future studies should consider all these factors systematically by conducting different types of studies. Benchmark test and phantom studies should be performed according to international standards.[53–55] Concerning *in vivo* studies, where no comparable recommendations exist, good experiences[70,72] were had in cooperation between cardiologists with profound knowledge in electrophysiology and electrical engineers with profound knowledge in exposure set-ups.

Additionally, worst-case scenarios should be considered in all study types where possible (i.e. unipolar sensing, maximum sensitivity, atrium sensing, sustained pacing of the CIED, left-sided implantation, lateral lead's tip position, vertical lead's tip orientation, homogeneous field exposure, and thorax perpendicular to the magnetic field exposure). That way, it might be possible to derivate EMF limit values for CIED patients in the future.

Supplementary material

Supplementary material is available at *Europace* online.

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COMMENT

Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest

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Abstract. The fifth generation, 5G, of radiofrequency (RF) radiation is about to be implemented globally without investigating the risks to human health and the environment. This has created debate among concerned individuals in numerous countries. In an appeal to the European Union (EU) in September 2017, currently endorsed by >390 scientists and medical doctors, a moratorium on 5G deployment was requested until proper scientific evaluation of potential negative consequences has been conducted. This request has not been acknowledged by the EU. The evaluation of RF radiation health risks from 5G technology is ignored in a report by a government expert group in Switzerland and a recent publication from The International Commission on Non-Ionizing Radiation Protection. Conflicts of interest and ties to the industry seem to have contributed to the biased reports. The lack of proper unbiased risk evaluation of the 5G technology places populations at risk. Furthermore, there seems to be a cartel of individuals monopolizing evaluation committees, thus reinforcing the no-risk paradigm. We believe that this activity should qualify as scientific misconduct.

Introduction

Most politicians and other decision-makers using guidelines for exposure to radiofrequency (RF) radiation seem to ignore the risks to human health and the environment. The fact that the International Agency for Research on Cancer (IARC) at the World Health Organization (WHO) in May 2011 classified RF radiation in the frequency range of 30 kHz to 300 GHz to be a 'possible' human carcinogen, Group 2B (1,2), is being ignored. This has been recently exemplified in a hearing at the Tallinn Parliament in Estonia (3).

An important factor may be the influence on politicians by individuals and organizations with inborn conflicts of interests (COIs) and their own agenda in supporting the no-risk paradigm (4,5). The International Commission on Non-Ionizing Radiation Protection (ICNIRP) has repeatedly ignored scientific evidence on adverse effects of RF radiation to humans and the environment. Their guidelines for exposure are based solely on the thermal (heating) paradigm and were first published in ICNIRP 1998 (6), updated in ICNIRP 2009 (7) and have now been newly published in ICNIRP 2020 (8), with no change of concept, only relying on thermal effects from RF radiation on humans. The large amount of peer-reviewed science on non-thermal effects has been ignored in all ICNIRP evaluations (9,10). Additionally, ICNIRP has successfully maintained their obsolete guidelines worldwide.

COIs can be detrimental, and it is necessary to be as unbiased as possible when assessing health risks. There are three points that should be emphasized. Firstly, the evidence regarding health risks from environmental factors may not be unambiguous, and therefore informed judgements must be made. Furthermore, there are gaps in knowledge that call for experienced evaluations, and no conclusion can be reached without value judgements. Secondly, paradigms are defended against the evidence and against external assessments by social networks in the scientific community. Thirdly, the stronger the impact of decisions about health risks on economic, military and political interests, the stronger will stakeholders try to influence these decision processes.

Since the IARC evaluation in 2011 (1,2), the evidence on human cancer risks from RF radiation has been strengthened based on human cancer epidemiology reports (9-11), animal carcinogenicity studies (12-14) and experimental findings on oxidative mechanisms (15) and genotoxicity (16). Therefore, the IARC Category should be upgraded from Group 2B to Group 1, a human carcinogen (17).

The deployment of the fifth generation, 5G, of RF radiation is a major concern in numerous countries, with groups of citizens trying to implement a moratorium until thorough research

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on adverse effects on human health and the environment has been performed. An appeal for a moratorium, currently signed by >390 international scientists and medical doctors, was sent to the European Union (EU) in September 2017 (18), currently with no EU response (19). Several regions have implemented a moratorium on the deployment of 5G motivated by the lack of studies on health effects, for instance Geneva (20).

In the present article, the current situation in Switzerland is discussed as an example (21). Additionally, the ICNIRP 2020 evaluation is discussed (8).

Evaluation of health risks in Switzerland

Several Swiss citizens have brought to our attention that Associate Professor Martin Röösli is the chair of two important government expert groups in Switzerland (directeur), despite possible COIs and a history of misrepresentation of science (22,23). These groups are Beratende Expertengruppe NIS (BERENIS; the Swiss advisory expert group on electromagnetic fields and non-ionizing radiation) (24), and the subgroup 3, the Mobile Communications and Radiation Working Group of the Department of the Environment, Transport, Energy and Communications/Eidgenössisches Departement für Umwelt, Verkehr, Energie und Kommunikation, evaluating RF-radiation health risks from 5G technology (25,26).

The conclusions made in the recent Swiss government 5G report are biased and can be found here (27,28). This 5G report concluded that there is an absence of short-term health impacts and an absence or insufficient evidence of long-term effects [see Table 17 (Tableau 17) on page 69 in the French version (27) and Table 17 (Tabelle 17) on page 67 in the German version (28)].

Furthermore, it was reported that there is limited evidence for glioma, neurilemmoma (schwannoma) and co-carcinogenic effects, and insufficient evidence for effects on children from prenatal exposure or from their own mobile phone use. Regarding cognitive effects, fetal development and fertility (sperm quality), the judgement was that the evidence on harmful effects is insufficient. These evaluations were strikingly similar to those of the ICNIRP (see Appendix B in ICNIRP 2020; 8). Other important endpoints, such as effects on blood-brain barrier, cell proliferation, apoptosis (programmed cell death), oxidative stress (reactive oxygen species) and gene and protein expression, were not evaluated.

According to Le Courrier November 19, 2019, Martin Röösli presented the conclusion in an interview in the following way: '*Sur l'aspect sanitaire pur, «le groupe de travail constate que, jusqu'à présent, aucun effet sanitaire n'a été prouvé de manière cohérente en dessous des valeurs limites d'immissions fixées», résume Martin Röösli, professeur d'épidémiologie environnementale à l'Institut tropical et de santé publique suisse*' (29). [*Regarding the health issue, the working group concludes that, until now, no health effect has been consistently proven below the given exposure limits, summarizes Martin Röösli, professor in environmental epidemiology at the Swiss Tropical and Public Health Institute*].

This Swiss evaluation is scientifically inaccurate and is in opposition to the opinion of numerous scientists in this field (18). In addition, 252 electromagnetic field (EMF) scientists from 43 countries, all with published peer-reviewed research on the biologic and health effects of nonionizing electromagnetic fields (RF-EMF) have stated that:

'*Numerous recent scientific publications have shown that RF-EMF affects living organisms at levels well below most international and national guidelines. Effects include increased cancer risk, cellular stress, increase in harmful free radicals, genetic damages, structural and functional changes of the reproductive system, learning and memory deficits, neurological disorders, and negative impacts on general well-being in humans. Damage goes well beyond the human race, as there is growing evidence of harmful effects to both plant and animal life*' (30).

We are concerned that the Swiss 5G report may be influenced by ties to mobile phone companies (COIs) by one or several members of the evaluating group.

COIs

Funding from telecom companies is an obvious COI. Martin Röösli has been a member of the board of the telecom funded Swiss Research Foundation for Electricity and Mobile Communication (FSM) organization and he has received funding from the same organization (31-33).

It should be noted that the FSM is a foundation that serves formally as an intermediate between industry and researchers. According to their website, among the five founders of FSM who '*provided the initial capital of the Foundation*' four are telecommunications companies: Swisscom, Salt, Sunrise, 3G Mobile (liquidated in 2011). The fifth founder is ETH Zurich (technology and engineering university). There are only two sponsors, Swisscom (telecommunications) and Swissgrid (energy), who '*support the FSM with annual donations that allow for both the management of the Foundation and research funding*' (34).

The same situation applies to being a member of ICNIRP (Table I) (35). In 2008, the Ethical Council at Karolinska Institute in Stockholm stated that being a member of ICNIRP is a potential COI. Such membership should always be declared. This verdict was based on activities by Anders Ahlbom in Sweden, at that time a member of ICNIRP, but is a general statement (2008-09-09; Dnr, 3753-2008-609). In summary: '*It is required that all parties clearly declare ties and other circumstances that may influence statements, so that decision makers and the public may be able to make solid conclusions and interpretations. AA [Anders Ahlbom] should thus declare his tie to ICNIRP whenever he makes statements on behalf of authorities and in other circumstances*' (translated into English).

COIs with links to industry are of great importance; these links may be direct or indirect funding for research, payment of travel expenses, participation in conferences and meetings, presentation of research, etc. Such circumstances are not always declared as exemplified above. A detailed description was recently presented for ICNIRP members (22).

ICNIRP

ICNIRP is a non-governmental organization (NGO) based in Germany. Members are selected via an internal process, and the organization lacks transparency and does not represent the

Table I. Members of the WHO core group and additional experts of the Environmental Health Criteria Document 2014 (54), EU SCENIHR 2015 (52), the SSM 2015-2020 (93) and ICNIRP commission or the Scientific Expert Group 1992-2020 (94).

Members	WHO, 2014	SCENIHR, 2015	SSM, 2015-2020	ICNIRP, 1992-2020
Emilie van Deventer	X		X	X[a]
Simon Mann	X			X
Maria Feychting	X		(X)[b]	X
Gunnhild Oftedal	X			X
Eric van Rongen	X		X	X
Maria Rosaria Scarfi	X	X	X	X
Jukkka Juutilainen	X			X
Denis Zmirou	X			
Theodoros Samaras		X		
Norbert Leitgeb		X		
Anssi Auvinen		X		X
Heidi Danker Hopfe		X	X	
Kjell Hansson Mild		X		
Mats Olof Mattsson		X		X
Hannu Norppa		X		
James Rubin	X	X		
Joachim Schüz		X		
Zenon Sienkiewicz	X	X		X
Olga Zeni	X	X		
Anke Huss			X	X[c]
Clemens Dasenbrock			X	X
Lars Klaeboe			X	
Martin Röösli	X		X	X
Aslak Harbo Poulsen			X	

[a]WHO Observer in the main commission (95); [b]2002-2011; [c]2020-2024. The table is based on members of WHO, SCENIHR and SSM during the defined time period(s). No other individuals among those within WHO or SCENIHR were found in the list of SSM participants. A total of 15 additional experts in WHO were not members of SCENIHR, SSM or ICNIRP. SCENIHR, Scientific Committee on Emerging and Newly Identified Health Risks; SSM, Swedish Radiation Safety Authority; WHO, World Health Organization; EU, European Union; ICNIRP, International Commission on Non-Ionizing Radiation Protection.

opinion of the majority of the scientific community involved in research on health effects from RF radiation. Independent international EMF scientists in this research area have declared that: '*In 2009, the ICNIRP released a statement saying that it was reaffirming its 1998 guidelines, as in their opinion, the scientific literature published since that time has provided no evidence of any adverse effects below the basic restrictions and does not necessitate an immediate revision of its guidance on limiting exposure to high frequency electromagnetic fields. ICNIRP continues to the present day to make these assertions, in spite of growing scientific evidence to the contrary. It is our opinion that, because the ICNIRP guidelines do not cover long-term exposure and low-intensity effects, they are insufficient to protect public health*' (30).

ICNIRP only acknowledges thermal effects from RF radiation. Therefore, the large body of research on detrimental non-thermal effects is ignored. This was further discussed in a peer-reviewed scientific comment article (3).

In 2018, ICNIRP published '*ICNIRP Note: Critical Evaluation of Two Radiofrequency Electromagnetic Field Animal Carcinogenicity Studies Published in 2018*' (36). It is surprising that this note claims that the histopathological evaluation in the US National Toxicology Program (NTP) study on animals exposed to RF radiation was not blinded (12,13). In fact, unfounded critique of the NTP study had already been rebutted (37); however, this seems to have had little or no impact on this ICNIRP note casting doubt on the findings of the animal study: '*This commentary addresses several unfounded criticisms about the design and results of the NTP study that have been promoted to minimize the utility of the experimental data on RFR [radiofrequency radiation] for assessing human health risks. In contrast to those criticisms, an expert peer-review panel recently concluded that the NTP studies were well designed, and that the results demonstrated that both GSM- and CDMA-modulated RFR were carcinogenic to the heart (schwannomas) and brain (gliomas) of male rats*' (37).

In contrast to the opinion of the 13 ICNIRP commission members, the IARC advisory group of 29 scientists from 18 countries has recently stated that the cancer bioassay in experimental animals and mechanistic evidence warrants high priority re-evaluation of the RF radiation-induced carcinogenesis (38).

ICNIRP draft. On July 11, 2018, ICNIRP released a draft on guidelines (39) for limiting exposure to time-varying electric, magnetic and electromagnetic fields (100 kHz to 300 GHz). It was open for public consultations until October 9, 2018. Appendix B was based on assessment of health risks based on a literature survey (39).

Surprisingly, the IARC classification of RF-EMF exposure as Group 2B ('possibly' carcinogenic to humans) from 2011 was concealed in the background material to the new ICNIRP draft on guidelines. Notably, one of the ICNIRP commission members, Martin Röösli (40), was also one of the IARC experts evaluating the scientific RF carcinogenicity in May 2011 (41). He should be well aware of the IARC classification. The IARC classification contradicts the scientific basis for the ICNIRP guidelines, making novel guidelines necessary and providing a basis to halt the rollout of 5G technology.

Therefore, the ICNIRP provides scientifically inaccurate reviews for various governments. One issue is that only thermal (heating) effects from RF radiation are considered, and all non-thermal effects are dismissed. An analysis from the UK demonstrates these inaccuracies (4), also discussed in another article (5). All members of the ICNIRP commission are responsible for these biased statements that are not based on solid scientific evidence.

ICNIRP release of novel guidelines for RF radiation. On March 11, 2020, ICNIRP published their novel guidelines for exposure to EMFs in the range of 100 kHz to 300 GHz, thus including 5G (8). The experimental studies demonstrating a variety of non-thermal biological/health effects (9,10) are not considered, as in their previous guidelines (6,7). Additionally, the ICNIRP increased the reference levels for the general public averaged over 6 min for RF frequencies >2-6 GHz (those that will be used for 5G in this frequency range), from 10 W/m^2 (Tables 5 and 7 in ref. no. 6) to 40 W/m^2 (Table 6 in ref. no. 8), which paves the way for even higher exposure levels to 5G than the already extremely high ones.

Background dosimetry is discussed in Appendix A of the ICNIRP 2020 guidelines (8). The discussion on 'Relevant Biophysical Mechanisms' should be criticized. The only mechanism considered by ICNIRP is temperature rise, which may also occur with 5G exposure, apart from the established non-thermal biological/health effects (42,43). It is well known among experts in the EMF-bioeffects field that the recorded cellular effects, such as DNA damage, protein damage, chromosome damage and reproductive declines, and the vast majority of biological/health effects are not accompanied by any significant temperature rise in tissues (44-47). The ion forced-oscillation mechanism (48) should be referred to as a plausible non-thermal mechanism of irregular gating of electrosensitive ion channels on cell membranes, resulting in disruption of the cell electrochemical balance and initiating free radical release and oxidative stress in the cells, which in turn causes genetic damage (15,49). The irregular gating of ion channels on cell membranes is associated with changes in permeability of the cell membranes, which ICNIRP admits in its summary (8).

Health risks are discussed in Appendix B of the ICNIRP 2020 guidelines (8). Again, only thermal effects are considered, whereas literature on non-thermal health consequences is disregarded (9,10,50). In spite of public consultations on the draft, the final published version on health effects is virtually identical to the draft version, and comments seem to have been neglected (19). In the following section, Appendix B on health effects (8) is discussed.

Appendix B starts with: '*The World Health Organization (WHO) has undertaken an in-depth review of the literature on radiofrequency electromagnetic fields (EMFs) and health, which was released as a Public Consultation Environmental Health Criteria Document in 2014... Further, the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), a European Commission initiative, also produced a report on potential health effects of exposure to electromagnetic fields (SCENIHR 2015), and the Swedish Radiation Safety Authority (SSM) have produced several international reports regarding this issue (SSM 2015, 2016, 2018). Accordingly, the present guidelines have used these literature reviews as the basis for the health risk assessment associated with exposure to radiofrequency EMFs rather than providing another review of the individual studies'*.

In the last 11 years since its previous ICNIRP 2009 statement (7), ICNIRP has not managed to conduct a novel evaluation of health effects from RF radiation. However, as shown in Table I, several of the present ICNIRP members are also members of other committees, such as the EU Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), the Swedish Radiation Safety Authority (SSM) and the WHO, thus creating a cartel of individuals known to propagate the ICNIRP paradigm on RF radiation (4,5,22,51). In fact, six of the seven expert members of the WHO, including Emelie van Deventer, were also included in ICNIRP (5,7). Therefore, Emilie van Deventer, the team leader of the Radiation Programme at WHO (the International EMF Project), is an observer on the main ICNIRP commission, and SSM seems to be influenced by ICNIRP. Among the current seven external experts (Danker-Hopfe, Dasenbrock, Huss, Harbo Polusen, van Rongen, Röösli and Scarfi), five are also members of ICNIRP, and van Deventer used to be part of SSM.

As discussed elsewhere (5), it is unlikely that a person's evaluation of health risks associated with exposure to RF radiation would differ depending on what group the person belongs to. Therefore, by selecting group members, the final outcome of the evaluation may already be predicted (no-risk paradigm). Additionally, we believe that this may compromise sound scientific code of conduct.

The SCENIHR report from 2015 (52) has been used to legitimate the further expansion of the wireless technology and has been the basis for its deployment in a number of countries. One method, applied in the SCENIHR report, to dismiss cancer risks involves the selective inclusion of studies, excluding studies reporting cancer risks and including some investigations with inferior epidemiological quality. The report has been heavily criticized by researchers with no COI (53): '*In January of 2015, the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) published its final opinion on (P)otential health effects of exposure to electromagnetic fields... SCENIHR has not answered the question it was appointed to investigate. The Committee has answered a*

different question, limiting its conclusions to whether certainty or causal effect is established, instead of possibility of health risks... Overall, SCENIHR has not conducted a scientific review process for judging possible health risks. This results in erroneous and deceptive conclusions by failing to conclude such possible health risks do exist. Evidence that SCENIHR has presented clearly and conclusively demonstrates that EMF health risks are possible, and in some cases are established. The Committee is obligated to draw to the attention of the European Commission that EMF is a new and emerging problem that may pose an actual or potential threat'.

Regarding the SSM, only yearly updates are available and no overall evaluations are made. Therefore, no thorough review is presented. Over the years, the ICNIRP has dominated this committee (Table I). Therefore, it is unlikely that the opinion of the SSM will differ from that of the ICNIRP.

In 2014, the WHO launched a draft of a Monograph on RF fields and health for public comments (54). It should be noted that the WHO issued the following statement: '*This is a draft document for public consultation. Please do not quote or cite*'. ICNIRP completely ignored that request and used the aforementioned document. The public consultations on the draft document were dismissed and never published.

In addition to van Deventer, five of the six members (Mann, Feychting, Oftedal, van Rongen, and Scarfi) of the Core Group in charge of the WHO draft were also affiliated with ICNIRP, which constitutes a COI (Table I). Scarfi is a former member of ICNIRP (5). Several individuals and groups sent critical comments to the WHO on the numerous shortcomings in the draft of the Monograph on RF radiation. In general, the WHO never responded to these comments and it is unclear to what extent, if any, they were even considered. Nevertheless, the final version of the WHO 'in-depth review' has never been published. Instead, WHO made a call on October 8, 2019 (Emelie van Deventer), for systematic reviews to analyze and synthesize the available evidence: '*Through this Call, WHO invites eligible teams to indicate their interest in undertaking a systematic review on one (or more) of the following topics: SR1 - Effect of exposure to RF on cancer (human observational studies); SR2 - Effect of exposure to RF on cancer (animal studies); SR3 - Effect of exposure to RF on adverse reproductive outcomes (human observational studies); SR4 - Effect of exposure to RF on adverse reproductive outcomes (animal and in vitro studies); SR5 - Effect of exposure to RF on cognitive impairment (human observational studies; SR6 - Effect of exposure to RF on cognitive impairment (human experimental studies); SR7 - Effect of exposure to RF on symptoms (human observational studies); SR8 - Effect of exposure to RF on symptoms (human experimental studies; SR9 - Effect of exposure to RF on biomarkers of oxidative stress; SR10 - Effect of exposure to heat from any source and pain, burns, cataract and heat-related illness*'.

The authors of the present article were part of a team that applied to review SR1- human cancer. On December 20, 2019, the following reply was received from the WHO Radiation Programme: '*After careful review, we have decided to choose another team for this systematic review*'.

Transparency is of importance for the whole process. Therefore, a query was sent to the WHO requesting information regarding the following points: '*Who did the evaluation of the groups that answered the call? What criteria were applied? How many groups had submitted and who were these? Which groups were finally chosen for the different packages?*'. In spite of sending the request four times, January 2, January 3, April 7 and April 30, 2020, there has been no reply from WHO. This appears to be a secret process behind closed doors. These circumstances have also been reported in Microwave News (55).

It is important to comment on the current ICNIRP evaluation. Notably, on February 27, 2020, two weeks before the ICNIRP publication, the WHO Team on Public Health, Environmental and Social Determinants of Health issued a statement on 5G mobile networks and health: '*To date, and after much research performed, no adverse health effect has been causally linked with exposure to wireless technologies*' (56). This statement is not correct based on current knowledge (4,5,9-11,17,19) and was without a personal signature. The lack of research on 5G safety has been previously discussed (19). Furthermore, there is no evidence that can 'causally link' an adverse effect to an exposure. Causality is no empirical fact, it is an interpretation.

In the following section, only one (cancer) of the eight different end points in the ICNIRP publication (8) is discussed, since it deals with our main research area.

viii) Cancer.

'I*n summary, no effects of radiofrequency EMFs on the induction or development of cancer have been substantiated.*

Summary

The only substantiated adverse health effects caused by exposure to radiofrequency EMFs are nerve stimulation, changes in the permeability of cell membranes, and effects due to temperature elevation. There is no evidence of adverse health effects at exposure levels below the restriction levels in the ICNIRP (1998) guidelines and no evidence of an interaction mechanism that would predict that adverse health effects could occur due to radiofrequency EMF exposure below those restriction levels'.

Comments

The ICNIRP draft (39) has been previously described to some extent (19). The published final version on health effects is virtually similar to the draft. It cannot be taken at face value as scientific evidence of no risk from RF radiation. One example is the following statement (p. 41): '*...a set of case-control studies from the Hardell group in Sweden report significantly increased risks of both acoustic neuroma and malignant brain tumors already after less than five years since the start of mobile phone use, and at quite low levels of cumulative call time'.*

This allegation is not correct according to our publication for glioma (11). In the shortest latency group >1-5 years, the risk of glioma was not increased (odds ratio (OR), 1.1; 95% CI, 0.9-1.4) for use of wireless phones (mobile phone and/or cordless phone). There was a statistically significant increased risk of glioma per 100 h of cumulative use (OR, 1.011; 95% CI, 1.008-1.014) and per year of latency (OR, 1.032; 95% CI,

1.019-1.046) (11). These published results are in contrast to the ICNIRP claims.

Regarding acoustic neuroma, the corresponding detailed results are reported in our previous study (57). The shortest latency period >1-5 years yielded an OR of 1.2 (95% CI, 0.8-1.6) for use of wireless phones; the risk increased per 100 h of cumulative use (OR, 1.008; 95% CI, 1.002-1.014) and per year of latency (OR, 1.056; 95% CI, 1.029-1.085) (57). Therefore, the allegation by ICNIRP is false.

It is remarkable that ICNIRP is uninformed and that their writing is based on a misunderstanding of the peer-reviewed published articles as exemplified above. Additionally, our studies (11,57) and another study by Coureau *et al* (58), as well as the IARC evaluation from 2011 (1,2), are not included among the references. Several statements by ICNIRP are made without any scientific references. On the other hand, the Danish cohort study on mobile phone use (59) is included, in spite of the fact that it was judged by IARC (1,2), as well as in our review (60), to be uninformative. A biased article written by authors including ICNIRP members, used to 'prove' the no-risk paradigm for RF radiation carcinogenesis (23), is cited by ICNIRP. Notably, the article has not undergone relevant peer-review and we believe that it should not have been published in its current version. The shortcomings in the aforementioned article are discussed in the following sections. As discussed below, another claim (23) is incorrect regarding increased risk of brain tumors associated with use of wireless phones: '*However, they are not consistent with trends in brain cancer incidence rates from a large number of countries or regions, which have not found any increase in the incidence since mobile phones were introduced'*.

The criticism of the ICNIRP draft guidelines from 2018 by the EMF call (61) can also be applied to the current ICNIRP publication. The call has been signed by 164 scientists and medical doctors, as well as 95 NGOs: '*The International Commission on Non-Ionizing Radiation Protection (ICNIRP) issued draft Guidelines on 11th July 2018 for limiting exposure to electric, magnetic and electromagnetic fields (100 kHz to 300 GHz).1 These guidelines are unscientific, obsolete and do not represent an objective evaluation of the available science on effects from this form of radiation. They ignore the vast amount of scientific findings that clearly and convincingly show harmful effects at intensities well below ICNIRP guidelines.2 The guidelines are inadequate to protect humans and the environment. ICNIRP guidelines only protect against acute thermal effects from very short and intense exposure. The guidelines do not protect against harmful effects from low-intensity and long-term exposure, such as cancer, reproductive harm, or effects on the nervous system, although these effects are convincingly shown to appear from chronic exposure at intensities below ICNIRP limits.2,3*

ICNIRP's mandate to issue exposure guidelines needs to be seriously questioned. ICNIRP is not independent of industry ties as it claims.12,13 Its opinions are not objective, not representative of the body of scientific evidence, but are biased in favor of industry. It is obvious from their reluctance to consider scientific findings of harm that ICNIRP protects industry, not the public health, nor the environment.

We ask the United Nations, the World Health Organization, and all governments to support the development and consideration of medical guidelines16, that are independent of conflict of interests in terms of direct or indirect ties to industry, that represent the state of medical science, and that are truly protective'.

In the recent report on ICNIRP published by two Members of the European Parliament it is concluded: '*That is the most important conclusion of this report: For really independent scientific advice we cannot rely on ICNIRP. The European Commission and national governments, from countries like Germany, should stop funding ICNIRP. It is high time that the European Commission creates a new, public and fully independent advisory council on non-ionizing radiation'* (22).

Other examples of scientific misrepresentation

Published article. This section discusses an article with conclusions not substantiated by scientific evidence, representing a biased evaluation of cancer risks from mobile phone use and is an example of lack of objectivity and impartiality (23). The aforementioned report was used by ICNIRP 2020 (8) to validate that no risks have been found for brain and head tumors. Therefore, the article should be discussed in further detail.

The aforementioned article has numerous severe scientific deficiencies. One is that the results on use of cordless phones as a risk factor for brain tumors are not discussed. In fact, detailed results on cordless phones in studies by Hardell *et al* (11,57) are omitted.

When discussing glioma risk, all results on cumulative use of mobile phones, as well as ipsilateral or contralateral use associated with tumor localization in the brain, are omitted from the figures in the main text. Some results in the article by Röösli *et al* (23), such as cumulative use, can be found in the Supplementary Material, although the increased risk among heavy users is disregarded (11,57,58,62). In Supplementary Figure 4, all odds ratios regarding long-term (≥10 years) use of mobile phones are above unity (>1.0) for glioma and neuroma (23). No results are provided for ipsilateral mobile phone use (same side of tumor localization and mobile phone use), which is of large biological importance. Results on cumulative use, latency and ipsilateral use are especially important for risk assessment and have shown a consistent pattern of increased risk for brain and head tumors (11,57).

In the aforementioned article, recall bias is discussed as the reason for increased risk (23). The studies by Hardell *et al* (11,57) included all types of brain tumors. In one analysis, meningioma cases in the same study were used as the 'control' entity (11), and still a statistically significant increased risk of glioma was identified for mobile phone use (ipsilateral OR, 1.4; 95% CI, 1.1-1.8; contralateral OR, 1.0; 95% CI, 0.7-1.4) and for cordless phone use (ipsilateral OR, 1.4; 95% CI, 1.1-1.9; contralateral OR, 1.1; 95% CI, 0.8-1.6). If the results were 'explained' by recall bias, similar results would have been obtained for both glioma and meningioma. Thus, this type of analyses would not have yielded an increased glioma risk. Also, for acoustic neuroma a statistically significant increased risk was found using meningioma cases as 'controls' (57). Therefore, the results in the studies by Hardell *et al* (11,57) cannot be explained by a systematic difference in assessment

of exposure between cases and controls. These important methodological findings were disregarded by Röösli *et al* (23).

In the analyses of long-term use of mobile phones, a Danish cohort study on mobile phone use is included (59), which was concluded to be uninformative in the 2011 IARC evaluation (1,2). A methodological shortcoming of the aforementioned study was that only private mobile phone subscribers in Denmark between 1982 and 1995 were included in the exposure group (59). The most exposed group, comprising 200,507 corporate users of mobile phones, were excluded and instead included in the unexposed control group consisting of the rest of the Danish population. Users with mobile phone subscription after 1995 were not included in the exposed group and were thus treated as unexposed at the time of cut-off of the follow up. No analysis of laterality of mobile phone use in relation to tumor localization was performed. Notably, this cohort study is now included in the risk calculations, although Martin Röösli was a member of the IARC evaluation group and should have been aware of the IARC decision. The numerous shortcomings in the Danish cohort study, discussed in detail in a peer-reviewed article (60), are omitted in the article by Röösli *et al* (23).

Regarding animal studies, a study by Falcioni *et al* (14) at the Ramazzini Institute on RF radiation carcinogenesis is only mentioned as a reference, but the results are not discussed. In fact, these findings (14) provide supportive evidence on the risk found in human epidemiology studies (3), as well as the results in the NTP study (12,13).

Furthermore, for incidence studies on brain tumors, the results are not presented in an adequate way. There is a lot of emphasis on the Swedish Cancer Register data (63,64), but the numerous shortcomings in the reporting of brain tumor cases to the register are not discussed. These shortcomings have been presented in detail in a previous study (63), but are disregarded by Röösli *et al* (23).

There is clear evidence from several countries regarding increasing numbers of patients with brain tumors, such as in Sweden (63,64), England (65), Denmark (66) and France (67).

The article by Röösli *et al* (23), does not represent an objective scientific evaluation of brain and head tumor risk associated with the use of wireless phones, and should thus be disregarded. By omitting results of biological relevance and including studies that have been judged to be uninformative, the authors come to the conclusion that there are no risks: '*In summary, current evidence from all available studies including in vitro, in vivo, and epidemiological studies does not indicate an association between MP [mobile phone] use and tumors developing from the most exposed organs and tissues'*.

Röösli *et al* (23), disregard the concordance of increased cancer risk in human epidemiology studies (11,57,58,62) animal studies (12-14,68,69) and laboratory studies (15,16,37). It is unfortunate that the review process of the aforementioned article has not been of adequate quality. Finally, there is no statement in the article of specific funding of this particular work, which is not acceptable. Only a limited number of comments on general funding are provided. It is not plausible that there was no funding for the study. We believe that, due to its numerous limitations, the aforementioned article should not have been published.

CEFALO. In 2011, a case-control study on mobile phone use and brain tumor risk among children and adolescents termed CEFALO was published (70). The study appears to have been designed to misrepresent the true risk, since the following question regarding cordless phone use was asked: '*How often did [child] speak on the cordless phone in the first 3 years he/she used it regularly?'*.

There are no scientific valid reasons to limit the investigation to the first 3 years. The result is a misrepresentation and a wrong exposure classification, since Aydin *et al* (70) willingly omitted any increase in the child's use of and exposure from cordless phone radiation after the first 3 years of use. This unscientific treatment of cordless phone exposure was not mentioned in the article other than in a footnote of a table and in the methods section (70); however, no explanation was provided: '*Specifically, we analyzed whether subjects ever used baby monitors near the head, ever used cordless phones, and the cumulative duration and number of calls with cordless phones in the first 3 years of use'*.

Since previous studies have demonstrated that these phone types, in addition to mobile phones, increase brain tumor risk (11,57), we believe that the exclusion of a complete exposure history on the use of cordless phones represents scientific misconduct.

In a critical comment the authors of the present study wrote: '*Further support of a true association was found in the results based on operator-recorded use for 62 cases and 101 controls, which for time since first subscription >2.8 years yielded OR 2.15 (95% CI 1.07-4.29) with a statistically significant trend (P = 0.001). The results based on such records would be judged to be more objective than face-to-face interviews, as in the study that clearly disclosed to the interviewer who was a case or a control. The authors disregarded these results on the grounds that there was no significant trend for operator data for the other variables - cumulative duration of subscriptions, cumulative duration of calls and cumulative number of calls. However, the statistical power in all the latter groups was lower since data was missing for about half of the cases and controls with operator-recorded use, which could very well explain the difference in the results*' (71).

Our conclusion was that: '*We consider that the data contain several indications of increased risk, despite low exposure, short latency period, and limitations in the study design, analyses and interpretation. The information certainly cannot be used as reassuring evidence against an association, for reasons that we discuss in this commentary*' (71).

This is in contrast to the authors that claimed that the study was reassuring of no risk in a press release from Martin Röösli, July 28, 2011: '*Kein erhöhtes Hirntumorrisiko bei Kindern und Jugendlichen wegen Handys... Die Resultate sind beruhigend*' ['*No increased brain tumour risk in children and adolescents for mobile phone users... The results are reassuring*'] (72).

A similar press release was issued by Maria Feychting at the Karolinska Institute in Stockholm stating: '*Reassuring results from first study on young mobile users and cancer risk… The so called CEFALO study does not show an increased brain tumor risk for young mobile users'* (73). Considering the results and the numerous scientific shortcomings in the study (70), the statements in these press releases are not correct.

Discussion

There is no doubt that several individuals included in Table I are influential, being members, as well as having consulting assignments, in several organizations, such as ICNIRP, BERENIS, the SSM, the Program Electromagnetic Fields and Health from ZonMw in the Netherlands, and the Rapid Response Group for the Japan EMF Information Center (74).

In fact, there appears to be a cartel of individuals working on this issue (75). Associate Professor Martin Röösli has had the chance to provide his view on the content of the present article relating to him. The only message from him was in an e-mail dated January 16, 2020: '*Just to be clear, all my research is funded by public money or not-for -profit fundations [foundations]. I think you will not help an important debate if you spread fake news'*. Obviously, as described in the present article, his comment is not correct considering his funding from the telecom industry (76,77).

As shown in Table I, few individuals, and mostly the same ones, are involved in different evaluations of health risks from RF radiation and will thus propagate the same views on the risks in agencies of different countries associated with the ICNIRP views (4,5). Therefore, it is unlikely that they will change their opinions when participating in different organizations. Furthermore, their competence in natural sciences, such as medicine, is often low or non-existent due to a lack of education in these disciplines (2). Therefore, any chance for solid evaluations of medical issues is hampered. Additionally, it must be concluded that if the 'thermal only' dogma is dismissed, this will have wide consequences for the whole wireless community, including permissions for base stations, regulations of the wireless technology and marketing, plans to roll out 5G, and it would therefore have a large impact on the industry. This may explain the resistance to acknowledge the risk by ICNIRP, EU, WHO, SSM and other agencies. However, the most important aspects to consider are human wellbeing and a healthy environment. Telecoms can make profit in a variety of ways, and wireless is just one of them. They have the capacity to maintain profits by using different techniques, such as optical fiber, that will provide more data with less RF radiation exposure. Particularly when considering the liability, they are incurring in their misguided insistence of wireless expansion that may ultimately catch up to them in the form of lawsuits, such as those previously experienced by asbestos and tobacco companies (78,79).

A recent book describes how deception is used to capture agencies and hijack science (80). There are certain tools that can be used for this. One is to re-analyze existing data using methods that are biased towards predetermined results (23). For example, this can be performed by hiring 'independent experts' to question scientific results and create doubt (81,82). As clearly discussed in a number of chapters of the books (80-82), front groups may be created to gain access to politicians and to influence the public with biased opinions. Other methods may involve intimidating and harassing independent scientists that report health risks based on sound science, or removing all funding from scientists who do not adhere to the no-risk pro-industry paradigm. Another tool would be economic support and courting decision makers with special information sessions that mislead them on science and mask bribery (3,5,19,80-82). An industry with precise marketing goals has a big advantage over a loose scientific community with little funding. Furthermore, access to regulatory agencies and overwhelming them with comments on proposed regulations is crucial (3). To counteract all these actions is time consuming and not always successful (19). Nevertheless, it is important that these circumstances are explored and published in the peer-reviewed literature as historical notes for future use.

Based on the Swiss and ICNIRP experiences, some recommendations can be made. One is to include only unbiased and experienced experts without COIs for evaluation of health risks from RF radiation. All countries should declare a moratorium on 5G until independent research, performed by scientists without any ties to the industry, confirms its safety or not. 2G, 3G, 4G and WiFi are also considered not to be safe, but 5G will be worse regarding harmful biological effects (42,83,84). The authors of the present article recommend an educational campaign to educate the public about the health risks of RF radiation exposure, and safe use of the technology, such as the deployment of wired internet in schools (85), as previously recommended by the European Council resolution 1815 in 2011 (86) and The EMF Scientist Appeal (87). Additionally, it is recommended that the government takes steps to markedly decrease the current exposure of the public to RF radiation, (88,89). Notably, DNA damage has been identified in peripheral blood lymphocytes using the comet assay technique, and in buccal cells using the micronucleus assay, in individuals exposed to RF radiation from base stations (90).

Finally, an alternative approach to the flawed ICNIRP safety standards may be the comprehensive work of the European Academy for Environmental Medicine (EUROPAEM) EMF working group that has resulted in safety recommendations, which are free from the ICNIRP shortcomings (50). Recently, the International Guidelines on Non-Ionising Radiation (IGNIR) have accepted EUROPAEM safety recommendations (91). The Bioinitiative group has recommended similar safety standards based on non-thermal EMF effects (92). WHO and all nations should adopt the EUROPAEM/Bioinitiative/IGNIR safety recommendations, supported by the majority of the scientific community, instead of the obsolete ICNIRP standards.

In conclusion, it is important that all experts evaluating scientific evidence and assessing health risks from RF radiation do not have COIs or bias. Being a member of ICNIRP and being funded by the industry directly, or through an industry-funded foundation, constitute clear COIs. Furthermore, it is recommended that the interpretation of results from studies on health effects of RF radiation should take sponsorship from the telecom or other industry into account. It is concluded that the ICNIRP has failed to conduct a comprehensive evaluation of health risks associated with RF radiation. The latest ICNIRP publication cannot be used for guidelines on this exposure.

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Authors' contributions

LH and MC contributed to the conception, design and writing of the manuscript. Both authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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INSURANCE FOR ARCHITECTS & ENGINEERS

ProSurance™ A&E Policy Document

Contents

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PREAMBLE

This Policy is a contract of insurance between **you** and **us**. **Your** Policy contains all the details of the cover that **we** provide, subject always to **our** receipt of the Premium. This Policy consists of and must be read together with the Declarations and any Endorsements. This Policy is not complete unless it is signed and a Declarations page is attached.

The Sections of this policy are identified by **BLUE LINES** across the page with **WHITE UPPER CASE PRINT**. Clause headings in blue **UPPER CASE PRINT** are for information only and do not form part of the cover given by this Policy. Other terms in **bold lower case print** are defined terms and have a special meaning as set forth in the **DEFINITIONS** section and elsewhere. Words stated in the singular shall include the plural and vice versa. However, this protocol does not apply to the **STATUTORY CONDITIONS** and subsequent Sections.

IMPORTANT: INSURING CLAUSES 1 and **2** provide cover on a claims made basis. Under these **INSURING CLAUSES** a **claim** must be first made against the company named as the Insured in the Declarations or any **subsidiary** during the **period of the policy** and notified to **us** during the **period of the policy** to be covered.

In consideration of the Premium and in reliance upon the information that **you** have provided to **us** prior to commencement of this insurance and which is deemed to form the basis of this insurance:

INSURING CLAUSES

INSURING CLAUSE 1: ERRORS & OMISSIONS

SECTION A: PROFESSIONAL LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of any:

a) negligent act, error or omission, negligent misstatement or negligent misrepresentation;
b) breach of any contractual term implied by law concerning necessary quality, safety or fitness, or **your** duty to use reasonable care and skill;
c) breach of warranty of authority, breach of duty, breach of trust, breach of confidence, misuse of information or breach of privacy;
d) libel, slander or defamation;
e) dishonesty of **your** directors, partners, officers or **employees** provided that **we** maintain all rights of subrogation to recover such legal **costs and expenses** from any director, partner, officer or **employee** if they are found guilty of such a dishonest act; or
f) other act, error or omission giving rise to civil liability to **your** clients but not any breach of contract save as specified above;

committed by **you** or on **your** behalf in the course of **your business activities**. **We** will also pay **costs and expenses** on **your** behalf.

SECTION B: BREACH OF CONTRACT

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** by a **client** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of any **breach of client contract**. **We** will also pay **costs and expenses** on **your** behalf.

SECTION C: INTELLECTUAL PROPERTY RIGHTS INFRINGEMENT

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of **your** infringement of any **intellectual property right** in the course of **your business activities**. **We** will also pay **costs and expenses** on **your** behalf.

SECTION D: POLLUTION LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of:

a) pollution or contamination of the atmosphere, or of any water, land, buildings or other property;
b) any enforcement action in connection with the containment, clean-up, removal or treatment of such pollution or contamination.

We will also pay **costs and expenses** on **your** behalf.

SECTION E: LOSS OF DOCUMENTS

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of destruction of, damage to, loss or mislaying of **your documents** or **documents** in **your** care, custody or control. **We** will also pay **costs and expenses** on **your** behalf.

SECTION F: COMPUTER VIRUS AND HACKING ATTACK

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** as a direct result of:

a) any **third parties**' financial losses arising directly from a **hacking attack** or **virus** that has emanated from or passed through **your** computer systems, or
b) any **third parties**' financial losses arising directly from their inability to access **your** computer systems in the way in which **you** have authorised them to as a direct result of **your** computer systems' failure or impairment due to a **hacking attack** or **virus**, or
c) any **third parties**' financial losses arising directly from the loss or theft of **your** data or data for which **you** are responsible or held to be responsible arising directly from a **hacking attack** or **virus**.

We will also pay **costs and expenses** on **your** behalf.

SECTION G: LOSS MITIGATION

We agree to pay any reasonable costs necessarily incurred by **you** with **our** prior written consent in respect of measures taken by **you** for the sole purpose of avoiding or mitigating a **claim** or potential **claim** for which **you** would be entitled to indemnity under **INSURING CLAUSE 1** of this Policy had such measures not been taken.

SECTION H: PAYMENT OF WITHHELD FEES

We agree to pay **your withheld fees** with **our** prior written consent in the event that **your client** brings or threatens to bring a **claim** against **you** that would be covered under **INSURING CLAUSE 1**, SECTIONS A or B for an amount greater than **your withheld fees** if **you** attempt to recover the **withheld fees** from them. Prior to payment of **your withheld fees you** must obtain written confirmation from **your client** that they will not bring a **claim** against **you** if **you** agree not to pursue them for **your withheld fees** and provide it to **us**.

ALL SECTIONS

We will not make any payment on **your** behalf under any **SECTION** of

this **INSURING CLAUSE** in respect of any **claim** arising directly or indirectly out of **injury** or **damage**:

a) from **products** or **workmanship**; or
b) that did not occur directly as a result of **your business activities**.

INSURING CLAUSE 2: EMPLOYEE BENEFITS LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during the **period of the policy** arising out of any negligent act, error or omission committed by **you** or on **your** behalf in the **administration** of **your employee benefit program**. **We** will also pay **costs and expenses** on **your** behalf.

Furthermore, **we** agree to pay on **your** behalf any **costs and expenses** necessarily incurred with **our** prior written consent in respect of measures taken by **you** with the object of avoiding or mitigating a **claim** for which **you** would be entitled to indemnity hereunder had such measures not been taken.

INSURING CLAUSE 3: THIRD PARTY LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** arising out of accidental **injury** or **damage** occurring during the **period of the policy** in the course of **your business activities**. **We** will also pay **costs and expenses** on **your** behalf.

However, **we** will not make any payment on **your** behalf under this **INSURING CLAUSE** in respect of any **claim**:

a) which is covered under **INSURING CLAUSE 1**, or would be covered under **INSURING CLAUSE 1** but for the exhaustion of the **limit of liability** or **aggregate limit of liability** of **INSURING CLAUSE 1**;
b) arising directly or indirectly out of any product.

INSURING CLAUSE 4: PRODUCTS LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** arising out of accidental **injury** or **damage** occurring during the **period of the policy** in the course of **your business activities** in connection with any product. **We** will also pay **costs and expenses** on **your** behalf.

However, **we** will not make any payment on **your** behalf under this **INSURING CLAUSE** in respect of any **claim** which is covered under **INSURING CLAUSE 1**, or would be covered under **INSURING CLAUSE 1** but for the exhaustion of the **limit of liability** or **aggregate limit of liability** of **INSURING CLAUSE 1**.

INSURING CLAUSE 5: TENANTS' LEGAL LIABILITY

We agree to pay on **your** behalf all sums which **you** become legally obliged to pay (including liability for claimants' **costs and expenses**) as a result of any **claim** arising out of accidental **damage** to premises leased to, hired by, on loan to or held in trust by **you** or otherwise in **your** care, custody or control occurring during the **period of the policy** in the course of **your business activities**. **We** will also pay **costs and expenses** on **your** behalf.

However, **we** will not make any payment on **your** behalf under this **INSURING CLAUSE** in respect of any **claim**:

a) which is covered under **INSURING CLAUSE 1**, or would be covered under **INSURING CLAUSE 1** but for the exhaustion of the **limit of liability** or **aggregate limit of liability** of **INSURING CLAUSE 1**;
b) arising directly or indirectly out of any product.

INSURING CLAUSE 6: MEDICAL EXPENSES

We agree to pay **medical expenses** for **injury** caused by an accident occurring during the **period of the policy**:

on premises **you** own or rent;

a) on ways next to premises **you** own or rent; or
b) because of **your business activities**;

provided that:

c) the injured person, at the time of the accident, is not entitled to benefits under any workers compensation or disability benefits law or similar law; and
d) the **medical expenses** are incurred and notified to **us** within one year of the date of the accident; and
e) the injured person submits to examination, at **our** expense, by physicians of **our** choice as often as **we** reasonably require.

We will make these payments regardless of fault.

INSURING CLAUSE 7: COMMERCIAL PROPERTY

We agree to reimburse **you** up to the **amount insured** shown in the Declarations for:

a) the cost of repairing **damage** occurring during the **period of the policy** to **your office** buildings, including landlord's fixtures and fittings, walls, gates and fences, yards, car parks and pavements, piping, ducting, cables, wires and associated control gear and accessories on the premises and extending to the public mains but only to the extent of **your** responsibility;
b) **damage** occurring during the **period of the policy** to **contents of every description** contained in **your office**;
c) **damage** occurring during the **period of the policy** to **contents of every description** kept at the home of **your** directors, officers, partners or **employees** in the course of **your business activities**;
d) **damage** occurring during the **period of the policy** to **contents of every description** temporarily elsewhere, including while in transit;
e) the necessary and reasonable costs **you** incur following **damage** occurring during the **period of the policy** to glass which belongs to **you** or for which **you** are legally responsible for:
   i) temporary boarding up;
   ii) repair of window frames or removal or replacement of fixtures and fittings in the course of replacing the glass;
   iii) replacement lettering or other ornamental work and alarm foil on glass;
f) **damage** occurring during the **period of the policy** to **money** held in the course of **your business activities**:
g) in the **office** during business hours, in transit or in a Bank Night Safe;
h) in the **office** outside business hours in a locked safe;
i) at the home of **your** directors, officers, partners or **employees**;
j) **damage** occurring during the **period of the policy** to the personal belongings of **your employees** or visitors to the **office** provided they are not covered under any other insurance;
k) the reasonable cost of compiling the **documents**, books of account, drawings, card index systems or other records including film, tape, disc, drum, cell or other magnetic recording or storage media for electronic data processing that **you** need to continue **your business activities** if these items have been lost or distorted as a direct result of **damage** covered under this **INSURING CLAUSE**;
l) the costs **you** incur to replace locks and keys necessary to maintain the security of **your office** or safes following theft of keys involving force and violence occurring during the **period of the policy**;
m) the amount of any rent for the **office** which **you** are legally obliged to pay for any period during which the **office** or any part of it is unusable as a result of **damage** covered under this **INSURING CLAUSE**.

We also agree to pay:

a) **costs and expenses** on **your** behalf;
b) compensation as shown in the Declarations if any of **your** directors, officers, partners or **employees** who are aged between 16 and 70 on the Inception Date shown in the Declarations suffers an **injury** in the course of **your business activities** in a robbery or attempted robbery and suffers:
   i) death, **permanent total disablement**, **loss of a limb** or **loss of sight** as a direct result of the **injury** within one year of the date of its occurrence;
   ii) **temporary total disablement**. The compensation for **temporary total disablement** will be the amount shown in the Declarations per week, for a maximum of 104 weeks.

However, **we** will not pay compensation under more than one heading in the Declarations for the same **injury**.

INSURING CLAUSE 8: BUSINESS INTERRUPTION

We agree to reimburse **you** up to the **amount insured** shown in the Declarations for **your loss** of **income**, extra expense, **loss** of **research and development expenditure** , **project delay costs** and **accounts receivable** resulting solely and directly from an interruption to **your business activities** caused by:

a) **insured damage** to **your office** or **contents of every description** or to any other property used by **you** at **your office**;
b) **insured damage** to property in the vicinity of **your office** which prevents or hinders **your** access to **your office**;
c) **insured damage** at the premises of one of **your** suppliers, other than a supplier of water, gas, electricity or telephone services;

d) failure in the supply of water, gas, electricity, or telephone services to **your office** for more than 24 consecutive hours caused by **insured damage** to any property;
e) **your** inability to use **your office** due to restrictions imposed by a public authority following:
   i) a murder or suicide;
   ii) an occurrence of a notifiable human disease;
   iii) **injury** traceable to food or drink consumed at **your office**;
   iv) vermin or pests at **your office**.

HOW MUCH WE WILL PAY

The maximum amount payable by **us** for all **claims**, **losses**, **damage**, **costs and expenses** and **medical expenses** shall not exceed the amounts shown in the Declarations in respect of each **INSURING CLAUSE** unless limited below.

However, solely with respect to any claims under this Policy that shall and must be governed by Quebec law on the scope of our liability, we agree to pay costs and expenses in addition to the Limits of Liability stated in the Declarations.

Where more than one **claim** or **loss** arises from the same original cause or single source or event all such **claims** or **losses** shall be deemed to be one **claim** or **loss** and only one **limit of liability** shall be payable in respect of the **aggregate** of all such **claims** or **losses**.

Where cover is provided under multiple **SECTIONS** of **INSURING CLAUSE 1** only one Limit of Liability shall be payable in respect of that **claim**.

In respect of **INSURING CLAUSES 1** to **5 we** may at any time pay to **you** in connection with any **claim** the amount of the **aggregate limit of liability** or **limit of liability** (after deduction of any amounts already paid). Upon such payment being made **we** shall relinquish the conduct and control of and be under no further liability in connection with such **claim** except for the payment of **costs and expenses** incurred prior to the date of such payment (unless the **aggregate limit of liability** or **limit of liability** is stated to be inclusive of **costs and expenses**).

In respect of **INSURING CLAUSE 7** only:
a) At **our** option, **we** will pay for any **damaged** property on the following basis:
   i) for the **office**, the cost of rebuilding or replacing the **damaged** property;
   ii) for **contents of every description**, the cost of repair or replacement as new.
b) If, at the time the **damage** occurs, the **amount insured** is less than 85% of the total value of the **office** or **contents of every description** insured, the amount **we** will pay will be reduced in the same proportion as the **amount insured** bears to the total value of the **office** or **contents of every description** insured.
c) The **amount insured** for the **office** and **contents of every description** will be adjusted monthly in line with any increase in nationally published indices. **We** will not reduce the **amount insured** without **your** consent.

In respect of **INSURING CLAUSE 8** the amount **we** will pay will be:
a) the difference between **your** actual **income** during the **indemnity period** and the **income** it is estimated **you** would have earned during that period or, if this is **your** first trading year, the difference between **your income** during the **indemnity period** and during the period immediately prior to the **loss**, less any savings resulting from the reduced **costs and expenses you** pay out of **your income** during the **indemnity period**; and
b) any additional **costs and expenses**;
c) any **project delay costs** during the **indemnity period**, including the total value of any milestone payments that were due in the **indemnity period** but will no longer be received by **you** either during the **indemnity period** or at any point in the future due to permanent termination of the project;
d) any **research and development expenditure** irrevocably lost during the **indemnity period**; and
e) any **accounts receivable**, provided **you** keep a record of all amounts owed to **you** and keep a copy of the record away from **your office**.

YOUR DEDUCTIBLE

We shall only be liable for that part of each and every **claim**, **loss** or **medical expenses** (which for the purpose of this clause shall be deemed to include all **costs and expenses** incurred) which exceeds the amount of the Deductible stated in the Declarations. Where more than one **claim**, **loss** or **medical expenses** arises from the same original cause or single source or event all such **claims**, **losses** or **medical expenses** shall be deemed to be one **claim**, **loss** or **medical expenses** and only one Deductible will apply.

If any expenditure is incurred by **us** which by virtue of this clause is **your** responsibility then **you** shall reimburse such amount to **us** on **our** request or where possible **we** will deduct such amount from any payment **we** make to **you**.

DEFINITIONS

1. **"Accounts receivable"**
   means:
   a) all sums due to **you** from customers, provided **you** are unable to effect collection thereof as the direct result of **insured damage** to records of accounts receivable;
   b) interest charges on any loan to offset impaired collections pending repayment of such sums made uncollectible by such **insured damage**;
   c) collection expense in excess of normal collection cost and made necessary because of **insured damage**.

2. **"Administration"**
   means:
   a) counseling **employees**, including their dependants and beneficiaries, with respect to **your employee benefit program**;
   b) handling records in connection with **your employee benefit program**;
   c) effecting enrolment or termination of any **employee's** participation in a plan included in **your employee benefit program**;
   d) interpreting **your employee benefit program**.

3. **"Aggregate limit of liability"**
   means the maximum amount payable as stated in the Declarations by **us** in respect of all **claims**, or in respect of all accidents giving rise to **medical expenses**.

4. **"Amount insured"**
   means the maximum amount payable by **us** as shown in the Declarations in respect of each of **INSURING CLAUSES 7** and **8**. The amount applies to each incident of **loss** or **damage** occurring during the **period of the policy** provided always that after the first incident of **loss** or **damage you** comply with **our** recommendations to prevent any further incidents of **loss** or **damage**.

5. **"Breach of client contract"**
   means **your** unintentional breach of a written contract relating to the performance of **your business activities** for a **client**.

6. **"Business activities"**
   means:

a) in respect of **INSURING CLAUSE 1**, the **business activities** as stated in the Declarations.
b) in respect of all other **INSURING CLAUSES**, the **business activities** as stated in the Declarations and shall include, for the purpose of those **business activities**:
   i) the ownership, repair and maintenance of **your** property;
   ii) provision and management of canteen, social, sports and welfare organisations for the benefit of **your** directors, officers, partners or **employees** and medical, fire fighting, and security services;
   iii) attendance at conferences and tradeshows as either an exhibitor or visitor.

7. **"Claim"**
means a demand received by **you** for **money** or services, including the service of suit or institution of arbitration proceedings. "Claim" shall also mean a threat or initiation of a suit seeking injunctive relief (meaning a temporary restraining order or a preliminary or permanent injunction).

8. **"Client"**
means any third party with whom **you** have a written or implied contract in place for the supply of **your business activities** in return for a fee.

9. **"Contents of every description"**
means the contents of **your office** used in connection with **your business activities** which are owned by **you** or for which **you** are legally responsible, including:
   a) computer and ancillary equipment (including monitors, keyboards, printers and software), television and video equipment, photographic, photocopying, surveying and telecommunications equipment;
   b) **documents**, briefs, manuscripts, plans, business books, computers systems records and programs;
   c) goods held in trust, stock and samples;
   d) wines, spirits and tobacco kept for entertainment purposes;
   e) works of art or precious metals;
   f) fixed glass in windows, doors and fanlights, glass showcases, glass shelves, mirrors and sanitary fixtures and fittings;
   g) heating oil for the **office** contained in fixed tanks in the open at the address shown in the Declarations;
   h) tenant's improvements, decorations, fixtures and fittings including, if attached to the building, external signs, aerials and satellite dishes;
   i) pipes, ducting, cables, wires and associated control equipment at the address shown in the Declarations and extending to the public mains.

   "**Contents of every description**" does not include **money** or the personal belongings of **your employees** or visitors to the **office**.

10. **"Costs and expenses"**
means:
   a) in respect of **INSURING CLAUSES 1** (**SECTIONS A** to **F** only) and 2,
      i) **your** legal **costs and expenses** in the defence or settlement of any **claim** made against **you**, and
      ii) **your** legal **costs and expenses** in the defence of any criminal claim made against **you**, provided that **we** maintain all rights of subrogation to recover such legal **costs and expenses** from any director, officer, partner or **employee** if they are found guilty of such a criminal act, and
      iii) the cost of bonds to release attachments but without any obligation to furnish these bonds, and
      iv) interest on that part of any judgment **we** pay that accrues after entry of the judgment and before **we** have paid, offered to pay, or deposited in court the part of the judgment that is within the **limit of liability**.
   b) in respect of **INSURING CLAUSES 3** to **5**,
      i) **your** legal **costs and expenses** in the defence or settlement of any **claim** made against **you**, and
      ii) the cost of bonds to release attachments but without any obligation to furnish these bonds, and
      iii) interest on that part of any judgment **we** pay that accrues after entry of the judgment and before **we** have paid, offered to pay, or deposited in court the part of the judgment that is within the **limit of liability**.
   c) in respect of **INSURING CLAUSES 7** and **8**, the **costs and expenses** incurred by **you** or on **your** behalf in establishing that **you** have sustained a **loss** or **damage** and the quantum of such **loss** or **damage** or the **costs and expenses** incurred by **you** or on **your** behalf in mitigating any such **loss** or **damage**.
   d) in respect of **INSURING CLAUSE 7** only, the necessary and reasonable **costs and expenses you** incur to remove debris from the premises or the area immediately adjacent, following **damage** covered under this **INSURING CLAUSE**.

   Subject to all **costs and expenses** being incurred with the Claims Managers' written consent (such consent not to be unreasonably withheld).

   If **costs and expenses** are shown in the Declarations to be in addition to the **aggregate limit of liability** or **limit of liability** in respect of any of **INSURING CLAUSES 1** to **5**, and if a payment in excess of the amount of indemnity available hereunder has to be made to dispose of any **claim** or number of **claims**, **our** liability for such **costs and expenses** shall be such proportion thereof as the amount of indemnity available hereunder bears to the amount required to dispose of such **claim** or **claims**.

   Costs and expenses are always included in the **amount insured** in respect of **INSURING CLAUSES 7** and **8**.

11. **"Damage/damaged"**
means direct physical damage to, or destruction of, or loss of possession of, or loss of use of, tangible property. In respect of **INSURING CLAUSES 1**, **3** and **4 damage** does not include damage to or destruction of, or loss of possession of, or loss of use of, or corruption of, data.

12. **"Documents"**
means deeds, wills, agreements, maps, plans, records, books, letters, certificates, forms, computer programmes or information stored, written or punched into card or tape or magnetic discs or tapes or any other data media and documents of any nature whatsoever, whether written, printed or reproduced by any other method (other than bearer bonds, coupons, banks notes, currency notes and negotiable instruments).

13. **"Employee"**
means any person employed by the company named as the Insured in the Declarations, or any **subsidiary**. Employee does not include any director, officer or partner of the company named as the Insured in the Declarations, or any **subsidiary**.

14. **"Employee benefit program"**
means group automobile insurance, group homeowners insurance, group life insurance, group dental insurance, group health insurance, profit sharing plans, pension plans, early retirement offerings, employee investment subscription plans, Workers' Compensation, Unemployment Insurance, Social Security, Disability Benefit Insurance, travel, savings or vacation plans or any similar benefit programs.

15. **"Extra expense"**
means the necessary and reasonable extra **costs and expenses you** incur in order to continue **your business activities** during the **indemnity period**.

16. **"Hacking attack"**
means any malicious or unauthorised electronic attack including but not limited to any fraudulent electronic signature, brute force attack, phishing, denial of service attack, that has been initiated by any **third parties** or by any **employees** and that is designed to damage, destroy, corrupt, overload, circumvent or impair the functionality of computer systems, software and ancillary equipment.

17. **"Income"**
means **your** total income from **your business activities**.

18. **"Indemnity period"**
means the period beginning at the date of the **damage**, or the date the restriction is imposed, and lasting for the period during which **your income** or expenditure is affected as a result of such **damage** or restriction, but for no longer than the number of months shown in the Declarations.

19. **"Injury"**
means:
a) in respect of **INSURING CLAUSE 1** death, bodily **injury**, mental **injury**, illness or disease;
b) in respect of all other **INSURING CLAUSES**:
i) death, bodily **injury**, mental **injury**, illness, disease, shock, mental anguish or humiliation; and
ii) false arrest, detention or imprisonment; and
iii) malicious prosecution; and
iv) wrongful entry into, or eviction of a person from, a room, dwelling or premises that the person occupies.

20. **"Insured damage"**
means **damage** to property provided that:
a) the **damage** is covered under **INSURING CLAUSE 7**; or
b) an insurer has paid the claim, or has agreed to pay the claim, under any other insurance covering such **damage**.

21. **"Intellectual property right"**
means any **intellectual property right** including but not limited to trademarks, trade secrets, broadcasting rights, domain names, metatags and copyrights but does not include patents.

22. **"Limit of liability"**
means the maximum amount payable by **us** as stated in the Declarations in respect of each **claim** or **loss**, or in respect of each accident giving rise to **medical expenses**.

23. **"Loss of a limb"**
means loss by physical separation of a hand at or above the wrist, of a foot at or above the ankle, and includes total and irrecoverable loss of use of a hand, arm or leg.

24. **"Loss of sight"**
means total and irrecoverable loss of sight.

25. **"Loss"**
means direct financial loss sustained by **you**.

26. **"Money"**
means cash, bank and currency notes, cheques, travellers' cheques, postal orders, **money** orders, crossed bankers' drafts, current postage stamps, savings stamps and certificates, trading stamps, gift tokens, customer redemption vouchers, company sales vouchers, credit card counterfoils, travellers tickets and contents of franking machines, all belonging to **you**.

27. **"Medical expenses"**
means reasonable expenses for:
a) first aid administered at the time of an accident;
b) necessary medical, surgical, x ray and dental services, including prosthetic devices;
c) necessary ambulance, hospital, professional nursing and funeral services.

28. **"Office"**
means the **office** space (including any outbuildings) **you** occupy at the address shown in the Declarations as more fully described in the application form.

29. **"Period of the policy"**
means the period between the Inception Date shown in the Declarations and the Expiry Date shown in the Declarations or until the Policy is cancelled in accordance with **GENERAL CONDITION 10** of this Policy.

30. **"Permanent total disablement"**
means disablement which entirely prevents the injured person from attending to any business or occupation for which he is reasonably suited by training, education or experience for 24 calendar months and at the expiry of that period being beyond hope of improvement.

31. **"Project delay costs"**
means any additional **costs and expenses** incurred by **you** as a direct result of a delay to a project, including the interest charges incurred from any reasonable loan required as a result of a delayed milestone payment.

32. **"Product"**
means any tangible property (including containers, packaging, labelling or instructions, but explicitly excluding any software, data, or source code) after it has left **your** custody or control which has been designed, specified, formulated, manufactured, constructed, installed, sold, supplied, distributed, treated, serviced, altered, processed, cleaned, renovated or repaired by **you** or on **your** behalf in the course of **your business activities**.

33. **"Research and development expenditure"**
means **your** expenditure on research and development less the cost of reusable materials consumed for the purposes of the research and development.

34. **"Subsidiary"**
means any company which the company named as the Insured in the Declarations controls through:
a) holding 50% or more of the voting rights, or
b) having the right to appoint or remove 50% or more of its board of directors; or
c) controlling alone, pursuant to a written agreement with other shareholders or members, 50% or more of the voting rights therein.

35. **"Temporary total disablement"**
means disablement which entirely prevents the injured person from attending to his business or occupation.

36. **"Third party"**
means any person or company who is not a director, officer, partner or **employee** of the company named as the Insured in the Declarations, or any **subsidiary**.

37. **"Virus"**
means any malicious software code including but not limited to any logic bomb, Trojan horse or worm that has been introduced by any **third parties** or by any **employees** and that is designed to **damage**, destroy, corrupt, overload, circumvent or impair the functionality of computer systems, software and ancillary equipment.

38. **"We/our/us"**
means the Underwriters named in the Declarations.

39. **"Withheld fees"**
means any contractually due fee that **your client** refuses to pay **you**, but excludes any part of the fee that represents **your** profit or mark-up or liability for taxes.

40. **"Workmanship"**
means any physical **workmanship** in manufacture, fabrication, construction, erection, installation, assembly, alteration, servicing, remediation, repair, demolition or disassembly (including any materials, parts or equipment furnished in connection therewith) by **you**.

41. **"Wrongful act"**
means any act or event the subject of **INSURING CLAUSE 1** of this Policy for which **you** have purchased coverage.

42. **"You/your"**
means:
a) the company named as the Insured in the Declarations, or any **subsidiary**, and
b) any past, present or future **employee**, trainee, director, officer or partner of the company named as the Insured in the Declarations or any **subsidiary**.

EXCLUSIONS

We will not

a) make any payment on **your** behalf for any **claim**, or
b) incur any **costs and expenses**, or
c) reimburse **you** for any **loss**, **damage**, legal expenses, fees or costs sustained by **you**, or
d) pay any **medical expenses**:

EXCLUSIONS RELATING TO OTHER INSURANCES:

1. **Marine and aviation**
arising directly or indirectly from the ownership, possession or use by **you** or on **your** behalf of any aircraft, hovercraft, offshore installation, rig, platform or watercraft.

2. **Auto**
arising directly or indirectly from the ownership, possession or use by **you** or on **your** behalf of any motor vehicle or trailer other than **injury** or **damage**:
a) caused by the use of any tool or plant forming part of or attached to or used in connection with any motor vehicle or trailer;
b) occurring beyond the limits of any carriageway or thoroughfare and caused by the loading or unloading of any motor vehicle or trailer;
c) arising out of the use of any motor vehicle or trailer temporarily in **your** custody or control for the purpose of parking;
provided always that **we** will not make any payment on **your** behalf or incur any **costs and expenses** in respect of any legal liability for which compulsory insurance or security is required by legislation or for which a government or other authority has accepted responsibility.

3. **Project-specific insurance**
arising out of any projects for which **you** have purchased project specific insurance.

4. **Product guarantee**
for costs incurred in the repair, alteration, reinstatement, inspection, reconditioning or replacement of any product or part thereof and any financial **loss** consequent upon the necessity for such repair, alteration, reinstatement, inspection, reconditioning or replacement, other than in respect of **INSURING CLAUSE** 1 when **you** are legally obliged to pay these sums to a **client**.

5. **Product recall**
arising directly or indirectly from the recall of any product or part thereof except for **claims** made under **INSURING CLAUSE** 1 where **you** are legally liable for these costs to a third party as the direct result of a **wrongful act** committed or alleged to have been committed by **you**.

6. **Employment practices**
arising out of or resulting from any employer-**employee** relations, policies, practices, acts, omissions, any actual or alleged refusal to employ any person, or misconduct with respect to **employees**.

7. **Employers' liability**
arising directly or indirectly out of **injury** to **your** directors, officers, partners or **employees**.

However, this **EXCLUSION** shall not apply to **employees** on whose behalf contributions are required to be made by **you** under the provisions of any Workers' Compensation Law in respect of whom liability has been denied by any Workers' Compensation authority.

8. **Directors' and Officers'**
arising out of any personal liability incurred by **your** directors or officers when they are acting in that capacity or managing **you**, or arising from any statement, representation or information regarding **your** business contained within any accounts, reports or financial statements.

9. **Double insurance**
for which **you** are entitled to indemnity under any other insurance except for:
a) any additional sum which is payable over and above such other insurance, or
b) any contribution that **we** are obliged to make by law and that contribution shall be in proportion to the respective limits of liability or **amounts insured** of the Policies.

EXCLUSIONS RELATING TO THE CONDUCT OF YOUR BUSINESS:

10. **Benefit laws**
arising directly or indirectly out of **your** failure to comply with the mandatory provisions of any law concerning workers compensation, unemployment insurance, social security, disability benefits or pension benefits.

11. **Circumstances known at inception**
arising out of any circumstances or occurrences which could give rise to a **claim**, **loss** or **damage** under this Policy or any accidents giving rise to **medical expenses** of which **you** are aware, or ought reasonably to be aware, prior to the Inception Date of this Policy, whether notified under any other insurance or not.

12. **Computer failure**
in respect of **INSURING CLAUSES 7** and **8** only, arising directly or indirectly from **loss** or distortion of **your** data or **damage** to **your** electrical or mechanical plant resulting from a failure of **your** computer or ancillary equipment (including monitors, keyboards, printers or software), television or video equipment, photographic, photocopying, surveying or telecommunications equipment. However, **we** will reimburse **you** up to the **amount insured** for **damage** occurring during the **period of the policy** to **your office** computer and ancillary equipment, but only if **your office** computer and ancillary equipment is subject to a manufacturer's guarantee or a maintenance contract providing free parts and labour in the event of a breakdown.

13. **Contractual fines and penalties**
for fines and penalties arising from **your** breach of contract, including any liquidated damages, service credits or associated penalties arising from **your** failure to perform under a service level agreement

14. **Employee benefit program advice**
arising directly or indirectly from:
a) advice given to any person to participate or not to participate in any plan included in your employee benefit program;
b) the failure of any investment to perform as represented by you.

15. **ERISA**
arising out of or resulting from **your** acts related to any pension, healthcare, welfare, profit sharing, mutual or investment plans, funds or trusts; or any violation of any provision of the Employee Retirement Income Security Act of 1974, or any amendment to the Act or any violation of any regulation, ruling or order issued pursuant to the Act.

16. **Failure to ensure feasibility of contracts**
arising from any contract where before entering into or amending the contract **you** failed to take reasonable steps to ensure that **you** could fulfil all **your** obligations in accordance with the terms of the contract.

17. **Faulty workmanship**
arising from **damage** to **your** property or **office** caused directly or indirectly by misuse, inadequate or inappropriate maintenance, faulty **workmanship**, defective design, the use of faulty materials or whilst being cleaned, worked on or maintained.

18. **Hazardous devices**
arising directly or indirectly from any product which with **your** knowledge is intended for incorporation into the structure,

machinery or controls of any aircraft, other aerial device, military vehicle, hovercraft, waterborne craft or any medical equipment.

19. **Legal Action**
where action for damages is brought in a court of law outside the territories specified in the Declarations, or where action is brought in a court of law within those territories to enforce a foreign judgement whether by way of reciprocal agreement or otherwise.

20. **Limiting recovery rights**
arising directly or indirectly out of **your** failure to take reasonable steps to ensure that **our** rights of recovery against any third party are not unduly restricted or financially limited by a specific term in any contract or agreement.

21. **Patents**
arising out of the actual or alleged infringement of any patent or inducing the infringement of any patent.

22. **Retroactive Date**
in respect of **INSURING CLAUSES 1** and **2** only, arising out of any actual or alleged **wrongful act** or negligent act, error or omission committed before the date specified as the Retroactive Date in the Declarations.

23. **RICO**
for or arising out of any actual or alleged violation of the Organised Crime Control Act of 1970 (commonly known as the Racketeer Influenced and Corrupt Organisation Act or RICO), as amended, or any regulation promulgated thereunder or any similar federal, state or local law, whether such law is statutory, regulatory or common law.

24. **SEC**
for or arising out of the actual or alleged violation of the Securities Act of 1933, the Securities Exchange Act of 1934, or any similar state or federal law, or any amendment to the above laws or any violation of any order, ruling or regulation issued pursuant to the above laws.

25. **Unjust enrichment**
in respect of **INSURING CLAUSE 1** only, for that part of any **claim** that results in **you** being in a better financial position as a direct result of **your wrongful act** than **you** would have been if **you** had not committed the **wrongful act**.

26. **Water ingress (applicable to British Columbia only)**
arising out of, or relating directly or indirectly to, in whole or in part, the **infiltration of precipitation** into the **building envelope** of a building located in the Province of British Columbia, or into a **multi-unit building** located in the Province of British Columbia.

    For the purposes of this exclusion the following definitions are added to the Policy:

    Multi-unit building means a building containing more than one unit, whether that unit is used for residential, industrial or any other purpose.

    Building envelope means the assemblies, components, and materials of a building which are intended to separate and protect the interior space of a building from the adverse effects of exterior climactic conditions.

    Infiltration of precipitation means, but is not limited to, the actual, alleged, threatened, or possible infiltration, migration, presence, accumulation, condensation or dispersal of water or moisture on, in, or into the **building envelope**.

27. **Wilful or dishonest acts of directors**
in respect of **INSURING CLAUSES 1** and **2** only, arising out of any wilful, malicious, reckless or dishonest act or omission by any director, partner or officer of the company named as the Insured in the Declarations or any **subsidiary**, unless such person had already ceased to be a director, partner or officer of the company named as the Insured in the Declarations and all **subsidiaries** at the time of their first wilful, malicious, reckless or dishonest act or omission, or unless specifically covered under **INSURING CLAUSE 1 SECTION A (e)**. **We** will not provide any cover for any director, partner or officer of the company named as the Insured in the Declarations or any **subsidiary** who commits, condones or ignores any dishonesty.

GENERAL INSURANCE EXCLUSIONS:

28. **Antitrust**
for or arising out of any actual or alleged antitrust violation, restraint of trade, unfair competition, false, deceptive or unfair trade practices, violation of consumer protection laws or false or deceptive advertising unless insurable under the applicable law.

29. **Asbestos**
arising from or contributed to by the manufacturing, mining, use, sale, installation, removal, distribution of or exposure to asbestos, materials or **products** containing asbestos, or asbestos fibres or dust, unless arising directly from a **wrongful act** committed by **you**:
    a) on or after 1st January 1990, or
    b) on or after the date specified as the Retroactive Date in the Declarations,
    c) whichever is the later, in the course of **your business activities**.

30. **Associated companies**
    a) in respect of any **claim** made by any company firm or partnership in which the company named as the Insured in the Declarations has an executive or financial interest, unless such **claim** emanates from an independent **third party**; or
    b) in respect of any **claim** made by any company firm partnership or individual which has an executive or financial interest in the company named as the Insured in the Declarations or any **subsidiary**, unless such **claim** emanates from an independent **third party**; or
    c) arising out of or resulting from any of **your** activities as a trustee, partner, officer, director or **employee** of any **employee** trust, charitable organization, corporation, company or business other than that of the company named as the Insured in the Declarations or any **subsidiary**; or
    d) in respect of any **claim** made by or on behalf of the company named as the Insured in the Declarations or any **subsidiary**.

31. **Earthquake**
in respect of **INSURING CLAUSES 7** and **8** only, caused by earthquake, except for:
    a) ensuing **loss** or **damage** which results directly from fire, explosion, smoke or leakage from fire protective equipment; or
    b) ensuing **damage** to **contents of every description** while in transit.

32. **Electromagnetic fields**
directly or indirectly arising out of, resulting from or contributed to by electromagnetic fields, electromagnetic radiation, electromagnetism, radio waves or noise.

33. **Flood**
in respect of **INSURING CLAUSES 7** and **8** only, caused by flood, including waves, tides, tidal waves, or the rising of, the breaking out, or the overflow, of any body of water whether natural or manmade, but this **EXCLUSION** does not apply to:
    a) ensuing **loss** or **damage** which results directly from fire, explosion, smoke or leakage from fire protective equipment; or
    b) ensuing **damage** to **contents of every description** while in transit.

34. **Fines**
for fines, penalties, civil or criminal sanctions and for punitive, multiple or exemplary damages unless insurable under the applicable law.

35. **Insolvency**
arising out of or relating directly or indirectly from **your** insolvency or bankruptcy, or the insolvency or bankruptcy of any third party. Furthermore, no coverage is provided under **INSURING CLAUSE 8** if **you** become insolvent or bankrupt.

36. **Land or water**
arising directly or indirectly from **damage** to land or water within or

below the boundaries of any land or premises presently or at any time previously owned or leased by **you** or otherwise in **your** care, custody or control.

37. **Miscellaneous property exclusions**
in respect of **INSURING CLAUSES 7** and **8** only, arising directly or indirectly from:
a) wear and tear, inherent defect, rot, vermin or infestation, or any gradually operating cause;
b) dryness or humidity, being exposed to light or extreme temperatures, unless the **damage** is caused by storm or fire;
c) coastal or river erosion;
d) theft from an unattended vehicle unless the item is out of sight;
e) frost, other than **damage** due to water leaking from burst pipes forming part of the permanent internal plumbing provided the **office** is occupied and in use;
f) arising directly or indirectly from unexplained **loss** or disappearance or inventory shortage of **your** property;
g) a **hacking attack** or **virus**.

38. **Nuclear**
arising directly or indirectly from or contributed to by :
a) ionising radiations or contamination by radioactivity from any nuclear fuel or from any nuclear waste from the combustion of nuclear fuel;
b) the radioactive, toxic, explosive or other hazardous properties of any explosive nuclear assembly or nuclear component thereof.

39. **Pollution**
arising directly or indirectly out of :
a) pollution or contamination of the atmosphere, or of any water, land, buildings or other property;
b) any enforcement action in connection with the containment, clean-up, removal or treatment of such pollution or contamination;
but this **EXCLUSION** shall not apply in respect of :
i) **INSURING CLAUSE 1**, **SECTION D**; or
ii) **INSURING CLAUSES 7** and **8** to the backing up of sewers, sumps, septic tanks or drains.

40. **Toxic mould / fungus**
arising directly or indirectly from any loss, **injury**, **damage**, costs or expenses, including, but not limited to, losses, **damage**, costs or expenses related to, arising from or associated with clean-up, remediation, containment, removal or abatement, caused directly or indirectly, in whole or in part, by:
a) any **fungus**, **mould**, mildew or yeast, or
b) any **spore** or toxins created or produced by or emanating from such **fungus**, **mould**, mildew or yeast, or
c) any substance, vapour, gas, or other emission or organic or inorganic body or substance produced by or arising out of any **fungus**, **mould**, mildew or yeast, or
d) any material, product, building component, building or structure, or any concentration of moisture, water or other liquid within such material, product, building component, building or structure, that contains, harbours, nurtures, or acts as a medium for any **fungus**, **mould**, mildew, yeast or **spore** or toxins emanating therefrom,

regardless of any other cause, event, material, product or building component that contributed concurrently or in any sequence to that loss, **injury**, **damage**, cost or expense.

However, this **EXCLUSION** shall not apply in respect of **INSURING CLAUSE 1** where the **loss**, **injury**, **damage**, costs or expenses arose directly from a **wrongful act** committed by **you** in the course of **your business activities**.

For the purposes of this **EXCLUSION** the following definitions are added to the Policy:

Fungus includes, but is not limited to, any plants or organisms belonging to the major group Fungi, lacking chlorophyll, and including **moulds**, rusts, mildews, smuts and mushrooms.

Mould includes, but is not limited to, any superficial growth produced on damp or decaying organic matter or on living organisms, and **fungi** that produced moulds.

Spore means any dormant or reproductive body produced by or arising from or emanating out of any **fungus**, **mould**, mildew, plants, organisms or microorganisms.

41. **Trade Debt**
arising out of or in connection with any trading losses or trading liabilities incurred by any business managed or carried on by **you**, or any **loss** of **your** profit arising from the **loss** of any **client**, account or business.

42. **War and terrorism**
directly or indirectly caused by, resulting from or in connection with any of the following regardless of any other cause or event contributing concurrently or in any other sequence to the **claim**, **loss**, **damage**, **costs and expenses** or **medical expenses**;
a) war, invasion, acts of foreign enemies, hostilities or warlike operations (whether war be declared or not), civil war, rebellion, insurrection, civil commotion assuming the proportions of or amounting to an uprising, military or usurped power; or
b) any **act of terrorism**.

For the purpose of this **EXCLUSION** an **act of terrorism** means an act, including but not limited to the use of force or violence or the threat thereof, of any person or groups of persons, whether acting alone or on behalf of or in connection with any organisations or governments, committed for political, religious, ideological or similar purposes including the intention to influence any government or to put the public, or any section of the public, in fear.

This **EXCLUSION** also excludes **claims**, **losses**, **damage**, **costs and expenses** or **medical expenses** of whatsoever nature directly or indirectly caused by, resulting from or in connection with any action taken in controlling, preventing, suppressing or in any way relating to a) or b) above.

This **EXCLUSION** does not apply to any **claim** or **loss** arising directly from a **hacking attack** or **virus**.

GENERAL CONDITIONS

1. **What you must do in the event of a claim or loss**
Should any director, partner, or senior executive officer of the company named as the Insured in the Declarations and any **subsidiary** become aware of any **claim**, **loss** or **damage** or of any situation that could give rise to a **claim** or **loss** or should an allegation, complaint or **claim** be made or intimated against **you**, the following obligations must be complied with by **you**:
a) **You** must not admit liability for or settle or make or promise any payment in respect of any **claim**, **loss** or **damage** which may be covered under this Policy. Neither must **you** incur any **costs** or **expenses** in connection with such a **claim**, **loss** or **damage** without **our** written consent. However, **you** should arrange for any urgent repairs following **damage** covered under **INSURING CLAUSE 7** to be done immediately. Before any other repair work begins **we** have the right to inspect **your damaged** property. **We** will notify **you** if **we** intend to do this.
b) The Claims Managers, as specified in the Declarations, must be notified as soon as is reasonably possible if during the **period of the policy**:
i) **you** suffer any **loss** or **damage** that could be covered by this Policy or any allegation, complaint or **claim** is made or intimated against **you**, whether verbal or made in writing.
ii) any director, partner, or senior executive officer of the company named as the Insured in the Declarations and any **subsidiary** become aware of the intention of any person to make a complaint allegation or **claim** against **you**, whether verbal or in writing. Once notice has been

made to **us**, **we** will regard any subsequent **claim** that may arise as notified under this Policy.

iii) **you** become aware of an action of yours that could give rise to a **loss**, allegation, complaint or **claim** being made or intimated against **you**. Once notice has been made **we** will regard any subsequent **claim** that may arise as notified under this Policy.

iv) **you** discover reasonable cause for suspicion of fraud or dishonesty whether this could give rise to a **claim** under this Policy or not and **we** shall not be liable under this Policy for any **claim** or **loss** sustained in consequence of any fraudulent or dishonest act or omission committed after the date of such discovery.

We have nominated the Claims Managers to accept notice on **our** behalf.

Due to the nature of the coverage offered by this Policy, any unreasonable delay by any director, partner, or senior executive officer of the company named as the Insured in the Declarations and any **subsidiary** in notifying the Claims Managers of (i), (ii), (iii) or (iv) above could lead to the size of the **claim**, **loss** or **damage** increasing or to **our** rights being restricted. **We** shall not be liable for that portion of any claim that is due to any unreasonable delay in any director, partner, or senior executive officer of the company named as the Insured in the Declarations and any **subsidiary** notifying the Claims Managers of any **claim**, **loss** or **damage** in accordance with this **GENERAL CONDITION**.

c) **We** will expect **you** to provide **us** with full and accurate information about any matter that **you** notify to **us** under **your** obligations set out above. Once notice has been made **you** must give the Claims Managers all the assistance and information that is reasonably required. **You** must follow their advice and do anything that they reasonably require **you** to do to avoid, minimise, settle or defend any **claim**, **loss** or **damage**.

If **you** think a crime has been committed **you** must report it to the appropriate law enforcement authorities. **You** must also permit the Claims Managers and any other parties that are appointed by the Claims Managers to notify the appropriate law enforcement authorities of any **claim**, **loss** or **damage** where this action is deemed necessary, and **you** must comply with the advice given by such authorities.

If any of **your** computer or ancillary equipment is lost or stolen while it is temporarily removed from the **office**, **we** will not make any payment unless **you** report the **loss** to the police within 48 hours after **you** become aware of it.

2. **Continuous cover**

If **you** have neglected, through error or oversight only, to report a **claim** made against **you** during the period of a previous renewal of this Policy issued to **you** by **us**, then provided that **you** have maintained uninterrupted insurance of the same type with **us** since the expiry of that earlier Policy, then, notwithstanding **EXCLUSION 11**, **we** will permit the matter to be reported under this Policy and will indemnify **you**, provided that:

a) the indemnity will be subject to the applicable **aggregate limit of liability** or **limit of liability** of the earlier Policy under which the matter should have been reported or the **aggregate limit of liability** or **limit of liability** of the current Policy, whichever is the lower;

b) **we** may reduce the indemnity entitlement by the monetary equivalent of any prejudice which has been suffered as a result of the delayed notification;

c) the indemnity will be subject in addition, to all of the terms, **CONDITIONS**, **DEFINITIONS** and **EXCLUSIONS**, other than the **aggregate limit of liability** or **limit of liability**, contained in this current Policy.

3. **Fraudulent claims**

If **you** notify **us** of any claim knowing that claim to be false or fraudulent in any way, **we** shall have no responsibility to pay that claim or any other claims under this insurance and the Policy will be treated as if it had not been effected.

4. **Agreement to pay claims**

We have the right and duty to take control of and conduct in **your** name the investigation settlement or defence of any **claim**. **We** shall also pay on **your** behalf **costs and expenses** incurred with **our** prior written consent (subject to the Limits of Liability and applicable Deductible shown in the Declarations) provided that **we** shall not

a) pay for the **costs and expenses** of any part of a **claim** that is not covered by this Policy.

b) incur any **costs and expenses** in the defence of any **claim** unless there is a reasonable prospect of success, taking into account the commercial considerations of the costs of defence.

We shall always endeavour to settle any **claim** through negotiation, mediation or some other form of alternative dispute resolution and shall pay on **your** behalf the amount so agreed by **us** and the claimant. If **we** cannot settle by such means, **we** shall pay the amount which **you** are found liable to pay either in court or through arbitration proceedings, subject always to the **limit of liability** shown in the Declarations.

If **you** refuse to consent to a settlement that **we** recommend and that the claimant will accept, **you** must then defend, investigate or settle the **claim** at **your** own expense. As a consequence of **your** refusal, **our** liability for any **claim** shall not be more than the amount that **we** could have settled the **claim** for had **you** consented, plus any **costs and expenses** incurred prior to the date of such refusal.

5. **Innocent non-disclosure**

We will not seek to avoid the Policy or reject any **claim** on the grounds of non-disclosure or misrepresentation except where the non-disclosure or misrepresentation was reckless or fraudulent or **you** failed to conduct a full inquiry prior to providing the information that forms the basis of this insurance. In the event that **we** seek to avoid the Policy or reject any **claim** on this basis the burden of proving otherwise rests solely with **you**.

6. **Your duty to advise us of changes**

If **you** become aware that any of the information that **you** have given **us** in the Application Form or elsewhere in connection with **your** application for this insurance has materially changed then **you** must advise **us** as soon as is practicable. In this event, **we** reserve the right to amend the terms, conditions or premium of the Policy.

7. **Risk management conditions**

If **we** attach any additional conditions to **your** Policy regarding any risk survey or risk management timetable or any other similar conditions then it is **your** responsibility to ensure that these conditions are complied with by the deadlines shown in the conditions.

8. **Our rights of recovery**

If any payment is made under this Policy in respect of a **claim**, **loss** or **damage** and there is available to **us** any of **your** rights of recovery against any other party then **we** maintain all such rights of recovery. **We** shall not exercise these rights against any past, present or future **employee**, director, officer or partner of the company named as the Insured in the Declarations or any **subsidiary**, unless such payment is in respect of any wilful, malicious or dishonest acts or omissions. **You** must do nothing to impair any rights of recovery. At **our** request **you** will bring proceedings or transfer those rights to **us** and help **us** to enforce them. Any recoveries shall be applied as follows:

a) first, to **us** up to the amount of **our** payment on **your** behalf including **costs and expenses**;

b) then to **you** as recovery of **your** Deductible or other amounts paid as compensation or **costs and expenses**.

9. **Waiver of subrogation**

Notwithstanding **GENERAL CONDITION 8** above **we** agree to waive **our** rights of subrogation against a responsible third party **client** of yours but only if **you** and **your client** have entered into a contract that contains a provision requiring **us** to do this.

10. **Cancellation**
This Policy may be cancelled:
a) by **you** at any time on request; or
b) by **us** if **we** give **you** 30 days written notice, or
c) by **us** if **we** give **you** 15 days written notice, should any amount in default not be paid within 15 days of the due date shown in the Debit Note that accompanies this Policy.

If **you** give **us** notice of cancellation in accordance with a) above, the earned Premium shall be computed at pro rata to the number of days that the Policy is in effect subject to a minimum amount of 30% of the Premium.

If **we** give **you** notice of cancellation in accordance with b) or c) above, the Premium shall be computed at pro rata to the number of days that the Policy is in effect.

The Policy Administration Fee shall be deemed fully earned upon inception of the Policy.

11. **Prior subsidiaries**
In respect of **INSURING CLAUSE 1** only, should an entity cease to be a **subsidiary** after the Inception Date of this Policy, cover in respect of such entity shall continue as if it was still a **subsidiary**, until the termination of this Policy, but only in respect of any **claim** or **loss** that arises out of any act, error or omission committed by that entity prior to the date that it ceased to be a **subsidiary**.

12. **Mergers and acquisitions**
During the **period of the policy**, if the company named as the Insured in the Declarations or any **subsidiary**
a) purchases assets or acquires liabilities from another entity in an amount greater than 10% of the assets of the company named as the Insured in the Declarations as listed in its most recent financial statement; or
b) acquires another entity whose annual revenues are more than 10% of the annual revenues of the company named as the Insured in the Declarations for their last completed financial year;
then **you** shall have no coverage under this Policy for any **claim**, **loss** or **damage** that arises directly or indirectly out of the purchased or acquired entity unless the company named as the Insured in the Declarations gives **us** written notice prior to the purchase or acquisition, obtains **our** written consent to extend coverage to such additional entities, assets or exposures, and agrees to pay any additional premium required by **us**.

If during the **period of the policy** the company named as the Insured in the Declarations consolidates or merges with or is acquired by another entity, then all coverage under this Policy shall terminate at the date of the consolidation, merger or acquisition unless **we** have issued an endorsement extending coverage under this Policy, and the company named as the Insured in the Declarations has agreed to any additional premium and terms of coverage required by **us**.

13. **Extended reporting period**
In respect of **INSURING CLAUSES 1** and **2** only, an Extended Reporting Period of 60 days following the Expiry Date as shown in the Declarations shall be automatically granted hereunder at no additional premium. Such Extended Reporting Period shall cover **claims** first made and reported to **us** during this 60 day Extended Reporting Period but only in respect of any act, error or omission committed prior to the date of cancellation or non-renewal, and subject to all other terms, conditions and exclusions of the policy. No **claim** shall be accepted by **us** in this 60 day Extended Reporting Period if **you** are entitled to indemnity under any other insurance, or would have been entitled to indemnity under such insurance but for the exhaustion thereof.

14. **Optional extended reporting period**
In respect of **INSURING CLAUSES 1** and **2** only, in the event of:
a) cancellation or non-renewal of this Policy by **us**, or
b) cancellation or non-renewal of this Policy by **you** because **you** have ceased to trade as the direct result of the retirement or death of all of **your** directors, officers or partners;

then **you** shall have the right, upon payment of the Optional Extended Reporting Period Premium shown in the Declarations in full and not proportionally or otherwise in part, to have issued an endorsement providing a 365 day Optional Extended Reporting Period from the cancellation or non-renewal date. Such Optional Extended Reporting Period shall cover **claims** first made against the company named as the Insured in the Declarations or any **subsidiary** and notified to **us** during this Optional Extended Reporting Period but only in respect of any **claim** arising out of any act, error or omission committed prior to the date of cancellation or non-renewal, and subject to all other terms, conditions and exclusions of the policy.

In order for **you** to invoke the Optional Extended Reporting Period option, the payment of the Optional Extended Reporting Period Premium shown in the Declarations for this Optional Extended Reporting Period must be paid to **us** within 15 days of the date of the non-renewal or cancellation.

At the commencement of this Optional Extended Reporting Period the entire premium shall be deemed earned and in the event that **you** terminate the Optional Extended Reporting Period for any reason prior to its natural expiration, **we** will not be liable to return any premium paid.

The right to the Extended Reporting Period or the Optional Extended Reporting Period shall not be available to **you** where:
a) Cancellation or non-renewal by **us** is due to non-payment of premium, or
b) Cancellation or non-renewal by **us** is due to **your** failure to pay such amounts in excess of the applicable **Limit of Liability** or within the amount of the applicable Deductible as is required by this Policy in the payment of **claims**.

At the renewal of this Policy, **our** quotation of different premium, Deductible or **Limit of Liability** or changes in policy language shall not constitute non-renewal by **us** for the purposes of granting this Optional Extended Reporting Period.

In no event shall the granting of the Extended Reporting Period or the Optional Extended Reporting Period increase **our Limit of Liability**, including **costs and expenses**, as shown in the Declarations.

15. **Choice of law**
This Policy shall be interpreted under, governed by and construed in all respects in accordance with the law of the jurisdiction of the place of registration of the company named as the Insured in the Declarations and for this purpose, we and you agree to submit to the exclusive jurisdiction of the courts within the territorial limits and jurisdiction of the place of registration of the company named as the Insured in the Declarations.

In any action to enforce our obligations under this Policy we can be designated or named as "Lloyd's Underwriters" and such designation shall be binding on Lloyd's Underwriters liable under this Policy as if we had each been individually named as defendant. Service of such proceedings may validly be made upon the Attorney In Fact in Canada for Lloyd's Underwriters, whose address for such service is 1155, rue Metcalfe, Suite 2220, Montreal, Quebec, H3B 2V6.

STATUTORY CONDITIONS

1. **Misrepresentation**
If a person applying for insurance falsely describes the property to the prejudice of the Insurer, or misrepresents or fraudulently omits to communicate any circumstance that is material to be made known to the Insurer in order to enable it to judge of the risk to be undertaken, the contract is void as to any property in relation to which the misrepresentation or omission is material.

2. **Property of others**
Unless otherwise specifically stated in the contract, the Insurer is not liable for loss or damage to property owned by any person other than the Insured, unless the interest of the Insured therein is stated in the contract.

3. **Change of interest**
The Insurer is liable for loss or damage occurring after an authorized assignment under the Bankruptcy Act or change of title by succession, by operation of law, or by death.

4. **Material change**
Any change material to the risk and within the control and knowledge of the Insured avoids the contract as to the part affected thereby, unless the change is promptly notified in writing to the Insurer or its local agent; and the Insurer when so notified may return the unearned portion, if any, of the premium paid and cancel the contract, or may notify the Insured in writing that, if he desires the contract to continue in force, he must within fifteen (15) days of the receipt of the notice, pay to the Insurer an additional premium; and in default of such payment the contract is no longer in force and the Insurer shall return the unearned portion, if any, of the premium paid.

5. **Termination**
1. This contract may be terminated:
   a) by the Insurer giving to the Insured fifteen (15) days' notice of termination by registered mail or five (5) days' written notice of termination personally delivered;
   b) by the Insured at any time on request.
2. Where this contract is terminated by the Insurer:
   a) the Insurer shall refund the excess of premium actually paid by the Insured over the pro rata premium for the expired time, but in no event, shall the pro rata premium for the expired time be less than any minimum retained premium specified; and
   b) the refund shall accompany the notice unless the premium is subject to adjustment or determination as to amount, in which case the refund shall be made as soon as practicable.
3. Where this contract is terminated by the Insured, the Insurer shall refund as soon as practicable the excess of the premium actually paid by the Insured over the short rate premium for the expired time, but in no event shall the short rate premium for the expired time, be deemed to be less than any minimum retained premium specified.
4. The refund may be made by money, postal or express company money order or cheque payable at par.
5. The fifteen (15) days mentioned in clause (1) (a) of this condition commences to run on the day following the receipt of the registered letter at the post office to which it is addressed.

6. **Requirements after loss**
1. Upon the occurrence of any loss of or damage to the insured property, the Insured shall, if the loss or damage is covered by the contract, in addition to observing the requirements of Conditions 9, 10 and 11:
   a) forthwith give notice thereof in writing to the Insurer;
   b) deliver as soon as practicable to the Insurer a proof of loss verified by a statutory declaration,
      i) giving a complete inventory of the destroyed and damaged property and showing in detail quantities, costs, actual cash value and particulars of amount of loss claimed,
      ii) stating when and how the loss occurred, and if caused by fire or explosion due to ignition, how the fire or explosion originated, so far as the Insured knows or believes,
      iii) stating that the loss did not occur through any wilful act or neglect or the procurement, means or connivance of the Insured,
      iv) showing the amount of other insurance and the names of other Insurers,
      v) showing the interest of the Insured and of all others in the property with particulars of all liens, encumbrances and other charges upon the property,
      vi) showing any changes in title, use, occupation, location, possession or exposures of the property since the issue of the contract,
      vii) showing the place where the property insured was at the time of loss,
   c) if required give a complete inventory of undamaged property and showing in detail quantities, cost, actual cash value;
   d) if required and if practicable, produce books of account, warehouse receipts and stock lists, and furnish invoices and other vouchers verified by statutory declaration, and furnish a copy of the written portion of any other contract.
2. The evidence furnished under Clauses 1 (c) and (d) of this condition shall not be considered proofs of loss within the meaning of Statutory Conditions 12 and 13.

7. **Fraud**
Any fraud or wilfully false statement in a statutory declaration in relation to any of the above particulars, vitiates the claim of the person making the declaration.

8. **Who may give notice and proof**
Notice of loss may be given, and proof of loss may be made, by the agent of the Insured named in the contract in case of absence or inability of the Insured to give the notice or make the proof, and absence or inability being satisfactorily accounted for, or in the like case, or if the Insured refuses to do so, by a person to whom any part of the insurance money is payable.

9. **Salvage**
1. The Insured in the event of any loss or damage to any property insured under the contract, shall take all reasonable steps to prevent further damage to any such property so damaged and to prevent damage to other property insured hereunder including, if necessary, its removal to prevent damage or further damage thereto.
2. The Insurer shall contribute pro rata towards any reasonable and proper expenses in connection with steps taken by the Insured and required under subparagraph 1 of this condition according to the respective interests of the parties.

10. **Entry, control, abandonment**
After loss or damage to insured property, the Insurer has an immediate right of access and entry by accredited agents sufficient to enable them to survey and examine the property, and to make an estimate of the loss or damage, and after the Insured has secured the property, a further right of access and entry sufficient to enable them to make appraisal or particular estimate of the loss or damage, but the Insurer is not entitled to the control or possession of the insured property, and without the consent of the Insurer there can be no abandonment to it of insured property.

11. **Appraisal**
In the event of disagreement as to the value of the property insured, the property saved or the amount of the loss, those questions shall be determined by appraisal as provided under the Insurance Act before there can be any recovery under this contract whether the right to recover on the contract is disputed or not, and independently of all other questions. There shall be no right to an appraisal until a specified demand therefor is made in writing and until after proof of loss has been delivered.

12. **When loss payable**
The loss is payable within sixty (60) days after completion of the proof of loss, unless the contract provides for a shorter period.

13. **Replacement**
1. The Insurer, instead of making payment, may repair, rebuild, or replace the property damaged or lost, giving written notice of its intention so to do within thirty days after receipt of the proofs of loss.
2. In that event the Insurer shall commence to so repair, rebuild or replace the property within forty-five (45) days after receipt of the proofs of loss, and shall thereafter proceed with all due diligence to the completion thereof.

14. **Action**
Every action or proceeding against the Insurer for the recovery of any claim under or by virtue of this contract is absolutely barred unless commenced within one year next after the loss or damage occurs.

15. **Notice**
Any written notice to the Insurer may be delivered at, or sent by registered mail to, the chief agent or head office of the Insurer in the

PRIVACY NOTICE

By purchasing insurance from certain Underwriters at Lloyd's, London ("Lloyd's"), a customer provides Lloyd's with his or her consent to the collection, use and disclosure of personal information, including that previously collected, for the following purposes:

- the communication with Lloyd's policyholders
- the underwriting of policies
- the evaluation of claims
- the detection and prevention of fraud
- the analysis of business results
- purposes required or authorised by law

For the purposes identified, personal information may be disclosed to Lloyd's related or affiliated organisations or companies, their agents/mandataries, and to certain non-related or unaffiliated organisations or companies.

Further information about Lloyd's personal information protection policy may be obtained from the customer's broker or by contacting Lloyd's on 514 861 8361 or through info@lloyds.ca.

ONTARIO COMMERCIAL LIABILITY NOTICE

Notice to Insureds:

Pursuant to the
Freedom Of Information And Protection Of Privacy Act, R.S.O. 1990, c.F.31 (as amended)

Important
The notice below applies to insurance contracts containing non-automobile legal liability coverages in provinces where statistical data relating to such contracts must be reported to the Superintendent of Insurance.

Legal authority for collection
Insurance Act, R.S.O. 1990, c.I.8, section 101(1).

Principal purpose for which personal informtion is intended to be used Information collected by insurers from insureds or supplied to insurers pertaining to the attached document will be used:

- to compile aggregate statistical data to be used in monitoring trends in the insurance industry;
- to develop statistical exhibits to be used in monitoring the insurance industry;
- to respond to requests for customized statistical information on the insurance industry;
- to respond to inquiries on statistical information made to Office of the Superintendent of Insurance; and
- to use and disclose such information for purposes which are consistent with the previous clauses.

The Public Official who can answer questions about the collection is:
Manager, Statistical Services
Financial Services Commission of Ontario
5160 Yonge Street, 17th Floor
Box 85
North York, Ontario M2N 6L9
Telephone: (416) 250-7250
Fax: (416) 590-7070

FOI (11/1999)

COMPLAINTS PROCEDURE

If you have a complaint with any aspect of your Lloyd's insurance, please refer to the broker/agent who arranged your policy for you.

OR

You may contact the General Insurance OmbudService (GIO) who will contact Lloyd's on your behalf. The GIO can be reached at:

GIO - Atlantic Provinces
(902) 429-2730
Toll-free: 1-800-565-7189
Website: www.gio-scad.org

GIO - British Columbia & Yukon
(604) 684-3635
Toll-free: 1-877-772-37777
Website: www.gio-scad.org

GIO - Ontario
(416) 362-9528
Toll-free: 1-800-387-2880
Website: www.gio-scad.org

GIO - Prairies, Northwest Territories & Nunavut
(780) 423-2212
Toll-free: 1-800-377-6378
Website: www.gio-scad.org

Province of Québec
GIO
(514) 288-6015
Toll-free: 1-800-361-5131
Website: www.gio-scad.org

OR

Autorité des marchés financiers (l'Autorité)
Québec City (418) 525-0311
Montréal (514) 395-0311
Toll-free: 1-866-526-0311
E-mail: Renseignements-consommateur@lautorite.qc.ca

GIO – Alberta
(780) 421-8181
Toll-free: 1-888-421-4212
Website: www.gio-scad.org

For more information or to submit the facts of your insurance-related dispute, please visit the GIO website at www.gio-scad.org.

Should you be dissatisfied with the outcome of your broker's resolution or with the GIO's / l'Autorité's assistance, please submit your written complaint to:

Lloyd's Canada Inc.
Broker Management Services
1155 rue Metcalfe, Suite 2220
Montreal, Quebec H3B 2V6
Tel: 1-877-4LLOYDS
Fax: (514) 861-0470
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REVIEW ARTICLE

Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation

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Abstract

This review aims to cover experimental data on oxidative effects of low-intensity radiofrequency radiation (RFR) in living cells. Analysis of the currently available peer-reviewed scientific literature reveals molecular effects induced by low-intensity RFR in living cells; this includes significant activation of key pathways generating reactive oxygen species (ROS), activation of peroxidation, oxidative damage of DNA and changes in the activity of antioxidant enzymes. It indicates that among 100 currently available peer-reviewed studies dealing with oxidative effects of low-intensity RFR, in general, 93 confirmed that RFR induces oxidative effects in biological systems. A wide pathogenic potential of the induced ROS and their involvement in cell signaling pathways explains a range of biological/health effects of low-intensity RFR, which include both cancer and non-cancer pathologies. In conclusion, our analysis demonstrates that low-intensity RFR is an expressive oxidative agent for living cells with a high pathogenic potential and that the oxidative stress induced by RFR exposure should be recognized as one of the primary mechanisms of the biological activity of this kind of radiation.

Keywords

Cellular signaling, cancer, free radicals, oxidative stress, radiofrequency radiation, reactive oxygen species

History

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Introduction

Intensive development of wireless technologies during the last decades led to a dramatic increase of background radiofrequency radiation (RFR) in the human environment. Thus, the level of indoor background RFR in industrialized countries increased 5,000-fold from 1985 to 2005 (Maes, 2005). Such significant environmental changes may have a serious impact on human biology and health. As a proof of such impact, a series of epidemiological studies on the increased risk of tumorigenesis in ''heavy'' users of wireless telephony exists (Hardell et al., 2007, 2011; Sadetzki et al., 2008; Sato et al., 2011). Some studies indicate that long-term RFR exposure in humans can cause various non-cancer disorders, e.g., headache, fatigue, depression, tinnitus, skin irritation, hormonal disorders and other conditions (Abdel-Rassoul et al., 2007; Buchner & Eger, 2011; Chu et al., 2011; Johansson, 2006; Santini et al., 2002; Yakymenko et al., 2011). In addition, convincing studies on hazardous effects of RFR in human germ cells have been published (Agarwal et al., 2009; De Iuliis et al., 2009).

All abovementioned studies dealt with the effects of low-intensity RFR. This means that the intensity of radiation was far below observable thermal effects in biological tissues, and far below safety limits of the International Commissions on Non-Ionizing Radiation Protection (ICNIRP) (ICNIRP, 1998). To date, molecular mechanisms of non-thermal effects of RFR are still a bottleneck in the research on the biological/health effects of low-intensity RFR, although recently many studies have been carried out on metabolic changes in living cells under low-intensity RFR, and comprehensive reviews were published (Belyaev, 2010; Consales et al., 2012; Desai et al., 2009; Yakymenko et al., 2011). In the present work, we analyze the results of molecular effects of low-intensity RFR in living cells and model systems, with a special emphasis on oxidative effects and free radical mechanisms. It might seem paradoxical that, despite being non-ionizing, RFR can induce significant activation of free radical processes and overproduction of reactive oxygen species (ROS) in living cells. We believe that the analysis of recent findings will allow recognition of a general picture of the potential health effects of already ubiquitous and ever-increasing RFR.

Radiofrequency radiation

RFR is a part of electromagnetic spectrum with frequencies from 30 kHz to 300 GHz. RFR is classified as non-ionizing,

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which means that it does not carry sufficient energy for ionization of atoms and molecules. A part of RFR with the highest frequencies (300 MHz to 300 GHz) is referred to as microwaves (MWs). MW is RFR with the highest energy, which can potentially generate the highest thermal effects in the absorbing matter.

The main indexes of RFR are (i) frequency (Hz); (ii) intensity or power density (PD) of radiation (W/m^2 or $\mu W/cm^2$); (iii) its modulated or non-modulated nature; and (iv) continuous or discontinuous pattern of radiation. For the absorbed RFR energy, a parameter of specific absorption rate (SAR) is used (W/kg). The most common digital standard of RFR for mobile communication is still GSM (Global System for Mobile communication), which utilizes frequencies at about 850, 900, 1800 and 1900 MHz. This radiation is frequency modulated, with channel rotation frequency of 217 Hz, and belongs to the radiation of the pulsed mode (Hyland, 2000).

As to the international safety limits, the ICNIRP recommendations restrict intensity of RFR to 450–1000 $\mu W/cm^2$ (depending on the frequency of radiation) and the SAR value to 2 W/kg, as calculated for human heads and torsos (ICNIRP, 1998). These indexes were adopted by ICNIRP based on the behavioral response of laboratory rats, which were exposed to gradually increased intensities of RFR to determine the point at which the animals became thermally distressed (Gandhi et al., 2012).

Low-intensity RFR is referred to as radiation with intensities which do not induce significant thermal effects in biological tissues. Accordingly, any intensity of RFR under the ICNIRP limits can be referred to as low-intensity. In this paper we will analyze only the effects of low-intensity RFR.

Physical/biophysical effects of low-intensity RFR in living cells

RFR, especially MW, can produce thermal effects in matter due to interaction with charged particles, including free electrons, ions or polar molecules, inducing their oscillations in electromagnetic field. The thermal effect of MW can be seen when warming food in the microwave. The effect strongly depends on the intensity of radiation and is mostly negligible under low-intensity RFR conditions. On the other hand, energy of RFR/MW is insufficient not only for the ionization of molecules, but even for activation of orbital electrons. Hence, RFR was often assessed as a factor producing only thermal effects. Nevertheless, evident biological effects of low-intensity RFR promoted research on physical mechanisms of non-thermal biological effects of this kind of radiation.

A biophysical model of a forced-vibration of free ions on the surface of a cell membrane due to external oscillating electromagnetic field (EMF) was proposed (Panagopoulos et al., 2000, 2002). According to the authors, this vibration of electric charges can cause disruption of the cellular electrochemical balance and functions.

A ''moving charge interaction'' model was proposed for low-frequency EMF (Blank and Soo, 2001). The authors explained activation of genes and synthesis of stress proteins under EMF exposure due to interaction of the field with moving electrons in DNA (Blank and Soo, 2001; Goodman and Blank, 2002). They also demonstrated that EMF increased electron transfer rates in cytochrome oxidase and accelerated charges in the Na,K-ATPase reaction. Moreover, they demonstrated acceleration of the oscillating Belousov–Zhabotinski reaction in homogeneous solutions due to the application of low-frequency EMF (Blank and Soo, 2003).

An ability of low-strength magnetic fields to trigger onset- and offset-evoked potentials was demonstrated (Marino et al., 2009). Effectiveness of a rapid magnetic stimulus (0.2 ms) has led the authors to a conclusion on direct interaction between the field and ion channels in plasma membrane. A plausible mechanism of overproduction of free radicals in living cell due to electron spin flipping in confined free radical pairs in magnetic field of RFR was proposed (Georgiou, 2010).

A significant effect of low-intensity RFR on ferritin, an iron cage protein present in most living organisms from bacteria to humans, was revealed (Céspedes and Ueno, 2009). Exposure of ferritin solution to low-intensity RFR significantly, up to threefold, reduced iron chelation with ferrozine. The authors explained that magnetic field of RFR plays a principle role in the observed effect, and that this effect is strongly non-thermal. The non-thermal mechanism of the interaction of RFR magnetic fields with ferritin is supposedly mediated by an inner super-paramagnetic nanoparticle ($9H_2O \times 5Fe_2O_3$ with up to 4500 iron ions), which is a natural phenomenon intrinsic to the cells. It results in reduction of input of iron chelates into the ferritin cage. The authors underlined the potential role of ferritin malfunction for oxidative processes in living cell due to the participation of Fe^{2+} ions in the Fenton reaction, which produces hydroxyl radicals. In this respect, it is interesting to point to the results of an *in vitro* study with RFR exposure of rat lymphocytes treated by iron ions (Zmyślony et al., 2004). Although RFR exposure (930 MHz) did not induce detectable intracellular ROS overproduction, the same exposure in the presence of $FeCl_2$ in the lymphocyte suspensions induced a significant overproduction of ROS.

Another set of studies indicates on a possibility of changes in protein conformation under RFR exposure. Thus, low-intensity 2.45 MHz RFR accelerated conformational changes in β-lactoglobulin through excitation of so-called collective intrinsic modes in the protein (Bohr and Bohr, 2000a, 2000b), which suggests a principal ability of RFR to modulate the non-random collective movements of entire protein domains. Similarly, a frequency-dependent effect on intrinsic flexibility in insulin structure due to applied oscillating electric field was demonstrated (Budi et al., 2007). Moreover, macromolecular structure of cytoskeleton was significantly altered in fibroblasts of Chinese hamster after the exposure to modulated RFR of the GSM standard (Pavicic and Trosic, 2010). Thus, a 3 h exposure of fibroblasts to modulated RFR (975 MHz) led to significant changes in the structure of microtubules and actin microfilaments, which have polar cytoskeleton structures, while non-polar vimentin filaments reportedly stayed unchanged. Taking into account an extensive regulatory potential of cytoskeleton on cell homeostasis, these data could obviously add to the nature of the biological effects of RFR.

It was shown that ornithine decarboxylase (ODC) can significantly change its activity under low-intensity RFR exposure (Byus et al., 1988; Hoyto et al., 2007; Litovitz et al., 1993, 1997; Paulraj et al., 1999).

In addition, so-called ''calcium effects'' under RFR exposure in living cells have been demonstrated (Dutta et al., 1989; Paulraj et al., 1999; Rao et al., 2008), which include a significant increase in intracellular Ca^{2+} spiking. Taking into account that calcium is a ubiquitous regulator of cellular metabolism, these data point to a possibility that non-thermal RFR can activate multiple Ca^{2+}-dependent signaling cascades.

Finally, an ability of low-intensity MW to dissociate water molecules was demonstrated in model experiments years ago (Vaks et al., 1994). In these experiments, MW of 10 GHz with radiated power 30 mW produced a significant level of H_2O_2 in deionized water (and also in $MgSO_4$ solution) under stable temperature conditions. According to the authors, a kinetic excitation of liquid water associates $C(H_2O)$ upon the absorption of MW leads to subsequent viscous losses due to friction between moving clusters of water molecules. It results in partial irreversible decomposition of water, including breaks of intramolecular bonds (H–OH) due to a mechanochemical reaction, and generation of $H^{\bullet}$; $OH^{\bullet}$; H^+ and OH^- groups. Among these, the hydroxyl radical ($OH^{\bullet}$) is the most aggressive form of ROS, which can break any chemical bond in surrounding molecules (Halliwell, 2007). The authors assessed that this type of mechanochemical transformation in water could be responsible for 10^{-4}–10^{-8} relative parts of the total MW energy absorbed. Given the fact that the water molecules are ubiquitous in living cells, even a subtle chance for dissociation of water molecules under low-intensity RFR exposure could have a profound effect on tissue homeostasis. It is of note here that one $OH^{\bullet}$ radical can initiate irreversible peroxidation of many hundreds of macromolecules, e.g. lipid molecules (Halliwell, 1991). Taken together, these data show that non-thermal RFR can be absorbed by particular charges, molecules and cellular structures, and in this way can potentially induce substantial modulatory effects in living cell.

Generation of reactive oxygen species under RFR exposure in living cells

NADH oxidase of cellular membrane was suggested as a primary mediator of RFR interaction with living cells (Friedman et al., 2007). Using purified membranes from HeLa cells, the authors experimentally proved that the exposure to RFR of 875 MHz, 200 μW/cm^2 for 5 or 10 min significantly, almost threefold, increased the activity of NADH oxidase. NADH oxidases are membrane-associated enzymes that catalyze one-electron reduction of oxygen into superoxide radical using NADH as a donor of electron, thus producing powerful ROS. This enzyme has been traditionally known due to its role in induction of oxidative burst in phagocytes as a part of immune response. Yet, later the existence of non-phagocytic NAD(P)H oxidases was revealed in various types of cells, including fibroblasts, vascular and cardiac cells (Griendling et al., 2000). Obviously, the presence of superoxide-generating enzyme in many types of non-phagocytic cells points to the considerable regulatory roles of ROS in living cells. On the other hand, an ability of low-intensity RFR to modulate the activity of the NADH oxidase automatically makes this factor a notable and potentially dangerous effector of cell metabolism. Notably, the authors pointed out that the acceptor of RFR is different from the peroxide-generating NADPH oxidases, which are also found in plasma membranes (Low et al., 2012).

The other powerful source of ROS in cells is mitochondrial electron transport chain (ETC), which can generate superoxide due to breakdowns in electron transport (Inoue et al., 2003). It was demonstrated that generation of ROS by mitochondrial pathway can be activated under RFR exposure in human spermatozoa (De Iuliis et al., 2009). The authors revealed a dose-dependent effect of 1.8 GHz RFR exposure on ROS production in spermatozoa, particularly in their mitochondria. The significantly increased level of total ROS in spermatozoa was detected under RFR with SAR = 1 W/kg, which is below the safety limits accepted in many countries. It was demonstrated recently in our laboratory that the exposure of quail embryos *in ovo* to extremely low-intensity RFR (GSM 900 MHz, 0.25 μW/cm^2) during the initial days of embryogenesis resulted in a robust overproduction of superoxide and nitrogen oxide radicals in mitochondria of embryonic cells (Burlaka et al., 2013). It is not clear yet which particular part of ETC is responsible for the interaction with RFR. To date, three possible sites of generation of superoxide in ETC have been shown: the ETC complex I (Inoue et al., 2003), complex II (Liu et al., 2002), and complex III (Guzy and Schumacker, 2006). A significant inverse correlation between mitochondrial membrane potential and ROS levels in living cell was found (Wang et al., 2003). As the authors underlined, such a relationship could be due to two mutually interconnected phenomena: ROS causing damage to the mitochondrial membrane, and the damaged mitochondrial membrane causing increased ROS production.

In addition to the well-established role of the mitochondria in energy metabolism, regulation of cell death is a second major function of these organelles. This, in turn, is linked to their role as the powerful intracellular source of ROS. Mitochondria-generated ROS play an important role in the release of cytochrome c and other pro-apoptotic proteins, which can trigger caspase activation and apoptosis (Ott et al., 2007). A few reports indicate on activation of apoptosis due to low-intensity RFR exposure. In human epidermoid cancer KB cells, 1950 MHz RFR induced time-dependent apoptosis (45% after 3 h) that is paralleled by 2.5-fold decrease of the expression of ras and Raf-1 and of the activity of ras and Erk-1/2 (Caraglia et al., 2005). Primary cultured neurons and astrocytes exposed to GSM 1900 MHz RFR for 2 h demonstrated up-regulation of caspase-2, caspase-6 and Asc (apoptosis associated speck-like protein containing a card) (Zhao et al., 2007). Up-regulation in neurons occurred in both "on" and "stand-by" modes, but in astrocytes only in the "on" mode. We should underline that, in that study an extremely high biological sensitivity to RFR was demonstrated, as a cell phone in the ''stand-by'' position emits negligibly low-intensity of radiation (up to hundredths μW/cm^2).

Based on the analysis of available literature data, we identified altogether 100 experimental studies in biological models which investigated oxidative stress due to low-intensity RFR exposures. From these 100 articles, 93 studies (93%) demonstrated significant oxidative effects induced by

low-intensity RFR exposure (Table 1–3), while 7 studies (7%) demonstrated the absence of significant changes (Table 4). The total number includes 18 *in vitro* studies, 73 studies in animals, 3 studies in plants and 6 studies in humans. Majority of the research was done on laboratory rats (58 studies, with 54 positive results), while 4 studies out of 6 in humans were positive. From the *in vitro* studies, 17 were positive (94.4%), including 2 studies on human spermatozoa and 2 studies on human blood cells.

Most of the studies utilized RFR exposure in MW range, including a use of commercial or trial cell phones as sources of radiation. The power densities of RFR applied in positive studies varied from 0.1 μW/cm^2 (Oksay et al., 2014) to 680 μW/cm^2 (Jelodar et al., 2013) and SAR values varied from 3 μW/kg (Burlaka et al., 2013) to the ICNIRP recommended limit of 2 W/kg (Naziroglu et al., 2012a; Xu et al., 2010). Exposure times in positive studies varied from 5 min (Friedman et al., 2007) to 12.5 years, 29.6 h/month (Hamzany et al., 2013).

The most often used indexes of oxidative stress analyzed in the studies were ROS production, levels of lipid peroxidation (LPO)/malondialdehyde (MDA), protein oxidation (PO), nitric oxides (NO_x), glutathione (GSH), activity of antioxidant enzymes (superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GSH-Px)). It is important that some studies directly pointed to induction of free radicals (superoxide radical, NO) as a primary reaction of living cells to RFR exposure (Burlaka et al., 2013; Friedman et al., 2007). As we pointed out earlier, direct activation of NADH oxidase (Friedman et al., 2007) and the mitochondrial pathway of superoxide overproduction (Burlaka et al., 2013; De Iuliis et al., 2009) have been experimentally proven. Besides, a significant overproduction of nitrogen oxide was revealed in some studies (Avci et al., 2012; Bilgici et al., 2013; Burlaka et al., 2013), although it is unclear whether an induction of expression of NO-synthases or direct activation of the enzyme took place. It is however clear that significantly increased levels of these free radical species (superoxide and nitrogen oxide) in cells due to RFR exposure result in an activation of peroxidation and repression of activities of key antioxidant enzymes. It is indicative that many studies demonstrated effectiveness of different antioxidants to override oxidative stress caused by RFR exposure. Such effects have been reported for melatonin (Ayata et al., 2004; Lai and Singh, 1997; Oktem et al., 2005; Ozguner et al., 2006; Sokolovic et al., 2008), vitamin E and C (Jelodar et al., 2013; Oral et al., 2006), caffeic acid phenethyl ester (Ozguner et al., 2006), selenium, L-carnitine (Turker et al., 2011) and garlic (Avci et al., 2012; Bilgici et al., 2013).

It is worthwhile to emphasize a strict non-thermal character of ROS overproduction under RFR exposure described in the cited reports. As low as 0.1 μW/cm^2 intensity of RFR and absorbed energy (specific absorption rate, SAR) of 0.3 μW/kg were demonstrated to be effective in inducing significant oxidative stress in living cells (Burlaka et al., 2013; Oksay et al., 2014). This observation is particularly important as the modern international safety limits on RFR exposure are based solely on the thermal effects of radiation and only restrict RFR intensity to 450–1000 μW/cm^2 and SAR to 2 W/kg (ICNIRP, 1998). Moreover, studies where high (thermal) intensities of RFR have been used could not reveal oxidative effects (Hong et al., 2012; Kang et al., 2013; Luukkonen et al., 2009), which might point to the variety of molecular mechanisms for different radiation intensities.

Taken together, the analysis of the contemporary scientific literature on the biological effects of RFR persuasively proves that the exposure to low-intensity RFR in living cells leads to generation of significant levels of ROS and results in a significant oxidative stress.

Oxidative damage of DNA under RFR exposure

To date more than hundred papers have been published on mutagenic effects of RFR and most of them revealed significant effects (Ruediger, 2009). There is a substantial number of studies which demonstrated the formation of micronuclei (Garaj-Vrhovac et al., 1992; Tice et al., 2002; Zotti-Martelli et al., 2005) or structural anomalies of metaphase chromosomes (Garson et al., 1991; Kerbacher et al., 1990; Maes et al., 2000) in living cells due to low-intensity RFR exposure. However, majority of the studies on the mutagenic effects of RFR successfully used a comet assay approach (Baohong et al., 2005; Belyaev et al., 2006; Diem et al., 2005; Kim et al., 2008; Lai and Singh, 1996; Liu et al., 2013a). Particular studies identified specific marker of oxidative damage of DNA, 8-hydroxy-2'-deoxyguanosine (8-OH-dG) (Burlaka et al., 2013; De Iuliis et al., 2009; Guler et al., 2012; Khalil et al., 2012; Xu et al., 2010). Thus, the level of 8-OH-dG in human spermatozoa was shown to be significantly increased after *in vitro* exposure to low-intensity RFR (De Iuliis et al., 2009). Likewise, we demonstrated that the exposure of quail embryos *in ovo* to GSM 900 MHz of 0.25 μW/cm^2 during a few days was sufficient for a significant, two-threefold, increase of 8-OH-dG level in embryonic cells (Burlaka et al., 2013).

It would be logical to assume that most mutagenic effects due to the RFR exposure are caused by oxidative damage to DNA, as the overproduction of ROS in living cells due to RFR exposure was reliably documented. It is known that superoxide itself does not affect DNA. The most aggressive form of ROS, which is able to affect the DNA molecule directly, is hydroxyl radical (Halliwell, 2007). The hydroxyl radicals are generated in cell in the Fenton reaction ($Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^{\bullet} + OH^-$) and in the Haber–Weiss reaction ($O_2^{\bullet -} + H_2O_2 \rightarrow O_2 + OH^{\bullet} + OH^-$) (Valko et al., 2006). On the other hand, increased concentration of NO in addition to superoxide in the RFR-exposed cells can lead to the formation of other aggressive form of ROS, peroxynitrite ($ONOO^-$), which can also cause DNA damage (Valko et al., 2006).

Free radicals induced under the RFR exposure can perturb cellular signaling

Taking into account the abovementioned data, we can state that the exposure to RFR leads to overproduction of free radicals/ROS in living cell. Certainly, free radicals can induce harmful effects via direct damage due to oxidation of biological macromolecules. To that, it becomes clear nowadays that free radicals/ROS are an intrinsic part of the cellular signaling cascades (Forman et al., 2014). Thus, hydrogen peroxide appears as a second messenger both in

Table 1. Publications which reported positive findings on oxidative stress caused by RFR exposure of cells *in vitro*.

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Agarwal et al., 2009)	Human spermatozoa	Cell phone RFR, in talk mode, for 1 h	Increase in reactive oxygen species (ROS) level, decrease in sperm motility and viability.
(Campisi et al., 2010)	Rat astroglial cells	900 MHz (continuous or modulated), electric field 10 V/m, for5; 10; 20 min	Increase in ROS levels and DNA fragmentation after exposure to modulated RFR for 20 min.
(De Iuliis et al., 2009)	Human spermatozoa	1.8 GHz, SAR = 0.4–27.5 W/kg	Increased amounts of ROS.
(Friedman et al., 2007)	HeLa membranes	875 MHz, 200 μW/cm^2, for 5 and 10 min	Increased NADH oxidase activity.
(Hou et al., 2014)	Mouse embryonic fibroblasts (NIH/3T3)	1800-MHz GSM-talk mode RFR, SAR = 2 W/kg, intermittent exposure (5 min on/10 min off) for 0.5–8 h	Increased intracellular ROS levels.
(Kahya et al., 2014)	Cancer cell cultures	900 MHz RFR, SAR = 0.36 W/kg, for 1 h	Induced apoptosis effects through oxidative stress, selenium counteracted the effects of RFR exposure.
(Lantow et al., 2006a)	Human blood cells	Continuous wave or GSM signal,SAR = 2 W/kg, for 30 or 45 min of continuous or 5 min ON, 5 min OFF	After continuous or intermittent GSMsignal a different ROS production was detected in human monocytes compared to sham.
(Lantow et al., 2006b)	Human Mono Mac 6 and K562 cells	Continuous wave, GSM speaking only, GSM hearing only, GSM talk, SARs of 0.5, 1.0, 1.5 and 2.0 W/kg.	The GSM-DTX signal at 2 W/kg produced difference in free radical production compared to sham.
(Liu et al., 2013b)	GC-2 cells	1800 MHz, SAR = 1; 2 W/kg,5 min ON, 10 min OFF for 24 h	In the 2 W/kg exposed cultures, the level of ROS was increased.
(Lu et al., 2012)	Human blood mononuclear cells	900 MHz, SAR = 0.4 W/kg, for 1–8 h	The increased level of apoptosis induced through the mitochondrial pathway and mediated by activating ROS and caspase-3.
(Marjanovic et al., 2014)	V79 cells	1800 MHz, SAR = 1.6 W/kg, for 10, 30 and 60 min	ROS level increased after 10 min of exposure. Decrease in ROS level after 30-min treatment indicating antioxidant defense mechanism activation.
(Naziroglu et al., 2012b)	HL-60 cells	2450 MHz, pulsed, SAR = 0.1–2.5 W/kg,for 1; 2; 12 or 24 h	Lipid peroxide (LPO) levels were increased at all exposure times.
(Ni et al., 2013)	Human lens epithelial cells	1800 MHz, SAR = 2; 3; 4 W/kg	The ROS and malondialdehyde (MDA) levels were increased.
(Pilla, 2012)	Neuronal cells and human fibroblasts	27.12 MHz, pulsed, electric field 41 V/m, 2 min prior to lipopolysaccharide administration or for 15 min	Increased level of nitric oxide (NO).
(Sefidbakht et al., 2014)	HEK293T cells	940 MHz, SAR = 0.09 W/kg, for 15, 30, 45, 60 and 90 min	ROS generation increased in the 30 min exposed cells. A sharp rise in catalase (CAT) and superoxide dismutase (SOD) activity and elevation of glutathione (GSH) during the 45 min exposure.
(Xu et al., 2010)	Primary cultured neurons	1800 MHz, pulsed, SAR = 2 W/kg, for 24 h	An increase in the levels of8-hydroxy-2'-deoxyguanosine (8-OH-dG).
(Zmyślony et al., 2004)	Rat lymphocytes	930 MHz, PD of 500 μW/cm^2, SAR = 1.5 W/kg, for 5 and 15 min	Intracellular ROS level increased in exposed $FeCl_2$ treated cells compared with unexposed $FeCl_2$ treated cells.

*All effects were statistically significant (at least $p < 0.05$) as compared to control or sham exposed groups.

insulin signaling and in growth factor-induced signalling cascades (Sies, 2014). These species are also implicated in biochemical mechanism of oxidation of ethanol and in other metabolic processes (Oshino et al., 1975) and is also required for initiation of wound repair (Enyedi and Niethammer, 2013). In addition, ROS at relatively low concentrations can modulate inflammation via activation of NF-kB pathway (Hayden and Ghosh, 2011). Therefore, even subtle exposures to RFR with generation of hardly detectable quantities of free radicals can have their meaningful biological consequences.

We could ascertain the signaling effects of moderate levels of free radicals from our experiments in quail embryos irradiated with the commercial cell phone. Thus, we were able to show that the prolonged exposures of embryos *in ovo* led to robust repression of their development (Tsybulin et al., 2013), which was concomitant with

Table 2. Publications which reported positive findings on oxidative stress caused by RFR exposure of animals and plants.

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Akbari et al., 2014)	Rat whole body	RFR from base transceiver station	Glutathione peroxidase (GSH-Px), SOD, and CAT activity decreased and level of MDA increased. Vitamin C reduced the effect.
(Al-Damegh, 2012)	Rat whole body	Cell phone RFR, 15, 30, or 60 min/day for 2 weeks	Levels of conjugated dienes, LPO and CAT activities in serum and testicular tissue increased, the total serum and testicular tissue GSH and GSH-Px levels decreased.
(Avci et al., 2012)	Rat whole body	1800 MHz, SAR = 0.4 W/kg, 1 h/day for 3 weeks	An increased level of protein oxidation (PO) in brain tissue and an increase in serum NO. Garlic administration reduced protein oxidation in brain tissue.
(Ayata et al., 2004)	Rat whole body	900 MHz, 30 min/day for 10 days	MDA and hydroxyproline levels and activities of CAT and GSH-Px were increased, andsuperoxide dismutase (SOD) activity was decreased in skin. Melatonin treatment reversed effect.
(Aynali et al., 2013)	Rat whole body	2450 MHz, pulsed, SAR = 0.143 W/kg,60 min/day for 30 days	LPO was increased, an administration of melatonin prevented this effect.
(Balci et al., 2007)	Rat whole body	''Standardized daily dose'' of cell phoneRFR for 4 weeks	In corneal tissue, MDA level and CAT activity increased, whereas SOD activity was decreased. In the lens tissues, the MDA level was increased.
(Bilgici et al., 2013)	Rat whole body	850–950 MHz,SAR = 1.08 W/kg,1 h/day for 3 weeks	The serum NO levels and levels of MDA and the PO in brain were increased. An administration of garlic extract diminished these effects.
(Bodera et al., 2013)	Rat whole body	1800 MHz, GSM, for 15 min	Reduced antioxidant capacity both in healthy animals and in those with paw inflammation.
(Burlaka et al., 2013)	Quail embryo *in ovo*	GSM 900 MHz, power density (PD) of 0.25 μW/cm^2, SAR = 3 μW/kg, 48 sec ON - 12 sec OFF, for 158–360 h	Overproduction of superoxide and NO, increased levels of thiobarbituric acid reactive substances (TBARS) and 8-OH-dG, decreased SOD and CAT activities.
(Burlaka et al., 2014)	Male rat whole body	Pulsed and continuous MWin the doses equivalent to the maximal permitted energy load for the staffs of the radar stations	Increased rates of superoxide production, formation of the iron-nitrosyl complexes and decreased activity of NADH-ubiquinone oxidoreductase complex in liver, cardiac and aorta tissues 28 days after the exposure.
(Cenesiz et al., 2011)	Guinea pig whole body	900; 1800 MHz RFR from base station antennas, 4 h/day for 20 days	Difference in guinea pigs subjected to 900 and 1800 MHz for plasma oxidant status levels. NO level changed in 900 MHz subjected guinea pigs, as compared to the control.
(Cetin et al., 2014)	Pregnant rats and offspring	900; 1800 MHz RFR, 1 h/day during pregnancy and neonatal development	Brain and liver GSH-Px activities, selenium concentrations in the brain and liver vitamin A and β-carotene concentrations decreased in offspring.
(Dasdag et al., 2009)	Head of rats	900 MHz, 2 h/day for 10 months	The total antioxidant capacity and CATactivity in brains were higher than that in the sham group.

(*continued*)

Table 2. Continued

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Dasdag et al., 2012)	Head of rats	900 MHz, cell-phones-like, 2 h/day for 10 months	Protein carbonyl level was higher in the brain of exposedrats.
(Dasdag et al., 2008)	Rat whole body	900 MHz, PD of 78 μW/cm^2, 2 h/days for10 months.	Increased levels of MDA and total oxidative status in liver tissue.
(Deshmukh et al., 2013)	Rat whole body	900 MHz, 2 h/day, 5 days a week for 30 days	The levels of LPO and PO were increased.
(Esmekaya et al., 2011)	Rat whole body	900 MHz, pulsed, modulated, SAR = 1.2 W/kg, 20 min/day for 3 weeks	The increased level of MDA and NOx, and decreased levels of GSH in liver, lung, testis and heart tissues.
(Furtado-Filho et al., 2014)	Rat whole body	950 MHz, SAR = 0.01–0.88 W/kg,30 min/day for 21 days during pregnancy (or additionally 6 or 15 days of postnatal period)	Neonatal rats exposed in utero had decreased levels of CAT and lower LPO, and genotoxic effect.
(Guler et al., 2012)	Rabbit infant whole body	GSM 1800 MHz, 15 min/day for 7 days (females) or 14 days (males)	LPO levels in the liver tissues of females and males increased,liver 8-OH-dG levels of females were increased.
(Guney et al., 2007)	Rat whole body	900 MHz, 30 min/day for 30 days	Endometrial levels of NO and MDA increased,endometrial SOD, CAT and GSH-Px activities were decreased.Vitamin E and C treatment prevented these effects.
(Gürler et al., 2014)	Rat whole body	2450 MHz, 3.68 V/m, 1 h/day for 30 days	Increased 8-OH-dG level in both plasma and brain tissue whereas it increased PO level only in plasma. Garlic prevented the increase of 8-OH-dG level in brain tissue and plasma PO levels.
(Ilhan et al., 2004)	Rat whole body	900 MHz, from cell phone,1 h/day for 7 days	Increase in MDA, NO levels, andxanthine oxidase (XO) activity, decrease in SOD and GSH-Px activities in brain. These effects were prevented by Ginkgo bilobaextract treatment.
(Jelodar, et al., 2013)	Rat whole body	900 MHz, PD of 680 μW/cm^2, 4 h/day for 45 days,	The concentration of MDA was increased and activities of SOD, GSH-Px and CAT were decreased in rat eyes. An administration of vitamin C prevented these effects.
(Jelodar et al., 2013)	Rat whole body	900 MHz, daily for 45 days	Increased level of MDA and decreased antioxidant enzymes activity in rat testis.
(Jing et al., 2012)	Rat whole body	Cell phone RFR, SAR = 0.9 W/kg,3 x 10; 30 or 60 min for 20 days during gestation	After 30 and 60 min the level of MDA was increased, the activities of SOD and GSH-Pxwere decreased.
(Kerman & Senol, 2012)	Rat whole body	900 MHz, 30 min/day for 10 days	Tissue MDA levels were increased, SOD, CAT and GSH-Pxactivities were reduced. Melatonin treatment reversed these effects.
(Kesari et al., 2010)	Male rat whole body	Cell phone RFR, SAR = 0.9 W/kg,2 h/day for 35 days	Reduction in protein kinase activity, decrease in sperm count and increase in apoptosis.
(Kesari et al., 2011)	Rat whole body	900 MHz, pulsed, SAR = 0.9 W/kg,2 h/day for 45 days	Increase in the level of ROS, decrease in the activities of SOD and GSH-Px,and in the level of pineal melatonin.
(Kesari et al., 2013)	Rat whole body	2115 MHz, SAR = 0.26 W/kg,2 h/day for 60 days	The level of ROS, DNA damage and theapoptosis rate were increased.
(Khalil et al., 2012)	Rat whole body	1800 MHz, electric field 15–20 V/m, for2 h	Elevations in the levels of 8-OH-dG in urine.

(*continued*)

Table 2. Continued

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Kismali et al., 2012)	Rabbit whole body (non-pregnant and pregnant)	1800 MHz, GSM modulation, 15 min/day for 7 days	Creatine kinases levels' changes.
(Koc et al., 2013)	Male rat whole body	Cell phone RFR at calling or stand-by	Oxidative stress detected at both calling and stand-by exposures.
(Koylu et al., 2006)	Rat whole body	900 MHz	The levels of LPO in the brain cortex and hippocampus increased.These levels in the hippocampus were decreased by melatonin administration.
(Koyu et al., 2009)	Rat whole body	900 MHz	The activities of XO, CAT and level of LPO increased in liver.XO, CAT activities and LPO levels were decreased by caffeic acid phenethyl ester (CAPE) administration.
(Kumar et al., 2014)	Rat whole body	Cell phone 1910.5 MHz RFR, 2 h/day for 60 days day (6 days a week).	Increase in LPO, damage in sperm cells and DNA damage.
(Lai & Singh, 1997)	Rat whole body	2450 MHz, pulsed, PD = 2 mW/cm^2, SAR = 1.2 W/kg	Melatonin or spin-trap compound blocked DNA strand breaks induced by RFR exposure in rat brain cells.
(Luo et al., 2014)	Rat whole body	900 MHz imitated cell phone RFR, 4 h/day for 12 days	Contents of liver MDA and Nrf2 protein increased, contents of liver SOD and GSH decreased.
(Mailankot et al., 2009)	Rat whole body	900/1800 MHz,GSM,1 h/day for 28 days	Increase in LPO and decreased GSH content in the testis and epididymis.
(Manta et al., 2013)	Drosophila whole body	1880–1900 MHz, DECT modulation, SAR = 0.009 W/kg, for 0.5–96 h	Increase in ROS levels in male and female bodies, a quick response in ROS increase in ovaries.
(Marzook et al., 2014)	Rat whole body	900 MHz from cellular tower, 24 h/day for 8 weeks	SOD and CAT activities were reduced in blood, sesame oil reversed the effect
(Meena et al., 2013)	Rat whole body	2450 MHz, PDof 210 μW/cm^2, SAR = 0.14 W/kg, 2 h/day for 45 days	Increased level of MDA and ROS in testis. Melatonin prevented oxidative stress.
(Megha et al., 2012)	Rat whole body	900; 1800 MHz, PD of 170 μW/cm^2, SAR = 0.6 mW/kg, 2 h/day, 5 days/week for 30 days	The levels of the LPO and PO were increased; the level of GSH was decreased.
(Meral et al., 2007)	Guinea pig whole body	890–915 MHz,from cell phone, SAR = 0.95 w/kg, 12 h/day for 30 days (11 h 45 min stand-by and 15 min spiking mode)	MDA level increased, GSH level and CAT activity weredecreased in the brain. MDA, vitamins A, D_3 and E levels and CAT enzyme activity increased, and GSH level was decreased in the blood.
(Motawi et al., 2014)	Rat whole body	Test cellphone RFR, SAR = 1.13 W/kg, 2 h/day for 60 days	Increments in conjugated dienes, protein carbonyls, total oxidant status and oxidative stress index along with a reduction of total antioxidant capacity levels.
(Naziroglu & Gumral, 2009)	Rat whole body	2450 MHz,60 min/day for 28 days	Decrease of the cortex brain vitamin A, vitamin C and vitamin E levels.
(Naziroglu et al., 2012a)	Rat whole body	2450 MHz, 60 min/day for 30 days	LPO, cell viability and cytosolic Ca^{2+} values in dorsal root ganglion neurons were increased.
(Oksay et al., 2014)	Rat whole body	2450 MHz, pulsed, PD of0.1 μW/cm^2, SAR = 0.1 W/kg, 1 h/day for 30 days	LPO was higher in exposed animals. Melatonin treatment reversed the effect.
(Oktem et al., 2005)	Rat whole body	900 MHz, 30 min/day for 10 days	Renal tissue MDA level increased, SOD, CAT and GSH-Px activities were reduced. Melatonin treatment reversed these effects.
(Oral et al., 2006)	Rat whole body	900 MHz, 30 min/day for 30 days	Increased MDA levels and apoptosis in endometrial tissue.Treatment with vitamins E and C diminished these changes.

(*continued*)

Table 2. Continued

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Ozguner et al., 2005a)	Rat whole body	900 MHz, 30 min/day for 10 days	Heart tissue MDA and NO levels increased, SOD, CAT and GSH-Px activities were reduced. CAPE treatment reversed these effects.
(Ozguner et al., 2006)	Rat whole body	900 MHz, from cell phone	Retinal levels of NO and MDA increased, SOD, GSH-Px and CAT activities were decreased.Melatonin and CAPE treatment prevented effects.
(Ozguner et al., 2005b)	Rat whole body	900 MHz	Renal tissue MDA and NO levels increased, the activities of SOD, CAT and GSH-Px were reduced. CAPE treatment reversed these effects.
(Ozgur et al., 2010)	Guinea pig whole body	1800 MHz,GSM, SAR = 0.38 W/kg, 10 or 20 min/day for 7 days	Increases in MDA and total NO(x) levels and decreases in activities of SOD, myeloperoxidase and GSH-Px in liver. Extent of oxidative damage was proportional to the duration of exposure.
(Ozgur et al., 2013)	Rabbit whole body	1800 MHz, pulsed, 15 min/day for 7 days in pregnant animals, for 7 or 15 days in infants	The amount of LPO was increased in the prenatal exposure group.
(Özorak et al., 2013)	Rat whole body	900; 1800; 2450 MHz, pulsed, PD of 12 $\mu W/cm^2$.SAR = 0.18; 1.2 W/kg, 60 min/day during gestation and 6 weeks following delivery	At the age of six weeks, an increased LPO in the kidney and testis, and decreased level of GSH and total antioxidant status.
(Qin et al., 2014)	Male mouse whole body	1800 MHz, 208 $\mu W/cm^2$, 30 or 120 min/d for 30 days	Decreased activities of CAT and GSH-Px and increasedlevel of MDA in cerebrum. Nano-selenium decreased MDA level, and increased GSH-Px and CAT activities.
(Ragy, 2014)	Rat whole body	Cell phone 900 MHz RFR, 1 h/d for 60 days	Increase in MDA levels and decrease total antioxidant capacity levels in brain, liver and kidneys tissues. These alterations were corrected by withdrawal of RFR exposure during 30 days.
(Saikhedkar et al., 2014)	Rat whole body	Cell phone 900 MHz RFR, 4 h/d for 15 days	A significant change in level of antioxidant enzymes and non-enzymatic antioxidants, and an increase in LPO.
(Shahin et al., 2013)	Mouse whole body	2450 MHz, PD of 33.5 $\mu W/cm^2$, SAR = 23 mW/kg, 2 h/day for 45 days	An increase in ROS, decrease in NO and antioxidant enzymes activities.
(Sharma et al., 2009)	Plant(mung bean) whole body	900 MHz, from cell phone, PD of 8.55 $\mu W/cm^2$; for 0.5; 1; 2, and 4 h	Increased level of MDA,H_2O_2 accumulation and root oxidizability, upregulation in the activities of SOD, CAT, ascorbate peroxidases, guaiacol peroxidases and GSHreductases in roots.
(Singh et al., 2012)	Plant (mung bean) whole body	900 MHz,from cell phone	The increased level of MDA, hydrogen peroxide and proline content in hypocotyls.
(Sokolovic et al., 2008)	Rat whole body	RFR from cell phone, SAR = 0.043–0.135 W/kg, for 20, 40 and 60 days	An increase in the brain tissue MDA and carbonyl group concentration. Decreased activity of CAT and increased activity of xanthine oxidase (XO). Melatonin treatment prevented the effects.

(*continued*)

Table 2. Continued

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Sokolovic et al., 2013)	Rat whole body	900 MHz, SAR = 0,043–0.135 W/kg,4 h/day for 29; 40 or 60 days,	The level of LPO and PO, activities of CAT, XO, number of apoptotic cells were increased in thymus tissue. An administration of melatonin prevented these effects.
(Suleyman et al., 2004)	Rat whole body	Cell phoneRFR,SAR = 0.52 W/kg,20 min/day for 1 month	MDA concentration was increased in brains.
(Tkalec et al., 2007)	Plant Lemna minor (duckweed)	400 and 900 MHz, 10, 23, 41 and 120 V/m, for 2 or 4 h	LPO and H_2O_2 content increased: CAT activity increased, pyrogallol peroxidase decreased.
(Tkalec et al., 2013)	Earthworm whole body	900 MHz, PD of 30–3800 μW/cm^2, SAR = 0.13–9.33 mW/kg, for 2 h	The protein carbonyl content was increased in all exposures above 30 μWc/m^2. The level of MDA was increased at 140μW/cm^2.
(Tök et al., 2014)	Rat whole body	2450 MHz, Wi-Fi RFR, 60 min/day for 30 days	Decreased GSH-Px activity. GSH-Px activity and GSH values increased after melatonin treatment.
(Tomruk et al., 2010)	Rabbit whole body	1800 MHz, GSM-like signal, 15 min/day for a week	Increase of MDA and ferrous oxidation in xylenol orange levels.
(Tsybulin et al., 2012)	Quail embryo *in ovo*	900 MHz, fromcell phone, GSM, PD of 0.024–0.21 μW/cm^2, intermittent for 14 days	Increased level of TBARS in brains and livers of hatchlings.
(Turker et al., 2011)	Rat partial body	2450 MHz, pulsed, SAR = 0.1 W/kg,1 h/day for 28 days	The increased level of LPO, the decreased concentrations of vitamin A, vitamin C and vitamin E. There was a protective effect of selenium and L-carnitine.
(Türedi et al., 2014)	Pregnant rat whole body	900 MHz, 13.7 V/m, 50 μW/cm^2, 1 h/day for 13–21 days of pregnancy	MDA, SOD and CAT values increased, GSH values decreased in exposed pups.
(Yurekli et al., 2006)	Rat whole body	945 MHz, GSM, PD of 367 μW/cm^2, SAR = 11.3 mW/kg	MDA level and SOD activity increased, GSH concentration was decreased.

*All effects were statistically significant (at least $p < 0.05$) as compared to control or sham exposed groups.

Table 3. Publications which reported positive findings on oxidative stress caused byRFR exposure of humans.

Reference	Biological system exposed	RFR exposure	Statistically significant effects reported*
(Abu Khadra et al., 2014)	Human male head	GSM 1800 MHz from cell phone, SAR = 1.09 W/kg, for 15 and 30 min	SOD activity in saliva increased.
(Garaj-Vrhovac et al., 2011)	Human whole body	3; 5.5; 9.4 GHz, pulsed, from radars	Increased level of MDA, decreased level of GSH.
(Hamzany et al., 2013)	Human head/whole body	RFR from cell phone a mean time of 29.6 h/month for 12.5 years	Increase in all salivary oxidative stress indices.
(Moustafa et al., 2001)	Human male body	Cell phone in a pocket in standby position, for 1; 2 or 4 h	Plasma level of LPO was increased, activities of SOD and GSH-Px in erythrocytes decreased.

*All effects were statistically significant (at least $p < 0.05$) as compared to control or sham-exposed groups.

significant overproduction of superoxide radical and NO radical, increased rates of lipid peroxidation and oxidative damage of DNA (Burlaka et al., 2013; Tsybulin et al., 2012). Notably, shorter exposures instead led to enhancement in embryonic development (Tsybulin et al., 2012, 2013). We demonstrated the favorable effects of shorter exposures also on the molecular level. Thus, after the short-time RFR exposure the DNA comets in embryonic cells were significantly shorter than in the control non-irradiated embryos, pointing to activation of mechanisms maintaining the integrity of DNA. The ''beneficial'' consequences of the irradiation could be explained by hormesis effect (Calabrese, 2008). However, one could hypothesize that the ''beneficial'' effects of the irradiation could be explained by the signaling action of free radicals induced at levels below the damaging concentrations. Obviously, any seemingly beneficial effect of external environmental impact should be treated with caution and possibly minimized before careful evaluation of the long-term consequences. Altogether, this gives a clear warning of the adverse health effects of

Table 4. Publications which reported no significant oxidative effectsafter RFR exposure.

Reference	Biological system exposed	RFR exposure	Effects reported
(Hook et al., 2004)	Mammalian cells *in vitro*	835.62 MHz (frequency-modulated continuous-wave, FMCW) and 847.74 MHz (code division multiple access, CDMA), SAR = 0.8 W/kg, for 20–22 h	FMCW- and CDMA-modulated RFR did not alter parameters indicative of oxidative stress.
(Ferreira et al., 2006a)	Rat whole body	800–1800 MHz, from cell phone	No changes in lipid and protein damage, and in non-enzymatic anti-oxidant defense in frontal cortex or hippocampus.
(Ferreira et al., 2006b)	Pregnant rat whole body	RFR from cell phone	No differences in oxidative parameter of offspring blood and liver, but increase in erythrocytes micronuclei incidence in offspring.
(Dasdag et al., 2003)	Rat whole body	Cell phone RFR, SAR = 0.52 W/kg, 20 min/day for 1 month	No alteration in MDA concentration.
(Demirel et al., 2012)	Rat whole body	3G cell phone RFR, ''standardized daily dose'' for 20 days	No difference in GSH-Px and CAT activity in eye tissues, in MDA and GSH levels in blood.
(Khalil et al., 2014)	Human head/whole body	Cell phone RFR (talking mode) for 15 or 30 min	No relationship between exposure and changes in the salivary oxidant/anti-oxidant profile.
(de Souza et al., 2014)	Human head/whole body	Cell phone RFR	No difference in the saliva from the parotid gland exposed to cell phone RFR to the saliva from the opposite gland of each individual.

low-intensity RFR, which could be evoked both by the direct oxidative damage and by disturbed cellular signaling.

Oxidative effects and non-cancer health effects of RFR

A new medical condition, so-called electrohypersensitivity (EHS), in which people suffer due to RFR exposure, has been described (Johansson, 2006). Typically, these persons suffer from skin- and mucosa-related symptoms (itching, smarting, pain, heat sensation), or heart and nervous system disorders after exposure to computer monitors, cell phones and other electromagnetic devices. This disorder is growing continuously: starting from 0.06% of the total population in 1985, this category now includes as much as 9–11% of the European population (Hallberg and Oberfeld, 2006). In Sweden, for example, EHS has become an officially recognized health impairment.

To that, a high percentage, up to 18–43% of young people, has recently been described to be suffering from headache/earache during or after cell phone conversations (Chu et al., 2011; Yakymenko et al., 2011). Likewise, a number of psychophysical and preclinical disorders including fatigue, irritation, headache, sleep disorders, hormonal imbalances were detected in high percent of people living nearby cell phone base transceiver stations (Buchner and Eger, 2011; Santini et al., 2002).

An allergy reaction to RFR in humans has been confirmed by a significant increase in the level of mast cells in skin of persons under exposure to electromagnetic devices (Johansson et al., 2001). Likewise, higher level of degranulated mast cells in dermis of EHS persons has been detected (Johansson, 2006). In turn, the activated mast cells can release histamine and other mediators of such reactions which include allergic hypersensitivity, itching, dermatoses, etc. Importantly, an implication of ROS in allergic reactions is rather clear nowadays. For example, in case of airway allergic inflammation, the lung cells generate superoxide in nanomolar concentrations following antigen challenges (Nagata, 2005). Then, mast cells generate ROS following aggregation of FcεRI, a high-affinity IgE receptor (Okayama, 2005). In addition, pollen NADPH oxidases rapidly increase the level of ROS in lung epithelium (Boldogh et al., 2005); and removal of pollen NADPH oxidases from the challenge material reduced antigen-induced allergic airway inflammation. Thus, it seems plausible that EHS-like conditions can be attributed at least partially to ROS overproduction in cells due to RFR exposures.

Oxidative effects and potential carcinogenicity of RFR

During recent years, a number of epidemiological studies indicated a significant increase in incidence of various types of tumors among long-term or ''heavy'' users of cellular phones (Yakymenko et al., 2011). Briefly, reports pointed to the increased risk in brain tumors (Cardis et al., 2010; Hardell and Carlberg, 2009; Hardell et al., 2007), acoustic neuroma (Hardell et al., 2005; Sato et al., 2011), tumors of parotid glands (Sadetzki et al., 2008), seminomas (Hardell et al., 2007), melanomas (Hardell et al., 2011) and lymphomas (Hardell et al., 2005) in these cohorts of people. To that, a significant increase in tumor incidence among people living nearby cellular base transceiver stations was also reported (Eger et al., 2004; Wolf and Wolf, 2007). Similarly, experimental evidences of cancer expansion in rodents caused by long-term low-intensity RFR exposure were published (Chou et al., 1992; Repacholi et al., 1997; Szmigielski et al., 1982; Toler et al., 1997). To that, activation of ODC was detected in RFR-exposed cells (Hoyto et al., 2007). ODC is involved in

processes of cell growth and differentiation, and its activity is increased in tumor cells. Although overexpression of ODC is not sufficient for tumorigenic transformation, an increased activity of this enzyme was shown to promote the development of tumors from pre-tumor cells (Clifford et al., 1995).

Significant overproduction of ROS leads to oxidative stress in living cells, induces oxidative damage of DNA and can cause malignant transformation (Halliwell and Whiteman, 2004; Valko et al., 2007). It is known that in addition to mutagenic effects, ROS play a role as a second messenger for intracellular signaling cascades which can also induce oncogenic transformation (Valko et al., 2006). Earlier we hypothesized (Burlaka et al., 2013) that low-intensity RFR exposure leads to dysfunctions of mitochondria, which result in overproduction of superoxide and NO, and subsequently to ROS-mediated mutagenesis. To that, it is well established that oxidative stress is associated with carcinogenesis; for instance, the oxidative stress elicited by Membrane-Type 1 Matrix Metalloproteinase is implicated in both the pathogenesis and progression of prostate cancer (Nguyen et al., 2011). Similarly, a progressive elevation in mitochondrial ROS production (chronic ROS) under both hypoxia and/or low glucose, which leads to stabilization of cells via increased HIF-2alpha expression, can eventually result in malignant transformation (Ralph et al., 2010). These data, together with the strong experimental evidences on activation of NADH oxidase under RFR exposure (Friedman et al., 2007) suggest that low-intensity RFR is a multifactorial stress factor for living cell, significant feature of which is oxidative effects and potential carcinogenicity as a result.

Conclusions

The analysis of modern data on biological effects of low-intensity RFR leads to a firm conclusion that this physical agent is a powerful oxidative stressor for living cell. The oxidative efficiency of RFR can be mediated via changes in activities of key ROS-generating systems, including mitochondria and non-phagocytic NADH oxidases, via direct effects on water molecules, and via induction of conformation changes in biologically important macromolecules. In turn, a broad biological potential of ROS and other free radicals, including both their mutagenic effects and their signaling regulatory potential, makes RFR a potentially hazardous factor for human health. We suggest minimizing the intensity and time of RFR exposures, and taking a precautionary approach towards wireless technologies in everyday human life.

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BRIEFING

European Parliament

Effects of 5G wireless communication on human health

SUMMARY

The fifth generation of telecommunications technologies, 5G, is fundamental to achieving a European gigabit society by 2025.

The aim to cover all urban areas, railways and major roads with uninterrupted fifth generation wireless communication can only be achieved by creating a very dense network of antennas and transmitters. In other words, the number of higher frequency base stations and other devices will increase significantly.

This raises the question as to whether there is a negative impact on human health and environment from higher frequencies and billions of additional connections, which, according to research, will mean constant exposure for the whole population, including children. Whereas researchers generally consider such radio waves not to constitute a threat to the population, research to date has not addressed the constant exposure that 5G would introduce. Accordingly, a section of the scientific community considers that more research on the potential negative biological effects of electromagnetic fields (EMF) and 5G is needed, notably on the incidence of some serious human diseases. A further consideration is the need to bring together researchers from different disciplines, in particular medicine and physics or engineering, to conduct further research into the effects of 5G.

The EU's current provisions on exposure to wireless signals, the Council Recommendation on the limitation of exposure of the general public to electromagnetic fields (0 Hz to 300 GHz), is now 20 years old, and thus does not take the specific technical characteristics of 5G into account.

In this Briefing

EPRS | European Parliamentary Research Service

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Background

Under the EU digital single market strategy, the European Commission presented new policy measures in its 2016 communication on Connectivity for a Competitive Digital Single Market – Towards a European Gigabit Society. The Commission's aim is to advance the digitalisation of the EU and to increase its competitiveness by launching networks with much higher capacities, with 5G as a building block to achieve a 'gigabit society' by 2025. Its main characteristics would enable the internet of things, which means that billions of connections between devices share information.[1] The Commission has established the following connectivity targets for 2025:

- schools, universities, research centres, hospitals, main providers of public services and digitally intensive enterprises should have access to internet download/upload speeds of one gigabit of data per second;
- urban and rural households should have access to connectivity of download speed of at least 100 megabits per second;
- urban areas, major roads and railways should have uninterrupted 5G coverage.

The '5G for Europe: An action plan' presents measures for timely and coordinated deployment of 5G networks in Europe through a partnership between the Commission, Member States, and industry. This initiative concerns all private and public stakeholders, in all EU Member States.

The connectivity objective has been regulated by the adoption of the European Electronic Communication Code (EECC) at the end of 2018, under which EU Member States have to authorise the use of the new 5G frequency bands at 700 MHz, 3.5 GHz and 26 GHz[2] and reorganise them by the end of 2020,[3] in line with the EECC. This decision enables the take-up of 5G services in the Union.

According to the European 5G observatory, supported by the European Commission, at the end of September 2019, 165 trials had been carried out in the European Union and 11 Member States had already published their national 5G action plans.

Challenges and opportunities of 5G

Advantages

Allowing much larger volumes of data to be transported more quickly, and reducing response time, 5G will enable instantaneous connectivity to billions of devices, the internet of things and a truly connected EU population. Furthermore, millions of jobs and billions of euros could be expected to be gained from the digital economy.

The possibilities that the fifth generation of wireless communication offers, such as downloading or uploading one gigabit of data per second, may provide advantages, for instance, for the military and medical research, which could benefit from having access to such extremely high gigabit connectivity. However, the military, hospitals, the police and banks continue to use wired connections, at least for their most essential communications, mainly for security reasons. Wired networks generally offer a faster internet speed and are considered to be more secure. This is due to the fact that a wired network is only accessible through a physical cable connection, whereas with wireless networks, the signal may be broadcast outside the physical premises. Wired connection offers more control than radio or wifi, because such organisations already provide protection for servers and internal IT facilities within their physical locations, taking advantage of almost 100 % of the bandwidth, which also reduces response times. That also contributes to increased security.

Disadvantages

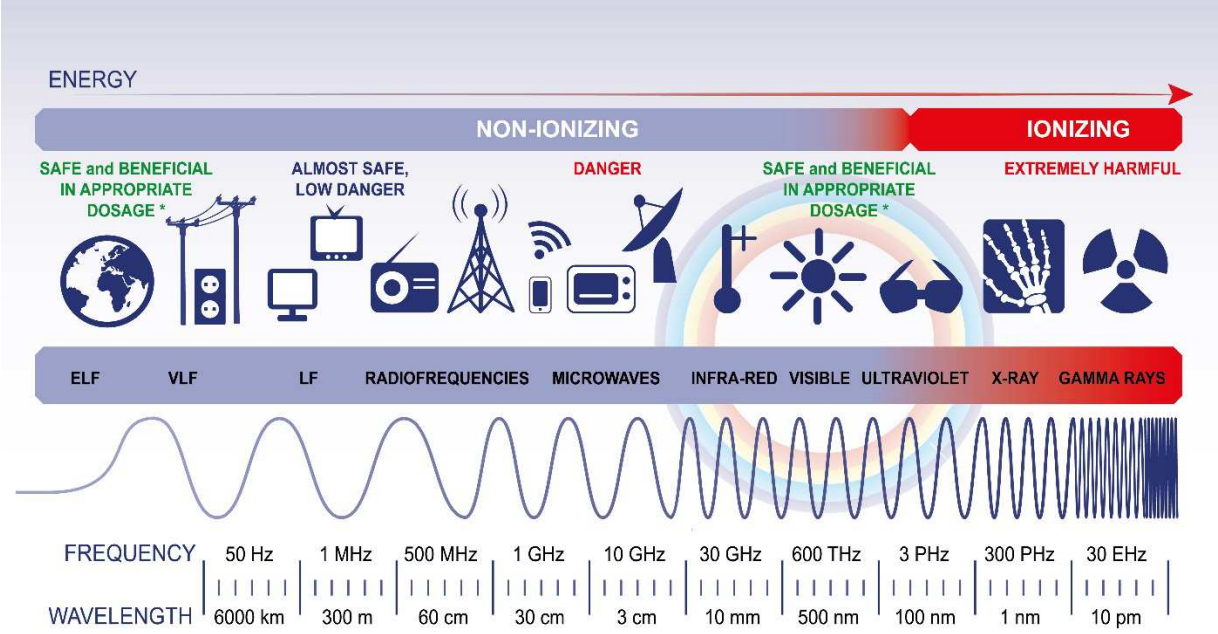
Because it is more complex and requires a denser coverage of base stations[4] to provide the expected capacity, 5G will cost much more to deploy than previous mobile technologies. According to European Commission estimates, to reach the target, including 5G coverage in all urban areas, this cost is estimated at around €500 billion by 2025.

Questions remain unanswered as to what 5G actually is, what it is for, whether it has impacts on human health and environment, whether it is secure, whether it offers good value for money or whether anyone will be prepared to pay for it.[5] As an alternative, according to some experts,[6] fibre optics would be more secure, safe and offer higher speed than 5G. However, fibre optics are not wireless.

Difference between 5G and current technology

Employing millimetre waves and higher frequencies than previous technologies, 5G needs a much more extensive network of antennas and other transmitting devices. Electromagnetic fields (EMF) are invisible areas of energy,[7] measured in hertz (Hz). Longer wavelengths with lower frequency are less powerful in terms of energy, while shorter wavelengths at higher frequencies are more powerful. Depending on the frequency, there are two categories of EMF: ionising and non-ionising radiation (see Figure 1).

Figure 1 – Electromagnetic spectrum



Source: Polina Kudelkina / Shutterstock.com.

Ionising radiation (mid to high-frequency) includes ultraviolet rays, x-rays and gamma rays. The energy from ionising radiation can damage human cells and cause cancer. Non-ionising radiation has lower frequencies and bigger wavelengths. Many experts are of the opinion that non-ionising radiation produces only thermal effects, or tissue heating, and that at high exposure levels, temperature-sensitive biological structures, including humans, and processes can become damaged.

Microwave and millimetre wavelength radiation is non-ionising. Millimetre wave ranges from around 10 to 1 millimetre. This is a very effective spectrum with large bandwidth, but it is also very sensitive to external variables and can be subject to interference from walls, trees or even rain.

For the first time, 5G will use millimetre waves in addition to the microwaves that have been used to date in 2G, 3G and 4G technology. Due to the limited coverage, to implement 5G, cell antennas will have to be installed very close to one another, which will result in constant exposure of the population to millimetre wave radiation. Use of 5G will also require new technologies to be employed, such as active antennas capable of beam-forming, massive inputs and outputs.[8] With higher frequencies and shortened ranges, base stations will be more closely packed into an area, to

provide complete coverage and avoid 'not-spots'. This could mean possible ranges of 20-150 metres with smaller coverage areas per 'small cell.[9] A cell radius of 20 metres would imply about 800 base stations per square kilometre (or 'small area wireless access points' (SAWAPs), the term used in the EECC). This contrasts with 3G and 4G technologies, which use large or 'macro' cells, offering ranges of 2-15 kilometres or more, and therefore covering a larger area but allowing fewer simultaneous users since they have fewer individual channels.[10]

Furthermore, 5G will employ higher frequencies[11] than previous 'G' networks and greater bandwidth which will enable users to transfer wireless data faster.

Regulation of electromagnetic fields and 5G exposure

European Union

Primary responsibility for protecting the population from the potential harmful effects of EMF falls to the governments of EU Member States under Article 168 of the Treaty on the Functioning of the European Union. In 1996, the World Health Organization (WHO) established the International EMF Project to evaluate the scientific evidence of possible health effects of EMF in the frequency range from 0 to 300 GHz. It has elaborated 'model legislation' to offer a legal framework for implementing protection programmes against non-ionising radiation.

The International Commission on Non-Ionising Radiation Protection (ICNIRP), a non-governmental organisation formally recognised by WHO, issues guidelines for limiting exposure to electric, magnetic and electromagnetic fields (EMF), which are revised periodically. In the EU, **Council Recommendation** 1999/519/EC, of 12 July 1999, on the limitation of exposure of the general public to EMF (0 Hz to 300 GHz), follows these guidelines.

As the Council Recommendation is the common protective framework guiding EU Member States and setting basic restrictions and reference levels, depending on frequency, the following physical quantities specify basic restrictions on electromagnetic fields:

- between 0 and 1 Hz, basic restrictions are provided for magnetic flux density for static magnetic fields (0 Hz) and current density for time-varying fields12 up to 1 Hz, to prevent effects on the cardiovascular and central nervous system;
- between 1 Hz and 10 MHz, basic restrictions are provided for current density13 to prevent effects on nervous system functions;
- between 100 kHz and 10 GHz, basic restrictions on the specific absorption rate (SAR) are provided to prevent whole-body heat stress and excessive localised tissue heating. In the 100 kHz to 10 MHz range, restrictions on both current density and SAR are provided;
- between 10 GHz and 300 GHz, basic restrictions on power density are provided to prevent tissue heating on or near the surface of the human body.

While these exposure limits are non-binding on EU Member States, some Member States have nevertheless adopted stricter limits than those recommended above.

The recommendation encourages Member States to establish a common protective framework and inform the public of the health impact of electromagnetic fields, as well as to harmonise national approaches for measurement. The Council suggests that the European Commission keep possible health effects under review.

The **European Environment Agency** (EEA) has long advocated precaution concerning EMF exposure, pointing out that there were cases of failure to use the precautionary principle in the past, which have resulted in often irreversible damage to human health and environments. Appropriate, precautionary and proportionate actions taken now to avoid plausible and potentially serious threats to health from EMF are likely to be seen as prudent and wise from future perspectives. The EEA requests that EU Member States do more to inform citizens about the risks of EMF exposure, especially to children.

In its 2 April 2009 resolution, the European Parliament urged the Commission to review the scientific basis and adequacy of the EMF limits in Recommendation 1999/519/EC and to report back. Parliament also requested that the Scientific Committee on Emerging and Newly Identified Health Risks carry out a review of the EMF limits. Parliament requested consideration of the biological effects, acknowledging the results of studies that reveal harmful effects at lowest levels of electromagnetic radiation, as well as calling for active further research and consequently development of solutions to negate or reduce pulsations used for transmission. It suggested that the Commission elaborate a guide to available technology options for reducing exposure to EMF in coordination with experts from Member States and the industries concerned.

The European Commission **Scientific Committee on Emerging and Newly Identified Health Risks** (SCENIHR) has a mandate to evaluate the risks of electromagnetic fields and periodically reviews the scientific evidence available to assess whether it still supports the exposure limits proposed in Council Recommendation 1999/519/EC. In its latest opinion of January 2015, SCENIHR suggested that there is a lack of evidence that EMF radiation affects cognitive functions in humans or contributes to an increase of the cases of cancer in adults and children. However, the International EMF Alliance (IEMFA) suggested that many members of SCENIHR could have a conflict of interests, as they had professional relationships with or received funding from various telecom companies.

Consequently, the Scientific Committee on Health, Environmental and Emerging Risks (SCHEER), replacing the former Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), indicated a preliminary estimate of the importance of 5G as high, in a statement in December 2018. Furthermore, it evaluates the scale, urgency and interactions (with ecosystems and species) of possible hazard as high. It suggested that there could be biological consequences from a 5G environment, due to the fact that there is a lack of 'evidence to inform the development of exposure guidelines to 5G technology'.

Council of Europe

Council of Europe Resolution 1815 (2011) points to the potential health effects of the very low frequency of electromagnetic fields surrounding power lines and electrical devices, which are the subject of ongoing research and public debate. It also states that some non-ionising frequencies appear to have more or less potentially harmful, non-thermal, biological effects on humans, other animals and plants, even when exposed to levels that are below the official threshold values. The resolution identifies young people and children as particularly vulnerable groups and suggests that there could be extremely high human and economic costs if early warnings are neglected. The issue of possible environmental and health effects of electromagnetic fields is considered to have clear parallels with other current issues: the licensing of medication, chemicals, pesticides, heavy metals or genetically modified organisms. The resolution highlights that the independence and credibility of the scientific expertise employed is crucial for a transparent and balanced assessment of possible negative effects on human health and environment. The resolution recommends:

- taking all reasonable measures to reduce exposure to EMF (especially from mobile phones) and particularly to protect children and young people who seem to be most at risk of developing head tumours;
- reconsidering the scientific basis for the present standards on exposure to electromagnetic fields set by the International Commission on Non-Ionising Radiation Protection, which have serious limitations;
- distributing information and awareness-raising campaigns on the risks of potentially harmful long-term biological effects on the environment and on human health, especially targeting children, teenagers and young people of reproductive age;
- giving preference to wired internet connections (for children in general and particularly in schools), and strictly regulating the use of mobile phones by schoolchildren on school premises;
- increasing public funding of independent research to evaluate health risks.

European Parliament

A resolution of 2 April 2009 on health concerns associated with electromagnetic fields urged the European Commission to review the scientific basis and adequacy of the EMF limits in Recommendation 1999/519/EC and to report back. It also requested that the Scientific Committee on Emerging and Newly Identified Health Risks carries out a review of the EMF limits.

Research on EMF and 5G effects on human health

The academic literature on EMF exposure effects and 5G in particular is growing rapidly. Some research papers support possible health risks, while others do not.

The WHO[14]/International Agency for Research on Cancer (IARC) classified radiofrequency EMF as possibly carcinogenic to humans in 2011. The IARC has recently prioritised EMF radiation for review in the next five years (2020-2024).

A section of the scientific community – mainly doctors and researchers in medical sciences – argues that there are negative impacts from EMF exposure and that these will increase with the implementation of 5G. A **5G appeal** was presented to the United Nations in 2015, and to the European Union from 2017, with an increasing number of scientists' signing (268 scientists and medical doctors as of 18 December 2019). The signatories state that with the increasingly extensive use of wireless technology, especially when 5G is deployed, nobody could avoid exposure to constant EMF radiation because of the huge number of 5G transmitters with an estimated 10 to 20 billion connections (to self-driving cars, buses, surveillance cameras, domestic appliances, etc.). In addition, the appeal states that a large number of scientific publications illustrate EMF exposure effects such as an elevated risk of cancer, genetic damage, learning and memory deficits, neurological disorders, etc. The appeal points out not only harm to humans, but also to the environment.

The appeal recommends a moratorium on the deployment of 5G for telecommunications until potential hazards for human health and the environment have been fully investigated by scientists independent of industry. They urge the EU to follow Resolution 1815 of the Council of Europe, and demand that a new assessment is carried out by an independent task force.

Ethics in research

The European Code of Conduct for Research Integrity (last revised in 2017) sets out principles of research integrity, criteria for good research practice, and describes how to prevent violations of research integrity.

The principles it states are the following:

- **Reliability** in ensuring the quality of research, reflected in the design, the methodology, the analysis and the use of resources.
- **Honesty** in developing, undertaking, reviewing, reporting and communicating research in a transparent, fair, full and unbiased way.
- **Respect** for colleagues, research participants, society, ecosystems, cultural heritage and the environment.
- **Accountability** for the research from idea to publication, for its management and organisation, for training, supervision and mentoring, and for its wider impacts.

In this regard, some scientists consider it necessary to establish new exposure limits that take account of the new characteristics of exposure. Such limits should be based on the biological effects of EMF radiation, rather than on the energy-based specific absorption rate.

Non-ionising radiation, which includes radiation from mobile phones and 5G, is perceived as harmless in general, due to its lack of potency. However, some of the above-mentioned scientists point out that, in the particular case of 5G, the issue is not the potency, but the pulse,[15] the frequency to which the whole population will be exposed due to the dense network of antennas and the estimated billions of simultaneous connections. As 5G employs a very high level of pulsations, the idea behind 5G is to use higher frequencies, which allows such high levels of pulsation, in order to

carry very large amounts of information per second. Studies show that pulsed EMF are in most cases more biologically active and therefore more dangerous than non-pulsed EMF. Every single wireless communication device communicates at least partially via pulsations, and the smarter the device, the more pulsations. Consequently, even though 5G can be weak in terms of power, its constant abnormal pulse radiation can have an effect. Along with the mode and duration of exposures, characteristics of the 5G signal such as pulsing seem to increase the biologic and health impacts of exposure, including DNA damage, which is considered to be a cause of cancer. DNA damage is also linked to reproductive decline and neurodegenerative diseases.

A 2018 review of more recently published peer-reviewed articles on the biological and health effects of radio frequency EMF, including 5G, also verifies the available evidence on the effects of millimetre waves. The review concludes that evidence of the biological properties of radiofrequency EMF are accumulating progressively and even though they are, in some cases, still preliminary or controversial, point to the existence of multi-level interactions between high-frequency EMF and biological systems, and to the possibility of oncological and non-oncological (mainly reproductive, metabolic, neurological, microbiological) effects. Moreover, it points out that the wide and increasing density of wireless devices and antennas raises particular concerns. Taking this into account, '... although the biological effects of 5G communication systems are very scarcely investigated, an international action plan for the development of 5G networks has started, with a forthcoming increase in devices and density of small cells, and with the future use of millimetre waves'. However, there are indications that millimetre waves can increase skin temperature, promote cellular proliferation, and inflammatory and metabolic processes. According to the review, further studies are necessary to improve independent exploration of the health effects of radio frequency EMF in general and of millimetre waves in particular.[16]

Far less research exists to determine the effects of 5G technologies on humans and the environment, according to another review of studies published in 2018. Considering the already existing complex mix of lower frequencies, it argues that in addition to those, the expected higher frequency 5G radiation would cause negative impacts on physical and mental public health. Concretely in the case of millimetre waves, it analyses the results of studies which find effects on the skin, eyes, and immune system, and bacterial antibiotic resistance. The review suggests that the effects of radiofrequency EMF will be problematic to sort out epidemiologically, as no unexposed control group will remain. The study consequently calls for precaution in the deployment of this new technology. The author argues that while physicists and engineers give assurances that the only measure to harm health is heat, medical scientists indicate that there are other mechanisms whereby cellular functioning can be disrupted by non-thermal exposures to radiofrequency.

A 2016 review of scientific articles, covering experimental data on the oxidative effects of low-intensity radiofrequency radiation in living cells, finds that, among 100 currently available peer-reviewed studies (18 *in vitro* studies, 73 studies in animals, 3 studies in plants and 6 studies in humans), '... dealing with oxidative effects of low-intensity radiofrequency radiation, in general, 93 confirmed that radiofrequency radiation induces oxidative effects in biological systems'. More precisely, in 58 studies of laboratory rats, 54 show positive results, and 4 of 6 studies in humans were positive. In addition, 17 of the 18 of the *in vitro* studies were positive, including two on human spermatozoa and two on human blood cells. According to the authors, 'The analysis of modern data on biological effects of low-intensity radiofrequency radiation (RFR) leads to a firm conclusion that this physical agent is a powerful oxidative stressor for living cells'.

A 2018 study carried out on animals, showed that electromagnetic radiation emitted by wifi networks can lead to hyperglycaemia, increased oxidative stress and impaired insulin secretion in rat pancreatic islets. A method of creating diabetes (which can lead to kidney deficiency in the long term) in laboratory rats is to expose them, even briefly, to 2.4 Ghz.

A 2019 report of the Swedish Radiation Safety Authority's Scientific Council on Electromagnetic Fields considers two large animal studies: the US National Toxicology Program (NTP) study and the

Italian Falcioni et al. study, which analyse the relationship between radio wave exposure and schwannoma of the heart in male rats.[17] The report concludes that there is some inconsistency in the results between the two studies and that no new causal relationship between EMF exposure and health risks was established. It recommends that further research is important, particularly regarding long-term effects and especially since the entire population will be exposed. It points out that a possible relationship between radio wave exposure and oxidative stress should be a subject of further research, as well as the association between weak low-frequency magnetic fields and childhood leukaemia, as observed in epidemiological studies.

The scientific community reaction in response to this report, is illustrated in the recent 'Commentary on the utility of the National Toxicology Program study on cell phone radiofrequency radiation data for assessing human health risks despite unfounded criticisms aimed at minimizing the findings of adverse health effects.' The author states that the NTP study was designed to test the hypothesis that, at non-thermal exposure intensities, mobile phone radiation could not lead to adverse health effects, and to provide data for assessment of health risks caused by any detected toxic or carcinogenic effects, as little was known about long-term exposure to mobile phone radiation health effects. Regarding the NTP study results, among others, the author defends the use of animal studies that can eliminate the need to wait until enough human cancer data are available before implementing strategies to protect public health. According to the author, the intensity of exposure in the brains of rats in the NTP study were similar to potential human mobile phone exposures.

In turn, a 2019 review of 94 articles, funded by Deutsche Telekom, states that the '... available studies do not provide adequate and sufficient information for a meaningful safety assessment, or for the question about non-thermal effects. There is a need for research regarding local heat developments on small surfaces, e.g., skin or the eye, and on any environmental impact. There was no consistent relationship between power density, exposure duration, or frequency, and exposure effects'.

There is no noticeable increase in everyday EMF exposure since 2012, despite the increasing use of wireless communication devices, according to another review of studies from 2019. Nevertheless, it remains unclear how well these studies of everyday exposure represent the population's absorbed radiofrequency EMF dose. This study maintains the urgent need for better quantification of the population's absorbed radiofrequency EMF dose from their own communication devices.

Stakeholders' views

Considering the huge estimated investment, the mobile telecommunications industry needs to convince governments of 5G's economic and social benefits and perform widespread marketing campaigns. 'It suits the industry if policy-makers believe that there is a race between nations to be the first to launch 5G services'.[18]

The EU telecommunications industry continues to state that the weight of evidence regarding harm from EMF exposures is inconclusive. The 5G Infrastructure Public Private Partnership (5G PPP), a joint initiative between the European Commission and European information and telecommunications (ICT) industry (ICT manufacturers, telecommunications operators, service providers, SMEs and research institutions), supports research and innovation to develop 5G networks that comply with international standards and regulations and develops systems designed to operate below the safe health limits of electromagnetic emissions.[19] However, it does not refer to the biological impacts of 5G radiation.

Nevertheless, according to the IEMFA, a need to measure real potential exposure to 5G and update the safety limits of such exposure does exist. The alliance calls for more research and scientific consent along these lines. It maintains that scientists with experience of long research into EMF health effects should be included in the SCENIHR, following the demands of the 2015 IEMFA complaint.[20]

The road ahead for 5G

There is an urgent need for economic recovery and leadership in implementing digital technologies; and for long-lasting economic growth in Europe. However, it is necessary to consider any possible collateral negative impacts. Taking the economic aspects of 5G into account, there are many challenges ahead on the path to achieving a 'gigabit society', such as for instance industry concerns whether the plans for commercial launch of 5G in 2020 will be fulfilled, considering the technical complexity and the necessary investment.

Other concerns relate to the creation of sufficient demand for 5G, security and health, safety and environmental issues.[21] These need wider public awareness and consent, however this is doubly salient regarding the possible negative health impacts due to the inescapability of constant exposure of citizens in a 5G environment. The recent academic literature illustrates that continuous wireless radiation seems to have biological effects especially considering the particular characteristics of 5G: the combination of millimetre waves, a higher frequency, the quantity of transmitters and the quantity of connections. Various studies suggest that 5G would affect the health of humans, plants, animals, insects, and microbes – and as 5G is an untested technology, a cautious approach would be prudent. The UN Universal Declaration of Human Rights, the Helsinki Accords and other international treaties recognise that informed consent prior to interventions that might affect human health is an essential, fundamental human right, which becomes even more controversial when considering children's and young people's exposure.

A certain divergence exists among scientists on the potential negative effects of EMF exposure and 5G. Experts rarely possess complementary backgrounds in both physics or engineering and medicine, therefore more complete scientific expertise could be achieved by combining research teams experienced in all relevant disciplines. Optical fibre technology has been suggested by some experts as a secure alternative to 5G, because the signal is confined within the fibre. Its potential is much higher than that of 5G and there is no comparison between optical fibre and wireless. Investment in optical fibre can be upgraded to superior speeds in the future, whereas it is necessary to change the whole system for wireless technologies.

According to the 2019 study '5G deployment: State of Play in Europe, USA and Asia' prepared for the European Parliament, long-term technology research is essential. 'One key problem is the unusual propagation phenomena, especially controlling and measuring radio frequency EMF exposure with Multiple Input Multiple Output (MIMO) at millimetre wave frequencies for the handset and the base station. The technology presents challenges to the current level of expertise (based on previous generations of mobile cellular radio engineering) both for suppliers and standards organisations who must incorporate the specifications in future 5G standards'. The study states that the main problem seems to be that it is not currently possible to accurately simulate or measure 5G emissions in the real world.

To understand potential mechanisms underlying possible health effects of EMF better and to characterise population levels of exposure, the Generalised EMF Research using Novel Methods (GERoNiMO) project was launched in 2014, funded under the EU's Seventh Framework Programme for Research and Technological Development to address pertinent questions on EMF and health. It proposes an integrated approach using epidemiological studies, exposure assessment techniques, mechanistic and animal models, and expert networks applying novel methods when possible. The project ended in 2018.

The European Commission has not yet conducted studies on the potential health risks of the 5G technology.[22]

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ENDNOTES

1 Industry estimates that 5G capacity will be 40 times that offered by current 4G technology. See M. Negreiro, Towards a European gigabit society Connectivity targets and 5G, EPRS, June 2017.

2 A Megahertz (MHz) is a million cycles per second and a Gigahertz (GHz) pulses at a billion cycles per second. In order to carry data at faster speeds, each new generation of telecommunications uses higher frequency radio waves.

3 See 5G deployment agenda.

4 In addition to spectrum licensing costs, a large share of the cost will be due to the much denser network needed, rolling out the small cells necessary to transmit signals in much higher frequency bands.

5 See '5G Deployment: State of Play in Europe, USA and Asia', European Parliament, June 2019.

6 'Fiber is safer, faster, more reliable, and far more cyber secure and energy efficient than wireless.' R. M. Powell. See also similar opinions from experts such as T. Schoechle and P. Héroux.

7 Also known as waves or radiation.

8 Which would make measuring radiation exposures even more difficult.

9 Usually, the longer the wavelength the further it travels. The higher frequency millimetre wavelengths of 5G travel only a few hundred metres.

10 See '5G Deployment: State of Play in Europe, USA and Asia', European Parliament, June 2019.

11 Radio frequency includes a continuum of the electromagnetic spectrum wavelengths from around 3 kHz to 300 GHz. The wavelengths in the radio frequency vary from hundreds of metres to fractions of a centimetre. The frequencies used in current digital communications have shorter wavelengths and faster data transfer. This enables the transfer of more data simultaneously.

12 Time-varying means that as time (t) increases, the magnetic field changes.

13 The amount of charge per unit of time that flows through a unit area of a chosen cross section.

14 According to the WHO, EMFs of all frequencies represent one of the most common and fastest growing environmental influences. Exposure of the whole population to EMFs will continue to increase along with technological advance.

15 An electromagnetic pulse is a short blowout of electromagnetic energy. Its origin can be manmade and can occur as a radiated, electric, or magnetic field or a conducted electric current.

16 Millimetre waves, which will be employed by 5G, are mostly absorbed within a few millimetres of human skin and in the surface layers of the cornea. Short-term exposure can have adverse physiological effects in the peripheral nervous system, the immune system and the cardiovascular system.

17 For more information on the two studies, see also the EPRS briefing on Mobile phones and health, March 2019.

18 See '5G Deployment: State of Play in Europe, USA and Asia', European Parliament, June 2019

19 According to the limits established by Council Recommendation 1999/519/EC.

20 In an open letter from 2011 to the Health and Consumer Policy Commissioner, public interest stakeholders expressed their concerns over the lack of transparency and pluralism in the evaluation of evidence by SCENIHR, and other EU risk assessment committees, of the health risks of non-ionising EMF radiation (see EPRS Briefing, March 2019).

21 See EPRS briefing 'Towards a European gigabit society: Connectivity targets and 5G', June 2017.

22 See answer given by the European Commission to parliamentary question E-005128/2018(ASW). See also 'MEP: Commission 'irresponsible' on 5G health risks', Euractiv, 12 December 2019.

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Habilitetserklæring

Undertegnede redegør herved for eventuelle økonomiske interesser inden for medicinalvirksomhed mv.

	Nej	Ja	Hvis ja, hvilke
Jeg har honoreret bibeskæftigelse i sådan virksomhed m.v	X		
Jeg er personlig aktionær, har patentrettigheder e.l. i sådan virksomhed	X		
Den arbejdsplads, jeg er ansvarlig for, modtager støtte fra sådan virksomhed		X	Se bilag

Undertegnede forpligter mig endvidere til at orientere Sundhedsstyrelsen, såfremt opgaven anvendes som meriterende i forbindelse med et uddannelses- eller efteruddannelsesforløb.

På tro og love Kbh den 6/10 2007

Christoffer Johansen
(underskrift)

Opgave: KONSULENT VEDR. IKKE-IONISERENDE STRÅLING
(blokbogstaver)

Journalnummer:

Bilag til habilitetserklæring til Sundhedsstyrelsen
Christoffer Johansen
Pr. januar 2010
København

Christoffer Johansens habilitetserklæring vedrørende finansiering og data fra industri, der er involveret i produktion, transmission og distribution af elektricitet samt mobiltelefonoperatører og lignende industri, der producerer, forhandler eller anvender genstande som skaber elektromagnetiske felter i hele frekvensområdet.

Finansiering

Jeg har i perioden 1994 til 2004 modtaget midler fra Dansk Energi for at undersøge risiko for en række sygdomme blandt danskere med ansættelse ved danske el-selskaber, samt midler til at undersøge risiko for kræft blandt børn med bopæl tæt på elektriske højspændingsinstallationer.

Jeg har i 2006 fået en fornyet bevilling fra Energinet.dk for perioden 2006 til 2008 med henblik på at opdatere de allerede gennemførte undersøgelser.

Jeg har i 1994/95 modtaget fondsmidler fra det daværende Tele Danmark Mobil og Sonofon med henblik på at gennemføre en undersøgelse af kræftrisiko blandt brugere af mobiltelefoner.

Jeg har fire gang siden 1994 modtaget et personligt honorar for at udfærdige et notat, der i form af en VVM rapport, gennemgår den videnskabelige litteratur inden for forskningsområdet elektromagnetiske felter og helbredseffekter hos mennesker, sidste gang i 2010. Notatet er udarbejdet som en del af en såkaldt VVM rapport i forbindelse med forskellige udbygninger af højspændingsnettet i Danmark.

Data

Jeg har i perioden 1994 - 1996 modtaget information om samtlige ansatte ved de 104 danske elselskaber med oplysning om navn, CPR nummer, arbejdsfunktion, stillingsbetegnelse og ansættelses periode(r).

Jeg har i 1996 modtaget data fra det daværende TeleDanmarkMobil og Sonofon vedrørende samtlige abonnenters navne, adresse, mobiltelefon type og abonnements periode(r).

I 2007/2008 vil vi her på instituttet, fra de største mobiltelefon operatører få oplysninger om mobiltelefon forbrug blandt en større gruppe danskere der indgår i en international forløbsundersøgelse.

Vi vil desuden fra de samme mobiltelefon operatører modtage de samme informationer om børn med hjerne svulster og raske kontrolbørn, der indgår i en nordisk undersøgelse af risiko for hjernetumorer blandt børn og unge.

Jeg er medarbejder i disse to projektgrupper, mens det videnskabelige og økonomiske ansvar ligger hos Dr Joachim Schuez.

International Agency for Research on Cancer

World Health Organization

IARC Monographs on the Identification of Carcinogenic Hazards to Humans

Report of the Advisory Group to Recommend Priorities for the *IARC Monographs* during 2020–2024

Non-ionizing radiation (radiofrequency) and extremely low-frequency magnetic fields

Radiofrequency electromagnetic fields (RF-EMF) were evaluated by the *IARC Monographs* as *possibly carcinogenic to humans* (Group 2B) (IARC, 2013e), on the basis of limited evidence of an increased risk of glioma. Extremely low-frequency magnetic fields (ELF-MF) were evaluated as *possibly carcinogenic to humans* (Group 2B) (IARC, 2002), on the basis of *limited evidence* of an increased risk of childhood leukaemia.

Exposure Data

Human exposures to RF-EMF can occur from use of personal devices (e.g. cell phones, cordless phones, and Bluetooth) and from environmental sources such as cell phone base stations, broadcast antennas, and medical applications. More than 5 billion people now have access to cell phone devices, and the technology is constantly evolving. Use has also expanded rapidly in low- and middle-income countries, where more than 75% of adults now report owning a cell phone; in high-income countries, the proportion is 96% (Pew Research Center, 2018).

Cancer in Humans

Since the previous *IARC Monographs* evaluation, several new epidemiological studies have been published on the association between RF-EMF and cancer, although the evidence remains mixed. In the Million Women Study cohort, there was no evidence of increased risk of glioma or meningioma, even among long-term users. There was an increased risk of acoustic neuromas with long-term use and a significant dose–response relationship (Benson et al., 2013). Updated follow-up in the Danish nationwide subscribers study did not find increased risks of glioma, meningioma, or vestibular schwannoma, even among those with subscriptions of 10 years or longer (Frei et al., 2011; Schüz et al., 2011). New reports from case–control studies that assessed long-term use also found mixed results; for example, increased risks of glioma and acoustic neuroma were reported by Hardell & Carlberg (2015) and Hardell et al. (2013), but no evidence of increased risks for these tumours were reported by Yoon et al. (2015) and Pettersson et al. (2014). Röösli et al. (2019) recently reviewed these new data. Several large-scale studies are still in progress and should report results within the next few years. Mobi-Kids is a multicentre case–control study of brain tumours in those aged 10–24 years. Cohort Study of Mobile Phone Use and Health (COSMOS) is a new European cohort of adult cell phone users. There will also be updated results from the Million Women Study.

Cancer in Experimental Animals

New data in experimental animals for exposure to RF-EMF have been published since the previous *IARC Monographs* evaluation. The large study by the United States National Toxicology Program found an increased risk of malignant schwannomas of the heart in male rats with high exposure to radiofrequency radiation at frequencies used by cell phones, as well as possible increased risks of certain types of tumours in

the brain and adrenal glands, but no increased risks in mice or female rats (NTP, 2018a, b). Another study in experimental animals also found an increase in schwannomas of the heart in highly exposed male rats and a possible increase in gliomas in female rats (Falcioni et al., 2018).

Mechanistic Evidence

The previous IARC evaluation concluded that there was weak evidence that radiofrequency radiation was genotoxic but that there was no evidence for mutagenicity (IARC, 2013e). Although there have been many new publications from a wide variety of experiments, uncertainty remains about the mechanisms, and there are few systematic reviews of the new data (Kocaman et al., 2018).

Although a future evaluation could be broadened to consider exposure to all non-ionizing radiation (including ELF-MF), ELF-MF were evaluated by IARC as *possibly carcinogenic to humans* (Group 2B), and the Advisory Group did not recommend an update, because of a lack of new informative epidemiological findings, no toxicological evidence, and little supporting mechanistic evidence.

Key References

The following key references were also identified: Coureau et al. (2014); Carlberg & Hardell (2015); Pedersen et al. (2017).

Recommendation for non-ionizing radiation (radiofrequency): High priority (and ready for evaluation within 5 years)

Recommendation for extremely low-frequency magnetic fields: No evaluation

Nuclear industry work

Different types of ionizing radiation have been evaluated repeatedly by the *IARC Monographs* programme (IARC, 2000b, 2012f), and all types have been classified as *carcinogenic to humans* (Group 1); overall evaluations are based on different evidence streams, often including *sufficient evidence* in humans for several cancer sites. New research in recent years has confirmed increased risks per unit of exposure to ionizing radiation for cancer sites and groups of cancer sites that have already been linked with ionizing radiation. No specific evaluation has been made in respect of work in the nuclear industry, which represents a specific exposure condition for agents already classified as *carcinogenic to humans* (Group 1).

Key References

The following key references were identified: Lee et al. (2015c); Leuraud et al. (2015); Richardson et al. (2015); Schubauer-Berigan et al. (2015); Grellier et al. (2017).

Recommendation: No evaluation

Corte di Cassazione n. 17438/2012 - l'uso massiccio del cellulare può avere "un ruolo almeno concausale" nella genesi di alcuni tumori – 12.10.2012. - - Giudice di Pace

claps carlo

 Con la sentenza n. 17438/2012, la Corte di Cassazione ha stabilito che l'uso massiccio del cellulare, per parecchie ore al giorno, e per un lungo periodo di anni, possa avere "un ruolo almeno concausale" nella genesi di alcuni tumori dei nervi cranici. Gli Ermellini hanno precisato che gli studi indipendenti condotti tra il 2005 e il 2009, che hanno evidenziato un maggiore rischio di insorgenza di neoplasie negli utilizzatori 'forti' di telefonia mobile, sono, correttamente, stati considerati di "maggiore attendibilità" per non essere stati cofinanziati, a differenza di altri, anche dalle stesse ditte produttrici di cellulari".

CORTE DI CASSAZIONE

SEZIONE LAVORO

SENTENZA n. 17438 DEL 12 ottobre 2012

SVOLGIMENTO DEL PROCESSO

Con sentenza del 10 - 22.12.2009 la Corte d'appello di Brescia, in riforma della pronuncia di prime cure, condannò l'Inail a corrispondere a M. I. la rendita per malattia professionale prevista per l'invalidità all'80%.

Il M. aveva agito in giudizio deducendo che, in conseguenza dell'uso lavorativo protratto, per dodici anni e per 5-6 ore al giorno, di telefoni cordless e cellulari all'orecchio sinistro aveva contratto una grave patologia tumorale; le prove acquisite e le indagini medico legali avevano permesso di accertare, nel corso del giudizio, la sussistenza dei presupposti fattuali dedotti, in ordine sia all'uso nei termini indicati dei telefoni ne! corso dell'attività lavorativa, sia all'effettiva insorgenza di un "neurinoma del Ganglio di Gasser" (tumore che colpisce i nervi cranici, in particolare il nervo acustico e, più raramente, come nel caso di specie, il nervo cranico trigemino), con esiti assolutamente severi nonostante le terapie, anche di natura chirurgica, praticate; sulla ricorrenza di tali elementi fattuali, come evidenziato nella sentenza impugnata, non erano state svolte contestazioni in sede di appello, incentrandosi la questione devoluta al Giudice del gravame sul nesso causale tra l'uso dei telefoni e l'insorgenza della patologia.

La Corte territoriale, rinnovata la consulenza medico legale, ritenne dì dover seguire le conclusioni a cui era pervenuto il CTU nominato in grado d'appello, osservando in particolare quanto segue:

- i telefoni mobili (cordless) e i telefoni cellulari funzionano attraverso onde elettromagnetiche e, secondo il CTU, "In letteratura gli studi sui tumori cerebrali per quanto riguarda il neurinoma considerano il tumore con localizzazione al nervo acustico che è il più frequente. Trattandosi del medesimo istotipo è del tutto logico assimilare i dati al neurinoma del trigemino"; in particolare era stato osservato che i due neurinomi appartengono al medesimo distretto corporeo, in quanto entrambi i nervi interessati si trovano nell'angolo

ponto-cerebellare, che è una porzione ben definita e ristretta dello spazio endocranico, certamente compresa nel campo magnetico che si genera dall'utilizzo dei telefoni cellulari e cordless;

nella CTU erano stati riassunti con una tabella alcuni studi effettuati dal 2005 al 2009 ed in tre, effettuati dall'Hardell group, era stato evidenziato un aumento significativo de! rischio relativo di neurinoma (intendendosi per rischio relativo la misura di associazione fra l'esposizione ad un particolare fattore di rischio e l'insorgenza di una definita malattia, calcolata come il rapporto fra i tassi di incidenza negli esposti [numeratore] e nei non esposti [denominatore]);

- un lavoro del 2009 del medesimo gruppo aveva considerato anche altri elementi quali età dell'esposizione, l'ipsilateralità e il tempo di esposizione, indicando, per quanto riguarda il neurinoma dell'acustico, un Odd ratio per l'uso dei cordless di 1,5 e per il telefono cellulare di 1,7;

considerando l'uso maggiore di 10 anni, gli Odd ratio erano rispettivamente di 1,3 e di 1,9, intendendosi per Odd ratio il rapporto tra la frequenza con la quale un evento si verifica in un gruppo di pazienti e la frequenza con la quale lo stesso evento si verifica in un gruppo di pazienti di controllo, onde se il valore dell'Odd ratio è superiore a 1 significa che la probabilità che si verifichi l'evento considerato (per esempio una malattia) in un gruppo (per esempio tra gli esposti) è superiore rispetto a quella di un altro gruppo (per esempio tra i non esposti), mentre significato opposto ha un valor inferiore a 1;

- una recente review della The International Commission on Non- lonizing Radiation Protection aveva evidenziato i limiti degli studi epidemiologici fino ad allora attuati, concludendo che, allo stato attuale, non vi era una convincente evidenza del ruolo delle radiofrequenze nella genesi dei tumori, ma aggiungendo che gli studi non ne avevano escluso l'associazione;

- un'ulteriore autorevole review (Kundi nel 2009) aveva confermato i dubbi che gli studi epidemiologici inducono per quanto riguarda il tempo di esposizione e concluso per un rischio individuale basso, ma presente; l'esposizione poteva incidere sulla storia naturale della neoplasia in vari modi: interagendo nella fase iniziale di induzione, intervenendo sul tempo di sviluppo dei tumori a lenta crescita, come i neurinomi, accelerandola ed evitando la possibile naturale involuzione;

- l'analisi della letteratura non portava quindi ad un giudizio esaustivo, ma, con tutti i limiti insiti nella tipologia degli studi, un rischio aggiuntivo per i tumori cerebrali, ed in particolare per il neurinoma, era documentato dopo un'esposizione per più di 10 anni a radiofrequenze emesse da telefoni portatili e cellulari;

- tale tempo di esposizione era un elemento valutativo molto rilevante, poiché, nello studio del 2006, l'esposizione per più di 10 anni comportava un rischio relativo calcolato di 2,9 sicuramente significativo;

- si trattava quindi di una situazione "individuale" che gli esperti riconducevano al "modello probabilistico-induttivo" ed alla "causalità debole", avente comunque valenza in sede previdenziale;

- doveva dunque riconoscersi, secondo il CTU, un ruolo almeno concausale delle radiofrequenze nella genesi della neoplasia subita dall'assicurato, configurante probabilità qualificata:

- la censura dell'lnail relativa agli studi utilizzati dal CTU non coglieva nel segno, poiché lo studio del 2000 dell'OMS, che aveva escluso effetti negativi per la salute, si era basato su dati ancor più risalenti, non tenendo quindi conto dell'uso più recente, ben più massiccio e diffuso, di tali apparecchi e del fatto che si tratta di tumori a lenta insorgenza, risultando quindi più attendibili gli studi svolti nel 2009;

- inoltre, come osservato dal CT di parte M., gli studi del 2009 non erano stati condotti su un basso numero di casi, ma, al contrario, sul numero totale dei casi (679) che si erano verificati in un anno in Italia; inoltre, a differenza dello studio della IARC, co-finanziato dalla ditte produttrici di telefonicellulari, gli studi citati dal CTU erano indipendenti;

- ancora, secondo quanto osservato dal CT di parte M., confrontando il dato di rischio individuale calcolato dal CTU (2,9) con quello rilevato per il fattore di rischio, universalmente riconosciuto, dell'esposizione alle radiazioni ionizzanti, doveva considerarsi come per i sopravvissuti alle esplosioni atomiche giapponesi di Hiroshima e Nagasaki fosse stato accertato un rischio relativo di tipo oncologico di 1,39 per "tutti i tumori" con un minimo di 1,22 per i tumori di "utero e cervice" ed un massimo di 4,92 per la "leucemia", il che stava a significare che il rischio oncogeno medio delle radiazioni ionizzanti era inferiore a quello che si aveva per l'esposizione alle radio frequenze in riferimento ai neurinomi endocranici, ciò che rendeva ancora più evidente la reale portata di quanto affermato dal CTU;

- secondo l'insegnamento della giurisprudenza di legittimità, nel caso di malattia professionale non tabellata, come anche in quello di malattia ad eziologia multifattoriale, la prova della causa di lavoro, che grava sul lavoratore, deve essere valutata in termini di ragionevole certezza, nel senso che, esclusa la rilevanza della mera possibilità dell'origine professionale, questa può essere invece ravvisata in presenza di un rilevante grado di probabilità; e, a tale riguardo, il giudice deve non solo consentire all'assicurato di esperire i mezzi di prova ammissibili e ritualmente dedotti, ma deve altresì valutare le conclusioni probabilistiche del consulente tecnico in tema di nesso causale, considerando che la natura professionale della malattia può essere desunta con elevato grado di probabilità dalla tipologia delle lavorazioni svolte, dalla natura dei macchinari presenti nell'ambiente di lavoro, dalla durata della prestazione lavorativa e dall'assenza di altri fattori extralavorativi, alternativi o concorrenti che possano costituire causa della malattia;

- doveva quindi ritenersi la sussistenza del requisito di elevata probabilità che integra il nesso causale richiesto dalla normativa. Avverso la suddetta sentenza della Corte territoriale rinati ha proposto ricorso fondato su due motivi e illustrato con memoria L'intimato M. I. ha resistito con controricorso, illustrato con memoria.

Motivi della decisione1. Con il primo motivo l'Istituto ricorrente denuncia violazione dell'art. 3 dpr n. 1124/65, rilevando che, secondo i principi di diritto elaborati in materia dalla giurisprudenza di legittimità, la corretta applicazione della norma suddetta richiede, in particolare, l'accertamento sulla base di dati

epidemiologici e di letteratura ritenuti affidabili dalla comunità scientifica, che l'agente dedotto in giudizio sia dotato di efficienza patogenetica, quanto meno probabile, per la specifica malattia allegata e diagnosticata; la suddetta relazione causale non poteva dunque essere suffragata "dalla personale valutazione dell'ausiliario del giudice, fondata sulla preferenza per taluni dati epidemiologici rispetto ad altri, ma deve essere supportata da un giudizio di affidabilità dei dati stessi espresso dalla comunità scientifica"; nel caso di specie il CTU si era soffermato esclusivamente sui risultati del gruppo Hardell, in contrasto con quelli della comunità scientifica;

inoltre il CTU aveva del tutto arbitrariamente utilizzato la contabilità tra esposizioni a radiofrequenze e neurinoma del nervo acustico, ipotizzata dal gruppo Hardeil, per affermare la relazione causale, addirittura con giudizio di probabilità qualificata, tra tali radiofrequenze e il neurinoma del trigemino; doveva al riguardo rilevarsi che la Commissione scientifica per l'elaborazione e la revisione periodica delle malattie di cui è obbligatoria la segnalazione ai sensi dell'art. 139 dpr n. 1124/65, in occasione dell'aggiornamento dell'elenco approvato con decreto ministeriale 11.12.2009, non aveva ritenuto di dover includere i tumori dei nervi cranici, indotti da esposizione alle radiofrequenze, tra le malattie di possibile origine professionale. 1.2Secondo la

giurisprudenza di questa Corte, nel caso di malattia professionale non tabellata, come anche in quello di malattia ad eziologia multifattoriale, la prova della causa di lavoro, che grava sul lavoratore, deve essere valutata in termini di ragionevole certezza, nel senso che, esclusa la rilevanza della mera possibilità dell'origine professionale, questa può essere invece ravvisata in presenza di un rilevante grado di probabilità; a tale riguardo, il giudice deve non solo consentire all'assicurato di esperire i mezzi di prova ammissibili e ritualmente dedotti, ma deve altresì valutare le conclusioni probabilistiche del consulente tecnico in tema di nesso causale, facendo ricorso ad ogni iniziativa ex officio diretta ad acquisire ulteriori elementi in relazione all'entità ed

all'esposizione del lavoratore ai fattori di rischio ed anche considerando che la natura professionale della malattia può essere desunta con elevato grado di probabilità dalla tipologia delle lavorazioni svolte, dalia natura dei macchinari presenti nell'ambiente di lavoro, dalla durata della prestazione lavorativa e dall'assenza di altri fattori extralavorativi, alternativi o concorrenti, che possano costituire causa della malattia (cfr, ex plurimis, Cass., nn. 6434/1994; 5352/2002; 11128/2004;15080/2009).

La sentenza impugnata ha fatto applicazione di tali principi, ravvisando, in base alle considerazioni diffusamente esposte nello storico di lite, la sussistenza del requisito di elevata probabilità che integra il nesso causale.

Non è quindi ravvisabile il denunciato vizio di violazione di legge, che si fonda infatti su una

pretesa erronea valutazione (da parte del CTU e della Corte territoriale) della affidabilità dei dati

presi in considerazione al fine di suffragare tale requisito e, pertanto, sostanzialmente su un vizio di

motivazione (in effetti dedotto con il secondo motivo di ricorso).

Il motivo all'esame va pertanto disatteso.

2. Con il secondo motivo l'Istituto ricorrente denuncia appunto vizio di motivazione, assumendo

che:

- il CTU di secondo grado, dopo avere evidenziato che la review della The International

Commission on Non-lonizing Radiation Protection aveva concluso che, allo stato attuale, non vi era

una convincente evidenza del ruolo delle radiofrequenze nella genesi dei tumori, pur non

escludendosene l'associazione, senza consequenzialità logica e senza motivazione aveva tratto la

conclusione della probabilità qualificata di un ruolo almeno concausale delle radiofrequenze nella

genesi della neoplasia per cui è causa;

- doveva ritenersi priva dì qualsivoglia fondamento scientifico la ritenuta assimilabilità, sul piano

eziopatogenetico, del neurinoma del nervo acustico e di quello del trigemino, essendo "nozione

comune" della scienza medica che tumori dello stesso istotipo, ma con localizzazione diversa, anche se nell'ambito dello stesso distretto anatomico, riconoscono cause diverse e che qualsiasi potenziale

agente cancerogeno che venga in contatto con il corpo umano modifica la sua azione a seconda dei

tessuti che attraversa o con cui viene in contatto; e, in effetti, il nervo acustico e il nervo trigemino,

in particolare il ganglio di Gasser, hanno una diversa collocazione nella teca cranica e diverse sono

le strutture anatomiche che li separano dall'esterno e fra loro;

la Corte territoriale non aveva risposto alle osservazioni svolte dall'Istituto, anche con riferimento

alla circostanza che era "in corso" uno studio epidemiologico internazionale "interphone",

coordinato dalla IARC e che l'OMS, in base al principio di precauzione, aveva suggerito "una politica di gestione del rischio che viene applicata in una situazione di "incertezza scientifica"":
- doveva ritenersi inconferente sul piano scientifico l'affermazione della Corte territoriale circa l'attendibilità, perché indipendente, dello studio del gruppo Hardell, a fronte del cofinanziamento della ricerca "interphone" da parte dei produttori di telefoni cellulari, trascurando che tale ricerca è finanziata dalla Unione Europea e diretta e coordinata dalla IARC (Agenzia internazionale ricerca sul cancro dell'OMS);
- neppure la Corte territoriale aveva ritenuto di chiamare il CTU a chiarimenti a fronte delle ricordate osservazioni critiche. 2.1 La giurisprudenza di legittimità ha reiteratamente affermato che nei giudizi in cui sia stata esperita CTU di tipo medico-legale, nei caso in cui il giudice del merito si basi sulle conclusioni dell'ausiliario giudiziario, affinché i lamentati errori e lacune della consulenza tecnica determinino un vizio di motivazione della sentenza denunciabile in cassazione, è necessario che i relativi vizi logico -formali si concretino in una palese devianza dalle nozioni della scienza medica o si sostanzino in affermazioni illogiche o scientificamente errate, con il relativo onere, a carico della parte interessata, di indicare le relative fonti, senza potersi la stessa limitare a mere considerazioni sulle prospettazioni operate dalla controparte, che si traducono in una inammissibile critica del convincimento del giudice di merito che si sia fondato, per l'appunto, sulla consulenza tecnica (cfr, ex plurimis, Cass., nn. 16392/2004; 17324/2005; 7049/2007; 18906/2007).
Nel caso all'esame l'Istituto ricorrente, nel contestare la ritenuta assimilabilità, sul piano eziopatogenetico, del neurinoma del nervo acustico e di quello del trigemino, non specifica - rifugiandosi nel concetto di "nozione comune" - le fonti scientifiche, ritualmente dedotte ed acquisite al giudizio, in base alle quali avrebbero dovuto ritenersi scientificamente errate le affermazioni rese al riguardo dal CTU e seguite dalla sentenza impugnata, finendo per richiedere al riguardo a questa Corte una valutazione di merito inammissibile in sede di legittimità.
Neppure è dato rilevare il preteso e denunciato vizio di mancanza di consequenzialità logica e di motivazione in ordine alle conclusioni della probabilità qualificata di un ruolo almeno concausale delle radiofrequenze nella genesi della neoplasia per cui è causa, posto che tale giudizio, come diffusamente esposto nello storico di lite, non discende dalla mera indicazione delle conclusioni

(evidentemente difformi) a cui era pervenuta la ricordata review della The International Commission on Non-lonizing Radiation Protection, ma, piuttosto, dai riscontri di altri studi a carattere epidemiologico svolti al riguardo.

Inoltre, e significativamente, la sentenza impugnata, seguendo le osservazioni del CTU, ha ritenuto di dover ritenere di particolare rilievo quegli studi che avevano preso in considerazione anche altri elementi, quali l'età dell'esposizione, l'ipsilateralità e il tempo di esposizione, atteso che, nella specie, doveva valutarsi la sussistenza del nesso causale in relazione ad una situazione fattuale dei tutto particolare, caratterizzata da un'esposizione alle radiofrequenze per un lasso temporale continuativo molto lungo (circa 12 anni), per una media giornaliera di 5 - 6 ore e concentrata principalmente sull'orecchio sinistro dell'assicurato (che, com'è di piana evidenza, concretizza una situazione affatto diversa da un normale uso non professionale del telefono cellulare).

L'ulteriore rilievo circa la maggiore attendibilità proprio di tali studi, stante la loro posizione di indipendenza, ossia per non essere stati cofinanziati, a differenza di altri, anche dalle stesse ditte produttrici di cellulari, costituisce ulteriore e non illogico fondamento delle conclusioni accolte.Né è stato dedotto - e tanto meno, dimostrato - che le indagini epidemiologiche Se cui conclusioni sono state prese in particolare considerazione provengano da gruppi di lavoro privi di serietà ed autorevolezza e, come tali, sostanzialmente estranei alla comunità scientifica.

L'asserita prevalenza che, secondo il ricorrente, dovrebbe essere attribuita alle conclusioni di altri gruppi di ricerca (le cui indagini, peraltro, secondo quanto dedotto, almeno all'epoca del giudizio di merito erano ancora "in corso"), si risolvono anch'essi nella richiesta di un riesame del merito, non consentito in sede di legittimità. Avendo inoltre la Corte territoriale riscontrato nelle considerazioni già svolte dal CTU e dal CT di parte M. elementi ritenuti sufficienti a confutare le osservazioni critiche dell'Istituto, non sussisteva la necessità di investire ulteriormente il CTU di una richiesta a chiarimenti.

Anche il secondo motivo di ricorso va quindi disatteso.

3. In definitiva il ricorso va rigettato

L'esito fra loro difforme dei giudizi di merito e la novità, sotto il profilo della peculiarità fattuale, della vicenda dedotta in causa, consigliano la compensazione delle spese.

P.Q.M.

Rigetta il ricorso; spese compensate.

Planetary electromagnetic pollution: it is time to assess its impact

As the Planetary Health Alliance moves forward after a productive second annual meeting, a discussion on the rapid global proliferation of artificial electromagnetic fields would now be apt. The most notable is the blanket of radiofrequency electromagnetic radiation, largely microwave radiation generated for wireless communication and surveillance technologies, as mounting scientific evidence suggests that prolonged exposure to radiofrequency electromagnetic radiation has serious biological and health effects. However, public exposure regulations in most countries continue to be based on the guidelines of the International Commission on Non-Ionizing Radiation Protection[1] and Institute of Electrical and Electronics Engineers,[2] which were established in the 1990s on the belief that only acute thermal effects are hazardous. Prevention of tissue heating by radiofrequency electromagnetic radiation is now proven to be ineffective in preventing biochemical and physiological interference. For example, acute non-thermal exposure has been shown to alter human brain metabolism by NIH scientists,[3] electrical activity in the brain,[4] and systemic immune responses.[5] Chronic exposure has been associated with increased oxidative stress and DNA damage[6,7] and cancer risk.[8] Laboratory studies, including large rodent studies by the US National Toxicology Program[9] and Ramazzini Institute of Italy,[10] confirm these biological and health effects in vivo. As we address the threats to human health from the changing environmental conditions due to human activity,[11] the increasing exposure to artificial electromagnetic radiation needs to be included in this discussion.

Due to the exponential increase in the use of wireless personal communication devices (eg, mobile or cordless phones and WiFi or Bluetooth-enabled devices) and the infrastructure facilitating them, levels of exposure to radiofrequency electromagnetic radiation around the 1 GHz frequency band, which is mostly used for modern wireless communications, have increased from extremely low natural levels by about 10^{18} times (figure). Radiofrequency electromagnetic radiation is also used for radar, security scanners, smart meters, and medical equipment (MRI, diathermy, and radiofrequency ablation). It is plausibly the most rapidly increasing anthropogenic environmental exposure since the mid-20th century, and levels will surge considerably again, as technologies like the Internet of Things and 5G add millions more radiofrequency transmitters around us.

Unprecedented human exposure to radiofrequency electromagnetic radiation from conception until death has been occurring in the past two decades. Evidence of its effects on the CNS, including altered neurodevelopment[14] and increased risk of some neurodegenerative diseases,[15] is a major concern considering the steady increase in their incidence. Evidence exists for an association between neurodevelopmental or

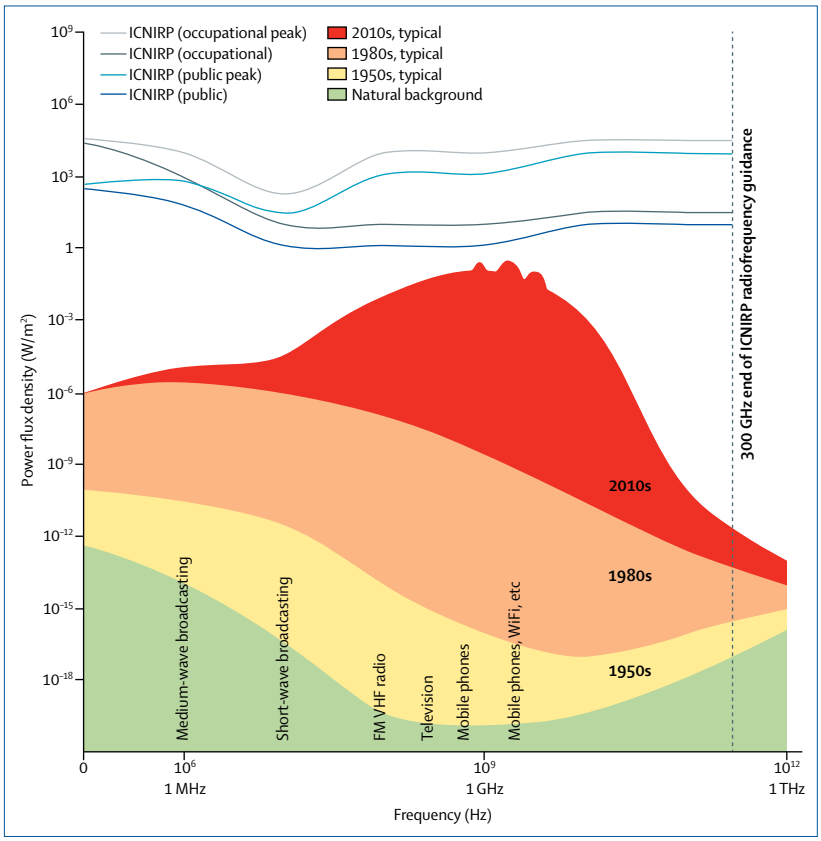

***Figure*: Typical maximum daily exposure to radiofrequency electromagnetic radiation from man-made and natural power flux densities in comparison with International Commission on Non-Ionizing Radiation Protection safety guidelines[1]**

Anthropogenic radiofrequency electromagnetic radiation levels are illustrated for different periods in the evolution of wireless communication technologies. These exposure levels are frequently experienced daily by people using various wireless devices. The levels are instantaneous and not time-averaged over 6 minutes as specified by International Commission on Non-Ionizing Radiation Protection for thermal reasons. Figure modified from Philips and Lamburn[12] with permission. Natural levels of radiofrequency electromagnetic radiation were based on the NASA review report CR-166661.[13]

behavioural disorders in children and exposure to wireless devices,[14] and experimental evidence, such as the Yale finding, shows that prenatal exposure could cause structural and functional changes in the brain associated with ADHD-like behaviour.[16] These findings deserve urgent attention.

For the **Oceania Radiofrequency Scientific Advisory Association** see www.orsaa.org

At the Oceania Radiofrequency Scientific Advisory Association, an independent scientific organisation, volunteering scientists have constructed the world's largest categorised online database of peer-reviewed studies on radiofrequency electromagnetic radiation and other man-made electromagnetic fields of lower frequencies. A recent evaluation of 2266 studies (including in-vitro and in-vivo studies in human, animal, and plant experimental systems and population studies) found that most studies (n=1546, 68·2%) have demonstrated significant biological or health effects associated with exposure to anthropogenic electromagnetic fields. We have published our preliminary data on radiofrequency electromagnetic radiation, which shows that 89% (216 of 242) of experimental studies that investigated oxidative stress endpoints showed significant effects.[7] This weight of scientific evidence refutes the prominent claim that the deployment of wireless technologies poses no health risks at the currently permitted non-thermal radiofrequency exposure levels. Instead, the evidence supports the International EMF Scientist Appeal by 244 scientists from 41 countries who have published on the subject in peer-reviewed literature and collectively petitioned the WHO and the UN for immediate measures to reduce public exposure to artificial electromagnetic fields and radiation.

For the **International EMF Scientist Appeal** see www.emfscientist.org

Evidence also exists of the effects of radiofrequency electromagnetic radiation on flora and fauna. For example, the reported global reduction in bees and other insects is plausibly linked to the increased radiofrequency electromagnetic radiation in the environment.[17] Honeybees are among the species that use magnetoreception, which is sensitive to anthropogenic electromagnetic fields, for navigation.

Man-made electromagnetic fields range from extremely low frequency (associated with electricity supplies and electrical appliances) to low, medium, high, and extremely high frequency (mostly associated with wireless communication). The potential effects of these anthropogenic electromagnetic fields on natural electromagnetic fields, such as the Schumann Resonance that controls the weather and climate, have not been properly studied. Similarly, we do not adequately understand the effects of anthropogenic radiofrequency electromagnetic radiation on other natural and man-made atmospheric components or the ionosphere. It has been widely claimed that radiofrequency electromagnetic radiation, being non-ionising radiation, does not possess enough photon energy to cause DNA damage. This has now been proven wrong experimentally.[18,19] Radiofrequency electromagnetic radiation causes DNA damage apparently through oxidative stress,[7] similar to near-UV radiation, which was also long thought to be harmless.

At a time when environmental health scientists tackle serious global issues such as climate change and chemical toxicants in public health, there is an urgent need to address so-called electrosmog. A genuine evidence-based approach to the risk assessment and regulation of anthropogenic electromagnetic fields will help the health of us all, as well as that of our planetary home. Some government health authorities have recently taken steps to reduce public exposure to radiofrequency electromagnetic radiation by regulating use of wireless devices by children and recommending preferential use of wired communication devices in general, but this ought to be a coordinated international effort.

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THE LARGEST UNETHICAL MEDICAL EXPERIMENT IN HUMAN HISTORY

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KEYWORDS

Unethical Research; Electromagnetic Fields; Wireless Radiation; Radiofrequency Radiation; RF; Non-Ionizing Radiation; Mobile Networking Technology; 5G; Adverse Health Effects

ABSTRACT

This monograph describes the largest unethical medical experiment in human history: the implementation and operation of non-ionizing non-visible EMF radiation (hereafter called wireless radiation) infrastructure for communications, surveillance, weaponry, and other applications. It is unethical because it violates the key ethical medical experiment requirement for "informed consent" by the overwhelming majority of the participants.

The monograph provides background on unethical medical research/experimentation, and frames the implementation of wireless radiation within that context. The monograph then identifies a wide spectrum of adverse effects of wireless radiation as reported in the premier biomedical literature for over seven decades. Even though many of these reported adverse effects are extremely severe, the ***true extent of their severity has been grossly underestimated***.

Most of the reported laboratory experiments that produced these effects are not reflective of the real-life environment in which wireless radiation operates. Many experiments do not include pulsing and modulation of the carrier signal, and most do not account for synergistic effects of other toxic stimuli acting in concert with the wireless radiation. These two additions ***greatly exacerbate the severity of the adverse effects from wireless radiation***, and their neglect in current (and past) experimentation results in substantial under-estimation of the breadth and severity of adverse effects to be expected in a real-life situation. This lack of credible safety testing, combined with depriving the public of the opportunity to provide informed consent, contextualizes the wireless radiation infrastructure operation as an unethical medical experiment.

Addition of the nascent fifth generation of mobile networking technology (5G) globally to the existing mobile technology network will contribute further to the largest unethical medical experiment in human history!

This monograph consists of four chapters and eight appendices. Chapter 1 focuses on unethical research, showing how wireless radiation infrastructure implementation fits into the

framework of unethical medical experimentation, and providing many examples of other types of unethical medical experimentation.

Chapter 2 is the main technical chapter, focusing on adverse health effects of wireless radiation. It describes:

- adverse effects from past research, and what additional adverse effects can be expected when 5G is implemented fully
- lack of full consensus among key stakeholders on adverse effects from wireless radiation, and the role played by conflicts-of-interest in this lack of consensus
- the main reason that this unethical medical experiment was allowed to take place:

The Federal government that ***promotes*** accelerated implementation of wireless radiation technology also 1) ***sponsors*** research examining the technology's potential adverse effects and 2) ***regulates*** the technology's potentially adverse impacts on the public. This unethical promotion-sponsorship-regulation conflict-of-interest lays the groundwork for unethical medical experimentation!

Chapter 3 contains the references for the main text, and Chapter 4 contains the eight appendices.

Appendix 1 presents more details about unethical medical experiments, including examples and many references for further study.

Appendix 2 contains a manual taxonomy of a representative adverse EMF effects database; Appendix 3 contains a factor analysis taxonomy of the same database; and, Appendix 4 contains a text clustering taxonomy of the same database. All three taxonomies contain links between the categories in the summary tables and the titles of papers associated with each category.

Appendix 5 shows the ***potential contribution of wireless radiation to the opioid crisis*** and ***potential contribution of wireless radiation to exacerbation of the coronavirus pandemic***.

Appendix 6 shows the ***link between funding source and research outcomes***, and presents many references on the topic of funding source-driven bias.

Appendix 7 describes the under-recognized adverse effects of wireless radiation related to ***medical implants*** (pacemakers, defibrillators, cochlear implants, dental implants, bone pins, etc) and metal appendages (metal jewelry, etc), and potential ***micro/nano***-implant analogues.

Appendix 8 shows ***adverse effects of wireless radiation on automotive vehicle occupants*** (and bystanders), and the under-advertised on-board and external sources of this radiation.

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PREFACE

Humanity is racing along two parallel paths to self-destruction: 1) accelerating irreversible climate change, and 2) rapidly increasing exposure to health and life-threatening mixtures of toxic stimuli. The most ubiquitous constituent of these toxic mixtures is wireless radiation, which is proceeding to blanket humanity and its ecological life support chain.

A small fraction of the population has given informed consent to wireless radiation exposure, gambling (like users of cigarettes, cocaine, fentanyl) that they can escape the severe adverse consequences of exposure. Another small fraction of the population has not given informed consent, but receives harmful second-hand exposure because of the broad-scale transmission of wireless radiation from terrestrial and satellite sources. The vast majority of the population has given ***Mis-***informed Consent to this exposure. This ***mis-***information is supplied by the telecommunications industry, its lobbyists, its government partners, its political enablers, its marketing arm (the mainstream media), and even some academic enablers.

While research over the past seventy+ years has shown hard evidence of severe adverse effects from wireless radiation, the full extent of the damage from existing wireless radiation infrastructure is not known, much less the damage expected from 4G/5G infrastructure being implemented rapidly today. Attempting to identify the full extent of these adverse effects is the global medical experiment being conducted today. The fact that this experiment is being conducted with ***mis***-informed consent makes it an unethical medical experiment. Because of the magnitude of this experiment, it is the *largest unethical medical experiment in human history!*

Chapter 1 of this monograph presents the case for wireless radiation infrastructure implementation without credible safety testing being not only an unethical medical experiment, but the largest in human history. It presents wireless radiation infrastructure implementation in the context of other recent examples of unethical medical experiments, and shows how these others pale in comparison to the projected suffering and lethality from wireless radiation exposure based on even the incomplete biomedical data gathered to date.

Chapter 2 is the main technical chapter in this monograph. It covers a broad scope of adverse health and life-supporting ecological effects from wireless radiation, mainly at communications frequencies. Some of these adverse effects are not well-known to the general public, but they are important nevertheless. While the majority of the chapter is technical, its initial section provides the context for evaluating the biomedical literature results. In particular, it emphasizes the conflicts-of-interest operable in all aspects of the wireless radiation biomedical research process, ranging from the initial health-effects research sponsorship to the final research results dissemination in the premier technical literature and other forums. As Chapter 2 shows, we have known about the adverse health and ecological effects of wireless radiation exposure for seventy+ years, but decision-makers of all stripes have nevertheless chosen to impose this health and life-threatening toxic stimulus on an unsuspecting global populace.

Additionally, there are eight appendices. The copious material contained in the appendices supports the statements made in the main text (Chapters 1 and 2). Three sub-appendices, while grounded in hard evidence, are somewhat more hypothetical than the rest. They include 1) linkages between wireless radiation exposure and exacerbation of the opioid crisis and the coronavirus pandemic, and 2) potentially enhanced heating and temperature increases to thermally-damaging levels from short RF pulses and tissue-imbedded nanoparticles. My purpose in presenting these three more hypothetical sub-appendices is to stimulate more discussion, and especially more research, on the nature and validity of these linkages.

Finally, it is my hope that this monograph receives the widest distribution, especially among those who have 1) been the targets of this decades-long mis-information campaign and 2) given their consent to wireless radiation exposure based upon mis-information. It is this segment of the public whose informed actions could reverse the increasing implementation of wireless radiation infrastructure, and prevent the infliction of even more damage, since the other stakeholders involved in the promotion of wireless radiation infrastructure have shown little desire to protect the public against the known and projected ravages of wireless radiation.

Ronald N. Kostoff, Gainesville, VA, 15 February 2020

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EXECUTIVE SUMMARY

ES-1. Overview

We are in the midst of the largest unethical medical experiment in human history. This experiment is the implementation and operation of a global wireless network for communications, surveillance, and other purposes. It is a ***medical experiment*** because we do not know the full extent of the adverse health effects that will result from this wireless network implementation and operation. It is an ***unethical*** medical experiment because it violates the key ethical medical experiment requirement of ***'informed consent'*** from the participants.

Even though the adverse health effects of wireless radiation reported over the past seventy+ years span the range of severity from discomfort to lethality, we do not know the full extent of adverse health effects from this technology because:

Most laboratory experiments aimed at identifying wireless radiation health effects bear no relation to real-life exposures, and are performed under the most benign conditions of

- single stressors (wireless radiation only)
- no pulsing and modulation of the carrier signal
- no synergistic effects of other toxic stimuli acting in concert with the wireless radiation

These experimental deficiencies are compounded by

- lack of access to the global classified literature on adverse health effects from wireless radiation
- lack of knowledge of proprietary basic and advanced studies on adverse health effects from wireless radiation.

The adverse wireless radiation health effects that have been identified already from the incomplete literature openly available are massive in scope and magnitude. They support the conclusion that ***wireless radiation as already implemented is extremely dangerous to human health***. It acts as both a ***promoter/accelerator*** and ***initiator*** of adverse health effects. Addition of the missing elements described above and more wireless radiation infrastructure will exacerbate further the adverse effects from wireless radiation on

- human health directly through contribution to chronic disease and
- human health indirectly through degradation of the food chain ecosystem.

ES-2. Adverse Impacts of Wireless Radiation on the Most Vulnerable Members of Society

In the spirit of the 'unethical' medical experiments described in this monograph,

it is the poor and dispossessed who will suffer the most from wireless radiation exposure.

This is because wireless radiation plays a dual role of ***initiator*** and ***promoter/accelerator*** of serious disease. In its ***promoter/accelerator*** role, it can accelerate the progression of existing serious diseases such as cancer, and/or, through synergy, can produce serious adverse health effects when combined with other toxic stimuli that neither constituent of the combination could produce in isolation.

Many toxic stimuli, such as harsh chemicals, biotoxins, ionizing radiation sources, vibrating machinery, prolonged sitting doing repetitive tasks, high air pollution, etc, are used/experienced by the poorest members of society in their occupations, and many toxic stimuli, such as air pollutants, toxic wastes, etc, are very prevalent in their residential environments. Thus, people who spray pesticides in farm labor or household applications, people who do cleaning with harsh chemicals, people who dispose of hazardous materials, basically, ***people who do the dirty work in our society and live in dirty environments***, are already leading candidates for higher risk of serious diseases. Adding a wireless radiation ***promoter/accelerator*** to their residential and occupational environments will radically increase their chances for developing serious diseases. Closing the 'digital divide' for them will translate to increased suffering and reduced longevity!

ES-3. Role of Conflicts-of-Interest in the Sponsorship, Conduct, and Dissemination of Wireless Radiation Research

The results shown in the literature cannot be separated from the context in which this research has been sponsored, conducted, and disseminated!

In the USA (and in most, if not all, countries), the two major sponsors of wireless radiation health and safety research are the Federal government and the wireless radiation industry, in that order. Both of these organizations have a strong intrinsic conflict-of-interest with respect to wireless radiation.

The Federal government is a strong promoter of wireless radiation infrastructure development and rapid expansion, most recently supporting accelerated implementation of 5G infrastructure.

The Federal government that ***promotes*** accelerated implementation of wireless radiation technology also 1) ***sponsors*** research examining the technology's potential adverse effects and 2) ***regulates*** the technology's potentially adverse impacts on the public. The fact that these development, regulation, and safety functions may be assigned to different Executive Agencies within the Federal government is irrelevant from an independence perspective. ***The separate Executive Agencies in the Federal government are like the tentacles of an Octopus; they operate synchronously under one central command.***

The wireless promoters' main objectives of developing and implementing the technology rapidly are enabled by suppressing knowledge (to the public) of potential adverse effects from the technology's operation. These fundamental conflicts impact the objectivity of the health and safety R&D sponsors and performers. Any ***Federal research sponsor*** of wireless radiation technology safety would be highly conflicted between 1) a desire to satisfy Executive and Legislative objectives of accelerating expansion of wireless radiation technology and implementation and 2) sponsoring objective research focused on identifying and reporting adverse effects of wireless radiation expected under real-life conditions. Likewise, any ***sponsored research performer*** addressing wireless radiation technology safety would be highly conflicted between 1) reporting the actual adverse effects expected under real-life conditions and 2) the desire to satisfy wireless radiation promotional objectives of the research sponsors in order to maintain long-range funding.

ES-4. Adverse Health Effects from Wireless Radiation Exposure.

In aggregate, for the high frequency (radiofrequency-RF) part of the spectrum, expert reviews show that RF radiation below the FCC (Federal Communications Commission) exposure guidelines can result in:

-carcinogenicity (brain tumors/glioma, breast cancer, acoustic neuromas, leukemia, parotid gland tumors),

-genotoxicity (DNA damage, DNA repair inhibition, chromatin structure),

-mutagenicity, teratogenicity,

-neurodegenerative diseases (Alzheimer's Disease, Amyotrophic Lateral Sclerosis),

-neurobehavioral problems, autism,

-reproductive problems, pregnancy outcomes,

-oxidative stress, inflammation, apoptosis, blood-brain barrier disruption,

-pineal gland/melatonin production, sleep disturbance, headache,

-irritability, fatigue, concentration difficulties, depression, dizziness, tinnitus,

-burning and flushed skin, digestive disturbance, tremor, cardiac irregularities, and can

-adversely impact the neural, circulatory, immune, endocrine, and skeletal systems.

The effects range from myriad feelings of discomfort to life-threatening diseases. From this perspective, RF exposure is a highly pervasive cause of disease!

ES-5. Adverse Impacts of Wireless Radiation on the Food Chain

The struggle for survival of human life on Earth is dependent on the logistical food supply chain. At the foundation of this supply chain (before the farmers become involved in harvesting its bounty) are the insects, seeds, flora, trees, etc, that enable the bountiful growth of the myriad potential foods. If the integrity of this foundational logistical supply chain is threatened in any way, then both the animals and plant products we consume become unavailable.

There is a substantial literature on the adverse impacts of wireless radiation on this foundational logistical supply chain. These adverse effects are from the pre-5G wireless radiation exposures, and would include enhanced coupling from the higher frequency harmonics of the RF signal. Many of these supply chain elements (e.g., insects, seeds, larvae, etc) are very small, and we could expect enhanced resonance/energy coupling with the shorter-wavelength 5G radiation when implemented. This indirect impact of wireless radiation may turn out to be at least as (if not more) important as the direct impact of wireless radiation on human survival!

From a broader perspective, most of the laboratory experiment component of the wireless radiation adverse effects literature can be viewed as related to the foundational food supply chain. Much of this research is focused on mice, rats, insects, small birds, small fish, etc. These species tend to be prey of larger animals/fowl/fish, and eventually make their way to the human food table. Any environmental factor that affects the health of these species adversely will eventually impact the humans who are at the end of that chain. In reality, we have accumulated a massive literature describing the adverse impacts of wireless radiation on myriad contributing components to our food supply, and the results do not bode well for our future ability to feed the growing world's population!

ES-6. Adverse Impacts of Wireless Radiation on Medical and Non-Medical Implants

There were two major types of medical implants covered by the database articles showing adverse effects: active implants that produced electrical signals mainly for controlling heart irregularities (e.g., pacemakers, defibrillators) and hearing deficiencies (e.g., cochlear implants), and passive metallic implants for structural support (e.g., dental implants, bone pins, plates, etc). Additionally, there are articles addressing adverse effects from wireless radiation in the vicinity of metallic appendages (e.g., metallic eyeglasses, metallic jewelry, etc).

The external EMF (electromagnetic fields) from microwaves (and other sources) could 1) impact the electrical operation of the active medical implants adversely, 2) increase the Specific Absorption Rate (SAR) values of tissue in the vicinity of the passive implants substantially because of resonance effects, and 3) increase the flow and acidity of saliva in the vicinity of dental structures. While the EMF effects on the cochlear implants could adversely affect auditory capability, EMF effects on the heart-related implants could potentially be life-threatening. The increased SAR values around the passive metal implants could result in increased tissue temperatures, and could adversely impact integration and longevity of the passive metallic implants.

In the mouth, the combination of 1) increased tissue temperatures in proximity to the implant or other orthodontic structures and 2) increased flow rate and acidity of saliva could lead to 3) increased leaching of heavy metals (a known contributor to serious diseases). This also raises the question: what other adverse health effects from the exposure of both the active and passive implants to increasing levels of wireless radiation have not been identified or addressed?

There is a third class of structures whose interaction physics with RF are related to those of the passive implants. These are termed implant analogues, and include myriad exogenous particles (mainly nanoparticles) that penetrate, and imbed in, the skin. The resultant nanoparticle-imbedded tissues have the potential for increased energy absorption from the incoming RF signal, thereby resulting in potentially increased thermal damage over and above the thermal damage resulting from the pulsed high-peak-to-average power of the RF signal. Additionally, more research needs to be done to ascertain the magnitudes of these thermal transients and associated stresses, in order to estimate the levels of enhanced potential damage from RF radiation.

ES-7. Studies in the USSR on Wireless Radiation Health Effects

Much research examining potential adverse effects from wireless radiation, especially in the athermal parameter range, was performed in the USSR as far back as seventy+ years ago. Their results confirm the wide scope of adverse effects reported in recent years and summarized in the present monograph. Unfortunately, their results appear to have had little effect in influencing wireless radiation safety standards in the USA and many other countries.

ES-8. Adverse Effects Expected from Addition of 5G to Existing Communications Networks

The potential 5G adverse health effects derive from the intrinsic nature of the radiation, and how this radiation interacts with tissue and other target structures. 4G networking technology was associated mainly with carrier frequencies in the range of ~1-2.5 GHz (cell phones, WiFi). The wavelength of 1 GHz radiation is 30 cm, and the penetration depth in human tissue is a few centimeters. The highest performance 5G networking technology (millimeter wave) is mainly associated with carrier frequencies at least an order of magnitude above the 4G frequencies, although, as stated in Chapter 2, "ELFs (0–3000Hz) are always present in all telecommunication EMFs in the form of pulsing and modulation". Penetration depths for the high-performance carrier frequency component of 5G radiation (aka high-band) will be on the order of a few millimeters.

For much of the early implementation of 5G, and perhaps later, 5G will be integrated with 4G. Some vendors will start out/have started out with 'low-band' 5G (~600-900 MHz); some will start out with 'mid-band' 5G (~2.5 GHz-4.2 GHz); and some will start out with 'high band' 5G (~24-47 GHz). All these modes are associated with potentially severe adverse health effects, and none have been tested for safety in any credible manner.

At the millimeter carrier wavelengths characteristic of high-band high-performance 5G, one can expect resonance phenomena with small-scale human structures, as well as resonances with insects/insect components, seeds, etc.

The common 'wisdom' being presented in the literature and the broader media is that, if there are adverse impacts resulting from millimeter-wave 5G, the main impacts will be focused on near-surface phenomena, such as skin cancer, cataracts, and other skin conditions, because of shallow RF penetration depths. However, there is evidence that biological responses to millimeter-wave irradiation can be initiated within the skin, and the subsequent systemic signaling in the skin can result in physiological effects on the nervous system, heart, and immune system. There is additional evidence that adverse effects from millimeter-wave radiation can occur in organs and tissue well below the skin surface. This should not be surprising, since there are myriad signaling conduits connecting the skin to deeper structures in the body.

ES-9. Lack of Full Consensus on Wireless Radiation Adverse Effects

Not all studies of wireless radiation have shown adverse effects on health. There are many possibilities to explain this.

1) There could be 'windows' in parameter space where adverse effects occur, and the studies/experiments were conducted outside these 'windows'. Operation outside these windows could show
    - no effects or
    - hormetic effects or
    - therapeutic effects.

The single stressor studies that constitute most of wireless radiation laboratory health research, and indeed constitute most of the laboratory medical research literature, essentially yield very narrow windows. Adverse effects are identified over very limited parameter ranges, and adverse effects shown by many combinations of stressors are not revealed when these stressors are tested in isolation over the same parametric ranges.

One could conclude that, whether by design or accident, ***the real-world impact of single stressor studies is to conceal, rather than reveal, many of the more serious adverse health effects of wireless radiation***.

The stressor variables to be used for health studies should not be limited to single stressors in isolation, but should include to the extent possible combinations of toxic stimuli stressors, since these combinations reflect more accurately real-life exposures.

2) Research quality could be poor, and adverse effects were overlooked.

3) Or, the research team could have had a preconceived agenda

where finding no adverse effects from wireless radiation was the main objective of the research!

ES-10. Potential Links of Wireless Radiation to Enhancement of Opioid Crisis

The previous findings reported in this Executive Summary are based on hard evidence and have been validated in numerous studies. The present section is based on hard evidence as well, but the link of wireless radiation to the opioid crisis is not as far along in the validation process. It should be viewed as a hypothesis at this point, and serve as a basis for discussion and further research.

It has been shown many times that one impact of wireless radiation (at myriad frequencies) is release of endogenous opioids. This release of endogenous opioids can enable analgesic effects by itself, or can enhance the analgesic effects of exogenous analgesics. This has been demonstrated at pulsed millimeter-wave frequencies, WiFi frequencies, mobile phone frequencies, radiofrequencies, and extremely low frequencies. Additionally, as has been demonstrated by the results of the current monograph, wireless radiation at all the above frequencies has resulted in serious mid-term and especially long-term adverse health effects.

Therefore, wireless radiation exposure, especially at cell phone, WiFi, and millimeter-wave pulsed and modulated frequencies, generates ***1) analgesic and pleasurable short-term effects and 2) serious adverse mid- and long-term effects***. There would be some exceptions for the short-term, such as electrohypersensitivity (EHS) sufferers, who are immediately affected adversely and strongly by wireless radiation exposure.

For most people, the enhanced analgesic short-term effects of the wireless radiation would in effect mask the long-term damage from this radiation.

As time proceeds, the increasing discomfort from the adverse mid-and long-term effects of wireless radiation requires increasingly stronger analgesics to suppress, and the increasing use of exogenous analgesics becomes necessary. This potentially enhanced use of exogenous analgesics could lead to opioid and/or other analgesic addictions.

ES-11. Potential Links of Wireless Radiation to Current Coronavirus Pandemic

The previous findings reported in this Executive Summary are based on hard evidence and have been validated in numerous studies. The present section is based on hard evidence as well, but the link of wireless radiation to the coronavirus pandemic is not as far along in the validation process. It should be viewed as a hypothesis at this point, and serve as a basis for discussion and further research.

There are on the order of 300,000 viruses, many/most of which have zoonotic potential. To develop vaccines for all of these viruses (before an epidemic or pandemic strikes) is unreasonable (based on present technology) because of the sheer numbers involved. To develop vaccines for any specific virus during an epidemic or pandemic (which was the mainstream approach taken for the coronavirus during the SARS pandemic of 2002-2003) is completely unrealistic, because of the lead times required for vaccine development, efficacy testing, credible mid-and long-term safety testing, and implementation.

Those who succumbed during the SARS pandemic had 1) myriad co-morbidities and 2) weakened immune systems unable to neutralize the SARS coronavirus. ***Having a strong immune system that allowed a smooth transition from innate immune system operation to adaptive immune system operation was the one intrinsic defense that worked!*** The SARS experience showed that the best and most realistic approach for defense against any potential viral attack is ***reversing immune-degrading lifestyles*** well before any pandemic or epidemic outbreaks. In that case, the immune system would be sufficiently strong to be able to handle viral exposure on its own without the emergence of serious symptoms, as was the case with those exposed to the SARS coronavirus (with coronavirus antibodies in their serum) who exhibited no (or minimal) symptoms.

This gets to the link between wireless radiation exposure and the latest coronavirus pandemic. To the degree that non-ionizing radiation exposure, superimposed on the myriad toxic stimuli to which many people are exposed by choice or imposition, degrades the operation of the innate and adaptive immune systems, it would increase the likelihood that the immune system could not counteract the exposure to the coronavirus (or any virus) as nature intended. Thus, ***it would contribute to the exacerbation of adverse effects from coronavirus exposure***. The bottom line is that exposures to essentially ALL the exogenous immune-damaging toxic stimuli (including, but not limited to, wireless radiation) need to be removed before resistance to viral exposures of any type can be improved substantially.

ES-12. Adverse Effects of Wireless Radiation in Automotive Sector

The modern automobile is a powerful source of wireless radiation at myriad frequencies, and is subject to external wireless radiation at myriad frequencies as well. The trend has not been to reduce these sources, but rather to add equipment both to the vehicle and to the external environment that will substantially increase the wireless radiation flux associated with the vehicle. The numbers and types of sources are not well-known, even among those experts and laymen concerned about adverse effects from wireless radiation.

An interesting diagram (and narrative) showing radars and other wireless sensors in modern cars can be found at the following link: (http://www.radiationdangers.com/automotive-radiation/automotive-radiation/). I would recommend the reader study that diagram in detail, to better appreciate how ubiquitous are these sources of wireless radiation. Not all the wireless radiation enters the cabin, since some/much is outward-directed, but some/much of it will enter the cabins of other cars on the road.

However, that diagram tells only part of the story. Assume there is a car pool commuting to work from the suburbs of a major city. It is not uncommon (in today's world) for a one-way trip to take from one-two hours, or more. Even in a regular car, or mid-size SUV, there might be four or so passengers. They may be using cell phones, WiFi, or both, thereby adding to the radiation from the automotive-based sensors/transmitters.

There will be cell towers lining the sides of a major highway, thereby increasing the radiation to the occupants substantially. Depending on conditions, there may be substantial air pollution to which the occupants are exposed. Additionally, the prolonged sitting is very dangerous, and is a contributing factor to many serious diseases. If the vehicle is new, there may be substantial out-gassing of toxic chemicals from the interior materials. Combined exposure to the wireless radiation, air pollution and other toxic substances, coupled with prolonged sitting and continual impacts from the car's motions, produces a synergistic effect that substantially exacerbates adverse impacts from any of the constituent components.

Chapter 1 – Unethical Research

1A. Monograph Overview

We are in the midst of the largest unethical medical experiment in human history. This experiment is the implementation and operation of a global wireless network for communications, surveillance, and other purposes. It is a ***medical experiment*** because we do not know the full extent of the adverse health effects that will result from this wireless network implementation and operation. It is an ***unethical*** medical experiment because it violates the key ethical medical experiment requirement of ***'informed consent'*** from the participants.

The current chapter provides 1) some background on the requirements for ethical medical research/experimentation and 2) examples of how those requirements have been violated in the past century. It places wireless radiation implementation and operation in the context of these other examples of unethical medical experiments.

Chapter 2 presents a detailed description of some of the adverse health effects of wireless radiation as reported in the unclassified open literature. Even though the adverse health effects of wireless radiation reported over the past seventy+ years span the range of severity from discomfort to lethality, we do not know the full extent of adverse health effects from this technology because:

Most laboratory experiments aimed at identifying wireless radiation health effects bear no relation to real-life exposures, and are performed under the most benign conditions of

- single stressors (wireless radiation only)
- no pulsing and modulation of the carrier signal
- no synergistic effects of other toxic stimuli acting in concert with the wireless radiation

These experimental deficiencies are compounded by

- lack of access to the global classified literature on adverse health effects from wireless radiation
- lack of knowledge of proprietary basic and advanced studies on adverse health effects from wireless radiation.

As Chapter 2 shows, the adverse wireless radiation health effects that have been identified already from the incomplete literature openly available are massive in scope and magnitude. They support the conclusion that ***wireless radiation as already implemented is extremely dangerous to human health***. It acts as both a ***promoter/accelerator*** and ***initiator*** of adverse health effects. Addition of the missing elements described above and more wireless radiation infrastructure will exacerbate further the adverse effects from wireless radiation on

- human health directly through contribution to chronic disease and
- human health indirectly through degradation of the food chain ecosystem.

Chapter 3 contains the references for the main text.

Chapter 4 contains eight Appendices:

- Appendix 1 contains examples of unethical medical experiments conducted in the last century, mainly (not entirely) in the USA or under USA auspices;
- Appendix 2 contains a manual taxonomy of the adverse health and biomedical effects component of a representative wireless radiation literature, and is derived in part from the taxonomies in Appendices 3 and 4;
- Appendix 3 contains a taxonomy based on factor analysis of the same representative wireless radiation literature;
- Appendix 4 contains a taxonomy based on text clustering of the same representative wireless radiation literature;
- Appendix 5 shows ***potential links between wireless radiation exposure and 1) expansion of the opioid crisis*** and ***2) exacerbation of coronavirus pandemic***;
- Appendix 6 lists references showing ***effects of industry funding on research outcomes*** for myriad (mainly biomedical) research disciplines;
- Appendix 7 overviews the oft-neglected topics of wireless radiation adverse effects on regions containing ***medical implants*** (e.g., pacemakers, defibrillators, cochlear implants, dental implants, bone pins, plates, etc) and appendages (e.g., metal eyeglasses, earrings, metal jewelry, etc), as well as other ***micro/nano*** exogenous implant analogues;
- Appendix 8 describes ***adverse effects of automotive-based wireless radiation***.

1B. Unethical Research

1B1. Broad Definition

There are myriad definitions for 'unethical' research (e.g., http://icahn.mssm.edu/about-us/services-and-resources/faculty-resources/handbooks-and-policies/faculty-handbook/research-environment/research-integrity; https://oprs.usc.edu/training/booklets/; https://history.nih.gov/about/timelines_laws_human.html).

These definitions of 'unethical' research encompass a broad spectrum of actions. Much reporting of 'unethical' medical research in myriad media tends to focus on one aspect only: biomedical experiments performed on subjects who did not give 'informed consent'. The classic example reflects the experiments performed on concentration camp inmates by the Nazi-regime doctors during WWII, and the lesser-known experiments performed by their Japanese counterparts during WWII. These experiments were certainly horrific, but not unique. The test subjects in these experiments were neither ***informed*** about the nature and consequences of these experiments, nor did they give ***consent***.

1B2. Informed Consent

A comprehensive discussion of the importance of 'informed consent' in medical experimentation was presented in a journal Special Issue [Goodwin, 2016]. An excellent overview and rationale for informed consent in human experiments is shown in the following box (obtained from a booklet titled Informed Consent in Human Subjects Research), prepared by the Office for Protection of Research Subjects, University of Southern California (https://oprs.usc.edu/training/booklets/).

> Informed Consent is a voluntary agreement to participate in research. It is not merely a form that is signed but is a process, in which the subject has an understanding of the research and its risks. Informed consent is essential before enrolling a participant and ongoing once enrolled. Informed Consent must be obtained for all types of human subjects' research including; diagnostic, therapeutic, interventional, social and behavioral studies, and for research conducted domestically or abroad. Obtaining consent involves informing the subject about his or her rights, the purpose of the study, the procedures to be undergone, and the potential risks and benefits of participation. Subjects in the study must participate willingly. Vulnerable populations (i.e. prisoners, children, pregnant women, etc.) must receive extra protections. The legal rights of subjects may not be waived and subjects may not be asked to release or appear to release the investigator, the sponsor, the institution or its agents from liability for negligence.

There are three important concepts in this definition: research, informed, and consent.

Research

What is a research experiment? According to myriad Web sources, an experiment is a set of actions undertaken to

- make a discovery or
- test a hypothesis or
- demonstrate a known fact.

The first two of these can be classified as **research** experiments, and the third is a **demonstration** experiment. A further breakdown would be informative. There are ***proactive*** experiments, where established rules and procedures (the scientific approach) are used to plan, conduct, and report the experiment. There are ***reactive*** experiments, where the experiment is secondary to higher priority actions, and consequently is conducted and reported under more constrained conditions. The proactive experiments can be viewed generally as explicit or 'a priori', and the reactive experiments can be viewed generally as implicit or 'a posteriori'.

Where does wireless technology implementation and operation fit in this research experiment categorization? Wireless technology implementation has two major characteristics: development and operation of a technology to achieve targeted technical goals (***explicit***), and conduct of an experiment that may result in serious adverse health impacts (***implicit***). Of interest in the current document is the experiment (***implicit***) component.

Identification of wireless radiation health effects will result from both proactive and reactive experiments. The proactive experiments are (mainly) the thousands of laboratory-based studies (performed to estimate wireless radiation health impacts) that have been reported in the biomedical literature. The reactive experiments are (mainly) those studies that have been done after the previous generations of mobile networking technologies have been implemented (usually epidemiology), and those studies that will be done after 5G is implemented.

Thus, 5G implementation can be viewed mainly as an implicit reactive ***research*** experiment with respect to identifying myriad adverse health effects on the exposed population. It will also have a ***demonstration*** component, confirming thousands of pre-5G research studies that have shown adverse health effects from wireless radiation in 5G and non-5G frequency ranges. Because these studies tend to under-estimate real-life effects of wireless radiation, the full scope of adverse health effects from 5G operation under real-life conditions are currently unknown. Ascertainment of these adverse health effects will require 'a posteriori' reactive research experiments after 5G implementation, under today's 5G implementation scenario. A major concern, especially in the current environment of accelerating 5G implementation, is that serious longer-term latent health effects will be discovered ***only after 5G has been fully implemented***.

Informed

There is much information available in the open literature detailing the adverse health effects of wireless radiation. These adverse effects reflect the role of wireless radiation both as a ***promotor/accelerator and/or initiator*** of myriad biomedical abnormalities and serious diseases. However, the vast public is not informed (or is misinformed) of these adverse health effects by the:

- developers of wireless radiation systems,
- vendors of these systems,
- mainstream media
- government regulators of these systems, and
- Federal, State, and Local politicians who pass laws that accelerate implementation of these systems.

These stakeholders 1) **do not inform** the public of the demonstrated adverse effects of wireless radiation and, in many cases, 2) **misinform** the public that wireless radiation is safe from a health perspective.

Consent

Many segments of the public **do provide** consent to be exposed to wireless radiation, because of its perceived benefits to them. A small amount of this consent may be informed, and the providers of this consent may be gambling that they can escape the adverse health effects. Most of the consent is probably not informed, since most people will not do the independent research required to gather in the relevant information on adverse health effects, but will rely on the government's and mainstream media's misleading assurances that wireless radiation is safe.

However, other segments of the public **do not provide** consent to be exposed to wireless radiation from these implemented technologies. Unlike other forms of toxic stimuli (e.g., cigarettes, cocaine, alcohol, etc), where exposures may be individual or very local, wireless radiation exposure is very large in extent. With the advent of the latest generation of wireless radiation (5G), there may be 1) small cell towers erected outside of every few houses, with the consequent radiation blanketing the environment, and 2) thousands of satellites blanketing the Earth's surface with wireless radiation. There are Federal laws that essentially prevent opposition to construction and operation of these small cell towers, and prevent opposition to the launching and operation of these satellites. Forcing exposure to this harmful wireless radiation on members of the public who do not provide consent is the cornerstone of wireless radiation implementation and operation being labeled unethical medical experimentation.

Its context differs from some other technologies with serious adverse effects, such as automotive technology and cigarette smoking. For the most part, users of these other technologies have been informed about potential serious consequences, and non-users are impacted minimally (at least today). Those users are able to make a more informed choice.

1B3. Examples of Unethical Medical Experimentation

Many books and articles have been written concerning horrific medical experiments (that were performed in the USA over the past century) without obtaining 'informed consent' from the test subjects. These books describe a wide spectrum of experiments. Individual readers could have different opinions on whether any of the individual experiments reported are more or less 'unethical' than those in the Nazi concentration camps, or whether they are 'unethical' at all. Appendix 1 contains references to books and journal articles that describe some of these experiments (mainly, but not entirely, conducted in the USA or under USA auspices), based on Medline searches and Web sources. Like most research of this type, the conduct of the experiments and the experimental results are not advertised widely. I was not aware of most of these experiments prior to conducting the analysis on under-reporting of adverse events in my 2015 eBook "Pervasive Causes of Disease" [Kostoff, 2015].

The experiments reported in Appendix 1 cover the full spectrum of toxic stimuli, including biological, chemical, and nuclear. These are the three types of toxic stimuli that constitute the core of Weapons of Mass Destruction (WMD). Interestingly, with all of the USA's concern about potential WMD attacks from Russia, China, Iran, and North Korea, we have completely overlooked the ongoing and exponentially increasing WMD attack on the Homeland that has been occurring for at least two decades: 24/7 spewing of harmful wireless radiation in almost every corner of the USA, with far more to come if 5G is implemented!

The copious references identified in Appendix 1 are not the result of an exhaustive search; they were obtained after a very brief survey. There are undoubtedly many other examples (of 'unethical' medical experiments) published already that were missed by the survey. Given the odious nature of these experiments, there are probably far more experiments whose disclosure has not yet seen the light of day. As shown in the tobacco and asbestos examples in section 9C of Kostoff [2015], most of this information comes to light either from 1) whistleblowers or 2) 'discovery' resulting from lawsuits. In addition, some investigators may stumble across evidence of this type of 'unethical' research while doing relatively unrelated types of investigations.

Documentation of many types of 'unethical' medical experiments may:

- not have been done, or
- have been done and destroyed, or
- have been done but distorted to protect the miscreants.

This is why retrospective analysis of this type of 'research', which in many cases relies heavily on the printed word as 'proof', may be highly under-reflective of the full spectrum of what was actually done in these experiments (e.g., Stephen Kinzer's description of the records destroyed by the Head of the CIA's MK-Ultra program https://www.c-span.org/video/?464648-1/poisoner-chief).

While there are many stages of the medical research process that could be subjected to 'unethical' practices (e.g., those outlined in Chapter 9 of Kostoff [2015], including selection of the most important research problems for funding, conducting the research, disseminating the results of the research, etc), conducting the medical research experiments 'unethically' has received the most attention by far. The references in Appendix 1, and additional books and journal and magazine articles on unethical medical research experiments, are testimony to this imbalance.

Books and articles only tell part of the larger story. A more representative reporting on the damage from any type of 'unethical' medical research would reflect the pain, suffering, and premature mortality resulting from the medical research experimentation. A simple estimate of the experiment's damage could be obtained by integrating the number of people affected by the 'unethical' medical experimentation and the degree of damage experienced by each person. This could be viewed as a 'weighted' impact of the adverse effects of the unethical medical experimentation.

In the most widely reported examples of 'unethical' medical research (the medical experiments performed in the Nazi concentration camps during WWII), perhaps a few thousand prisoners were involved; it is difficult to find accurate information for actual numbers of prisoners involved. Further, it is difficult to separate out the 1) many thousands of German citizens subjected to forced sterilization procedures starting in 1933 and 2) many deliberately exterminated in the concentration camps, from 3) those who suffered from the medical experiments in the camps and died as a result of the experiments alone.

In the references in Appendix 1

- some of the 'unethical' medical experiments described involved under a hundred test subjects,
- many of the 'unethical' medical experiments described tended to involve on the order of hundreds of test subjects (who did not provide 'informed consent'), and
- in some rarer cases, perhaps thousands of test subjects were involved.

Many of these experiments, in parallel with the spirit of the Nazi concentration camp experiments, involved people confined in large institutions who were (usually) not told the full story of the nature of the experiments, or, if they were told, either did not 1) understand it or 2) give 'informed consent'. These people were confined in prisons, the military service, mental institutions, children's institutions, etc.

How do the above odious procedures in these references differ conceptually from the recent trend toward government effectively promoting/mandating implementation of wireless radiation infrastructure whose safety has not been demonstrated, but (a fraction of) whose adverse health effects have been widely demonstrated?

Based on what has been reported in the experiments referenced in Appendix 1 (which could in fact be the tip of a much larger unreported iceberg), perhaps on the order of 10,000-30,000 people may have been subjected to 'unethical' medical experiments in the past century (excluding those who unwittingly participated in clinical trials that were "off-shored" to (typically) developing countries with knowingly less stringent test subject protections [Kostoff, 2015, section 9D3]). A few thousand of these test subjects would have died prematurely, and most would have suffered unnecessarily. These, of course, are horrific numbers. Unfortunately, they pale in comparison to what can be expected if wireless radiation infrastructure is expanded domestically and globally to satisfy the requirements of 5G. The following box shows one estimate of potential adverse effects from wireless radiation.

One of the many adverse health effects of wireless radiation is cancer of the brain, especially gliomas. What approximate increases in glioma incidence can be expected from widespread expansion of wireless radiation?

There are different estimates of glioma incidence and trends in glioma incidence. For an approximate estimate, Rasmussen et al [2017] estimates the glioma incidence in the Danish population at about 7/100,000, a figure in line with other national and global estimates. Additionally, Phillips et al [2018] presents evidence of a 100% increase in Glioblastoma Multiforme from 1995-2015, a major component of glioma. Some of this increase may have been due to wireless radiation exposure, since that time period was associated with a major expansion of cell phone and other wireless device use. For approximate estimation purposes, assume the wireless-free glioma incidence to be about 5/100,000.

Hardell et al [2011] showed, in a case-controlled study, that glioma incidence doubled for those who starting using cell phones as adults (>20 years old), were 'heavy' users (>30 minutes per day), and used cell phones for more than ten years. Hardell also showed glioma incidence quadrupled for those who started using cell phones younger than twenty years old, were heavy users, and used cell phones for more than ten years.

If we apply Hardell's conservative doubling estimate to all potential users, then we can expect an increased glioma incidence per year of about 5/100,000. By the time 5G is rolled out, the global population will be at least eight billion. If we assume ¾ of the global population will be cell phone users and/or exposed to cell towers and other sources of wireless radiation, then about six billion people would be the pool for potential glioma victims from wireless radiation. Multiplying 5/100,000 by 6,000,000,000 yields 300,000 new cases of glioma/year.

In one year, the deaths from glioma alone attributed to wireless radiation will swamp all the deaths from all the horrific unethical medical experiments of the twentieth century referenced in Appendix 1!

This number was obtained using the most conservative estimates of Hardell and the incidence data, and it didn't take into account the increase in glioma incidence that would be expected as latency times increase. For smoking, the average latency period between initiation of smoking and lung cancer is between twenty and thirty years, depending on which database was examined. The fact that glioma incidence shows measurable increases after only a ten-year latency period should be most disturbing, and does not bode well for glioma incidences after a twenty, thirty, or forty-year latency!

Again, glioma is but one of the large numbers of adverse health effects potentially resulting from exposure to wireless radiation. Integrating over all the adverse health effects potentially resulting from the wireless radiation experiment would yield numbers of ***experiment-based*** premature deaths and enhanced suffering unparalleled in human history!

Given the magnitude of 5G projected global implementation, the numbers of people that will be exposed to this radiation, the numbers of people expected to suffer myriad adverse effects from this technology, and the lack of credible 'informed consent' from the vast majority of these people, we are well justified in calling global implementation of mobile networking technology **The Largest Unethical Medical Experiment in Human History!**

Finally, in the spirit of the 'unethical' medical experiments referenced in Appendix 1,

it is the poor and dispossessed who will suffer the most from wireless radiation exposure.

This is because wireless radiation plays a dual role of ***initiator*** and ***promoter/accelerator*** of serious disease, as will be shown in the next chapter. In its ***promoter/accelerator*** role, it can accelerate the progression of existing serious diseases such as cancer, and/or, through synergy, can produce serious adverse health effects when combined with other toxic stimuli that neither constituent of the combination could produce in isolation.

Many toxic stimuli, such as harsh chemicals, biotoxins, ionizing radiation sources, vibrating machinery, prolonged sitting doing repetitive tasks, high air pollution, etc, are used/experienced by the poorest members of society in their occupations, and many toxic stimuli, such as air pollutants, toxic wastes, etc, are very prevalent in their residential environments. Thus, people who spray pesticides in farm labor or household applications, people who do cleaning with harsh chemicals, people who dispose of hazardous materials, basically, ***people who do the dirty work in our society and live in dirty environments***, are already leading candidates for higher risk of serious diseases. Adding a wireless radiation ***promoter/accelerator*** to their residential and occupational environments will radically increase their chances for developing serious diseases. Closing the 'digital divide' for them will translate to increased suffering and reduced longevity!

Chapter 2 – Adverse Impacts of Wireless Radiation

2A. Overview

Wireless communications have been expanding globally at an exponential rate. The latest imbedded version of mobile networking technology is called 4G (fourth generation), and the next generation (5G) is in the early implementation stage. Neither 4G nor 5G have been tested for safety in any credible real-life scenarios. The current chapter assesses the medical and biological studies that have been performed and then published in the biomedical literature, and shows why they are deficient relative to identifying adverse health and safety effects.

However, even in the absence of the missing real-life components (which tend to exacerbate the adverse effects of the wireless radiation shown in the biomedical literature), the published literature shows there is much valid reason for concern about potential adverse health effects from both 4G and 5G technology. The studies reported in the literature should be viewed as extremely conservative, underestimating the adverse impacts substantially.

2A1. The Context of Wireless Radiation Health and Safety Research

Before addressing the technical and biological details of wireless radiation health and safety research shown in the published literature, the context in which this literature has been generated will be discussed.

The results shown in the literature cannot be separated from the context in which this research has been sponsored, conducted, and disseminated!

In the USA (and in most, if not all, countries), the two major sponsors of wireless radiation health and safety research are the Federal government and the wireless radiation industry, in that order. Both of these organizations have a strong intrinsic conflict-of-interest with respect to wireless radiation.

2A1a. Intrinsic Federal government wireless radiation conflict-of-interest

The Federal government is a strong **promoter** of wireless radiation infrastructure development and rapid expansion, most recently supporting accelerated implementation of 5G infrastructure. Every

- Congressional evaluation of 5G I have heard (or read),
- Congressperson's statement on 5G I have heard (or read),
- Presidential proclamation on 5G I have heard (or read), and
- FCC proclamation on 5G I have heard (or read),

has unabashedly supported the ***most accelerated implementation of 5G infrastructure***.

The Federal government that ***promotes*** accelerated implementation of wireless radiation technology also 1) ***sponsors*** research examining the technology's potential adverse effects and 2) ***regulates*** the technology's potentially adverse impacts on the public. The fact that these development, regulation, and safety functions may be assigned to different Executive Agencies within the Federal government is irrelevant from an independence perspective. ***The separate Executive Agencies in the Federal government are like the tentacles of an Octopus; they operate synchronously under one central command.***

The wireless promoters' main objectives of developing and implementing the technology rapidly are enabled by suppressing knowledge (to the public) of potential adverse effects from the technology's operation. These fundamental conflicts impact the objectivity of the health and safety R&D sponsors and performers. Any ***Federal research sponsor*** of wireless radiation technology safety would be highly conflicted between 1) a desire to satisfy Executive and Legislative objectives of accelerating expansion of wireless radiation technology and implementation and 2) sponsoring objective research focused on identifying and reporting adverse effects of wireless radiation expected under real-life conditions. Likewise, any ***sponsored research performer*** addressing wireless radiation technology safety would be highly conflicted between 1) reporting the actual adverse effects expected under real-life conditions and 2) the desire to satisfy wireless radiation promotional objectives of the research sponsors in order to maintain long-range funding.

2A1b. Intrinsic wireless radiation industry conflict-of-interest

The wireless radiation industry is obviously a strong promoter of accelerated development and implementation of wireless radiation devices and infrastructure, and is a sponsor of wireless radiation and safety research. ***Trillions of dollars in revenues are potentially at stake in successful promotion and adoption of wireless radiation infrastructure and technology!*** The industry's conflicts with respect to promotion and safety research are similar to those of the Federal government listed above.

The wireless industry's role in suppressing information about the adverse impacts of wireless radiation was described eloquently in a 2018 Nation article (https://www.thenation.com/article/how-big-wireless-made-us-think-that-cell-phones-are-safe-a-special-investigation/). As this exposé shows, studies on health effects were commissioned by the wireless radiation industry in the 1990s under the management of Dr. George Carlo. The adverse effects shown were downgraded and suppressed, in the spirit of similar suppression by the tobacco and fossil energy industries, as stated in the Nation article:

"Carlo's story underscores the need for caution, however, particularly since it evokes eerie parallels with two of the most notorious cases of corporate deception on record: the campaigns by the tobacco and fossil-fuel industries to obscure the dangers of smoking and climate change, respectively. Just as tobacco executives were privately told by their own scientists (in the 1960s) that smoking was deadly, and fossil-fuel executives were privately told by their own scientists (in the 1980s) that burning oil, gas, and coal would cause a "catastrophic" temperature rise, so Carlo's testimony reveals that wireless executives were privately told by their own scientists (in the 1990s) that cell phones could cause cancer and genetic damage…..Like their tobacco and fossil-fuel brethren, wireless executives have chosen not to publicize what their own scientists have said about the risks of their products. On the contrary, the industry—in America, Europe, and Asia—has spent untold millions of dollars in the past 25 years proclaiming that science is on its side, that the critics are quacks, and that consumers have nothing to fear. This, even as the industry has worked behind the scenes—again like its Big Tobacco counterpart—to deliberately addict its customers. Just as cigarette companies added nicotine to hook smokers, so have wireless companies designed cell phones to deliver a jolt of dopamine with each swipe of the screen."

While the wireless radiation industry doesn't play a formal role in regulating the safety aspects of wireless radiation, it plays a strong de facto role. In addition to its lobbying efforts to minimize regulations on wireless radiation exposure levels, it plays a revolving-door role with respect to regulation.

The previous FCC Chairman had been President of the National Cable & Telecommunications Association (NCTA) and CEO of the Cellular Telecommunications & Internet Association (CTIA) before assuming his FCC Chairmanship. In recognition of his work in promoting the wireless industry, he was inducted into the Wireless Hall of Fame in 2003 and in 2009 (https://en.wikipedia.org/wiki/Tom_Wheeler). The present FCC Chairman served as Associate General Counsel at Verizon Communications Inc., where he handled competition matters, regulatory issues, and counseling of business units on broadband initiatives (https://en.wikipedia.org/wiki/Ajit_Pai#cite_note-Bio-2). As is the case with so many other Federal regulatory agencies [Kostoff, 2015-Chapter 9; 2016], the FCC is essentially an agency captured by industry [Alster, 2015]!

So, in the two most recent Administrations, under two supposedly very different Presidents, the FCC Chairmen had been, in different ways, lobbyists for the wireless radiation technology industry. Both were (and are) extremely ardent promoters of the most rapid acceleration of implementation of 5G infrastructure and associated devices and technologies.

2A1c. Relation of wireless radiation health and safety research to sponsors' and performers' conflicts-of-interest

The incentives for ***sponsors*** of wireless radiation health and safety research to fund studies that will help promote accelerated expansion of wireless radiation devices and infrastructure are many and the disincentives are essentially non-existent. Likewise, incentives for ***performers*** of wireless radiation health and safety research to conduct studies that will help promote accelerated expansion of wireless radiation devices and infrastructure are many and the disincentives are few. Because of this unfortunate reality,

EVERY wireless radiation health and safety study/experiment whose results support the wireless radiation promotion objectives of the organization(s) that sponsor these studies must receive the highest level of scrutiny.

There is not a credibility symmetry between studies whose results 1) support the promotional objectives of their sponsors or 2) do not support the promotional objectives of their sponsors. For studies/experiments of equally high research/scientific quality, those studies that do not support the promotional objectives of their sponsors should be assigned relatively higher credibility priority than those that do support the promotional objectives of their sponsors. This should not be interpreted as a lack of absolute credibility for studies that support the promotional objectives of their sponsors. Many may very well be credible, as discussed further in section 2F.

However, research findings opposing the promotional objectives of the sponsors may result in termination of further funding for the project, and adverse career and financial consequences for the performer(s). Conversely, research findings supporting the promotional objectives of the sponsors will most likely lead to continued and enhanced funding for the project, and very positive career and financial impacts for the performer(s). Therefore, high quality research studies whose results could impose serious career and financial risks for their performers should rank higher in the credibility chain.

These conflicts-of-interest of researchers who accept funding from wireless radiation promoters extend well beyond the papers and studies they publish. This category of wireless radiation researchers tends to populate the Advisory Committees that help set the exposure safety studies imposed by government regulatory agencies. Hardell has done a comprehensive evaluation of some of the more influential Advisory Committees [Hardell, 2017], especially ICNIRP and WHO, and has shown clearly the inter-locking linkages among these proxies of the wireless radiation promoters.

Operationally, the wireless radiation regulatory commissions, their advisory committees, their health and safety research sponsors, and some of the researchers sponsored by the wireless radiation promoters, along with the mainstream media, serve as ***the de facto marketing arm of the wireless radiation promoters***, in their attempts to mislead the public into believing wireless radiation under present day exposure limits is safe!

2A1d. Relation of wireless radiation health and safety research to publishers' conflicts-of-interest

Some journal publishers of articles concerning health and safety effects of wireless radiation have similar conflicts of interest. Many journals are not independent from government or industry sponsorship, in whole or in part, directly or indirectly. This conflict-of-interest is addressed further in section 2F. These journals control the review process by which articles are selected for publication, and it is extremely easy for a journal to select articles for publication that will align strongly with the promotional interests of the organizations or people that contribute to their revenue stream. These direct or indirect journal sponsors include:

- Promotional organizations that contribute directly to the journals;
- Promotional organizations that contribute directly to professional societies that sponsor many of the 'leading' journals;
- Individuals who receive funding from industrial or governmental organizations promoting wireless radiation technology and who
    - contribute directly to the journals and/or
    - contribute to professional societies that sponsor many of the 'leading' journals

Anyone who has read thousands of wireless radiation journal article abstracts on health and safety would have little problem in identifying those journals that rarely publish results opposing the promotional objectives of government and industry (see Slesin [2006] for ***allegations*** of possible bias in one journal's publication patterns of microwave-induced genotoxic results). Equally, they would have little problem in identifying those authors or author institutions that even more rarely publish results opposing the promotional objectives of government and industry. If we take into account the credibility asymmetry between studies whose results 1) support the promotional objectives of their sponsors or 2) do not support the promotional objectives of their sponsors, then a much different picture of the wireless radiation health and safety research literature emerges. Many of the so-called conflicting results disappear when credibility weightings are applied, and the true serious adverse effects resulting from this harmful technology are shown in detail. The reader should keep this credibility asymmetry in mind when evaluating the myriad adverse health effects shown in sections 2D and 2E.

2B. Wireless Radiation/Electromagnetic Spectrum

This section overviews the electromagnetic spectrum, and delineates the parts of the spectrum on which this monograph will focus. The electromagnetic spectrum encompasses the entire span of electromagnetic radiation. The spectrum includes: ionizing radiation (gamma rays, x-rays, and the extreme ultraviolet, with wavelengths below ~10^{-7} m and frequencies above ~$3x10^{15}$ Hz); non-ionizing visible radiation (wavelengths from ~$4x10^{-7}$ m to ~$7x10^{-7}$ m and frequencies between ~$4.2x10^{14}$ Hz and ~$7.7x10^{14}$ Hz); non-ionizing non-visible radiation (short wavelength radio waves and microwaves, with wavelengths between ~10^{-3} m and ~10^{5} m and frequencies between ~$3x10^{11}$ to ~$3x10^{3}$ Hz; long wavelengths, ranging between ~10^{5} m and ~10^{8} m and frequencies ranging between $3x10^{3}$ and 3 Hz).

The low frequencies (3 Hz–300 KHz) are used for electrical power line transmission (60 Hz in the U.S.) as well as maritime and submarine navigation and communications. Medium frequencies (300 KHz–900 MHz) are used for AM/FM/TV broadcasts in North America. Lower microwave frequencies (900 MHz–5 GHz) are used for telecommunications such as microwave devices/communications, radio astronomy, mobile/cell phones, and wireless LANs. Higher microwave frequencies (5 GHz– 300GHz) are used for radar and proposed for microwave WiFi, and will be used for 'high-band' 5G communications. Terahertz frequencies (300 GHz–3000 GHz) are used increasingly for imaging to supplement X-rays in some medical and security scanning applications [Kostoff and Lau, 2017; Kostoff, 2019a; Kostoff et al, 2020].

In the study of non-ionizing EMF radiation health effects reported in this monograph, the frequency spectrum ranging from 3 Hz to 300 GHz is covered, with particular emphasis on the high frequency communications component ranging from ~1 GHz to ~300 GHz. A previous review found that pulsed electromagnetic fields applied for relatively short periods of time could sometimes be used for therapeutic purposes, whereas chronic exposure to electromagnetic fields in the power frequency range (~60 Hz) and microwave frequency range (~1 GHz-tens GHz) tended to result in detrimental health effects [Kostoff and Lau, 2013, 2017]. Because of present concerns about the rapid expansion of new communications systems without adequate safety testing, more emphasis will be placed on the communications frequencies in this monograph.

2C. Modern Non-Ionizing EMF Radiation Exposures

In ancient times, sunlight and its lunar reflections provided the bulk of the visible spectrum for human beings (with fire a distant second and lightning a more distant third). Now, many varieties of artificial light (incandescent, fluorescent, and light emitting diode) have replaced the sun as the main supplier of visible radiation during waking hours. Additionally, EMF radiation from other parts of the non-ionizing spectrum has become ubiquitous in daily life, such as from wireless computing and telecommunications. In the last two or three decades, the explosive growth in the cellular telephone industry has placed many residences in metropolitan areas within less than a mile of a cell tower. Future implementation of the next generation of mobile networking technology, 5G, will increase the cell tower geographical densities by an

order of magnitude. Health concerns have been raised about non-ionizing EMF radiation from (1) mobile communication devices, (2) occupational exposure, (3) residential exposure, (4) wireless networks in homes, businesses, and schools, and (5) other non-ionizing EMF radiation sources such as 'smart meters' and 'Internet of Things'.

2D. Demonstrated Biological and Health Effects from Prior Generations of Wireless Networking Technology

2D1. Limitations of Previous Wireless Radiation Health Effects Studies

There have been two major types of studies performed to ascertain biological and health effects of non-ionizing radiation: laboratory and epidemiology. The laboratory tests provide the best scientific understanding of the effects of wireless radiation, but do not reflect the real-life operating environment in which wireless radiation is embedded. There are three main reasons that laboratory tests do not reflect real-life exposure conditions for human beings.

First, the laboratory tests have been performed mainly on animals, especially rats and mice. Because of physiological differences, there have been continual concerns about extrapolating small animal results to human beings. Additionally, while inhaled or ingested substances can be scaled from small animals to human beings relatively straight-forwardly, radiation may be more problematical. For non-ionizing radiation, penetration depth is a function of frequency, tissue, and other parameters, and radiation of a given wavelength could penetrate much deeper into the (small) animal's interior than similar wavelength radiation in humans. Different organs and tissues would be affected, with different power densities.

Second, the typical incoming EMF signal for many/most laboratory tests performed in the past consisted of the single carrier wave frequency; the lower frequency superimposed signal containing the information was not always included. This omission may be important. As Panagopoulos states: "It is important to note that except for the RF/microwave carrier frequency, Extremely Low Frequencies – ELFs (0–3000Hz) are always present in all telecommunication EMFs in the form of pulsing and modulation. There is significant evidence indicating that the effects of telecommunication EMFs on living organisms are mainly due to the included ELFs…. While ~50% of the studies employing simulated exposures do not find any effects, studies employing real-life exposures from commercially available devices display an almost 100% consistency in showing adverse effects". [Panogopoulos, 2019]. These effects may be exacerbated further with 5G: "with every new generation of telecommunication devices…..the amount of information transmitted each moment…..is increased, resulting in higher variability and complexity of the signals with the living cells/ organisms even more unable to adapt [Panogopoulos, 2019]"

Third, these laboratory tests typically involved one stressor (wireless radiation) and were performed under pristine conditions. This contradicts real-life exposures, where humans are exposed to multiple toxic stimuli, in parallel or over time. In perhaps five percent of the wireless radiation studies reported in the literature, a second stressor (mainly biological or chemical toxic stimuli) was added, to ascertain whether additive, synergistic, potentiative, or antagonistic effects were generated by the combination [Kostoff and Lau, 2013, 2017; Juutilainin et al, 2008; Juutilainin et al, 2006].

Combination experiments are extremely important because, when other toxic stimuli are considered in combination with non-ionizing EMF radiation, the synergies tend to enhance the adverse effects of each stimulus in isolation. In other words, combined exposure to 1) toxic stimuli and 2) non-ionizing EMF radiation translates into much lower levels of tolerance for each toxic stimulus in the combination relative to its exposure levels that produce adverse effects in isolation. So, the regulatory exposure limits for non-ionizing EMF radiation when examined in combination with other potentially toxic stimuli should be far lower for safety purposes than those derived from non-ionizing EMF radiation exposures in isolation [Kostoff et al, 2020].

Thus, almost all of the laboratory tests that have been performed are flawed with respect to demonstrating the full adverse impact of the wireless radiation. Either 1) non-inclusion of signal information or 2) using single stressors only 3) tends to underestimate the seriousness of the adverse effects from non-ionizing radiation. Excluding ***both*** of these phenomena from experiments, as was done in the vast majority of cases, tends to amplify this underestimation substantially. Therefore, the results (of adverse effects from wireless radiation exposure) reported in the biomedical literature should be viewed as 1) extremely conservative and 2) the very low 'floor' of the seriousness of the adverse effects, not the 'ceiling'.

The epidemiology studies typically involved human beings who had been subjected to myriad known and unknown stressors prior to (and during) the study. The wireless radiation exposure levels from e.g. the cell tower studies reported in Kostoff and Lau [2017] associated with increased cancer incidence tended to be orders of magnitude lower than e.g. those exposure levels generated in the recent highly-funded NTP studies [Melnick, 2019] and other laboratory studies associated with increased cancer incidence. The inclusion of real-world effects in the cell tower studies most likely accounted for the orders of magnitude wireless radiation exposure level decreases that were associated with the initiation of increased cancer incidence.

Thus, the laboratory tests were conducted under very controlled conditions not reflective of the real-world, while the epidemiology studies were performed in the presence of many stressors, known and unknown, reflective of the real-world. The exposure levels of the epidemiology studies were, for the most part, uncontrolled.

2D2. Adverse Health Effects Identified in Major Review Studies

Many thousands of papers have been published over the past sixty+ years showing adverse effects from wireless radiation applied in isolation or as part of a combination with other toxic stimuli. Extensive reviews of these wireless radiation biological and health effects have been published, including [Belpomme et al, 2018; Desai et al, 2009; Di Ciaula, 2018; Doyon and Johansson, 2017; Havas, 2017; Kaplan et al, 2016; Kostoff and Lau, 2013, 2017; Kostoff et al, 2020; Lerchl et al, 2015; Levitt and Lai, 2010; Miller et al, 2019; Pall, 2016, 2018; Panagopoulos, 2019; Panagopoulos et al, 2015; Russell, 2018; Sage and Burgio, 2018; Van Rongen et al, 2009; Yakymenko et al, 2016; Bioinitiative, 2019].

In aggregate, for the high frequency (radiofrequency-RF) part of the spectrum, these reviews show that RF radiation below the FCC guidelines can result in:

-carcinogenicity (brain tumors/glioma, breast cancer, acoustic neuromas, leukemia, parotid gland tumors),

-genotoxicity (DNA damage, DNA repair inhibition, chromatin structure),

-mutagenicity, teratogenicity,

-neurodegenerative diseases (Alzheimer's Disease, Amyotrophic Lateral Sclerosis),

-neurobehavioral problems, autism,

-reproductive problems, pregnancy outcomes,

-oxidative stress, inflammation, apoptosis, blood-brain barrier disruption,

-pineal gland/melatonin production, sleep disturbance, headache,

-irritability, fatigue, concentration difficulties, depression, dizziness, tinnitus,

-burning and flushed skin, digestive disturbance, tremor, cardiac irregularities, and can

-adversely impact the neural, circulatory, immune, endocrine, and skeletal systems.

The effects range from myriad feelings of discomfort to life-threatening diseases. From this perspective, RF exposure is a highly pervasive cause of disease!

2D3. Adverse Health Effects from Open Literature Analysis

2D3a. Overview

To corroborate the findings from the major review studies of the previous section, an analysis of a representative sample of the wireless radiation adverse health effects literature was performed. A relatively simple query was used to retrieve records related to adverse health effects from wireless radiation. Some filtering was done to remove records that did not identify adverse health effects, but because of extensive use of titles (and sometimes abstracts) that discuss methodologies rather than results, some/many records were retrieved that did not demonstrate adverse health effects.

In all, 5311 records with abstracts were retrieved from Medline (Pubmed), and these records were categorized by three different methods: manual taxonomy; factor analysis taxonomy; text clustering taxonomy. The three methods and their results will be briefly summarized here, and the more detailed results, including category record titles, will be presented in Appendices 2-4.

2D3b. Manual taxonomy results

Based on the factor analysis (section 2D3c) and text clustering (2D3d) results, as well as reading thousands of abstracts from the full database, a manual taxonomy of adverse health effects from wireless radiation was constructed. Appendix 2 presents this taxonomy (Table A2-1), and the titles of the records that were assigned to each category in the taxonomy. The record titles give a better appreciation for the contents of each category than the brief category heading.

This ***manual taxonomy is the most relevant*** (of the three taxonomies presented) to the main objective of identifying and categorizing specific adverse health effects from wireless technology, since it was not dependent on any algorithm to determine adverse effects categories and received a ***higher level of title filtering*** than the other two. Table A2-1 (reproduced in the following) presents the categories in the taxonomy, and a strong condensation of the key phrases 1) used to define the category and 2) link to the record titles shown in Appendix 2. A more detailed manual taxonomy, with orders-of-magnitude more phrases, is shown in Appendix 2.

The adverse effects identified in the manual taxonomy cover those summarized in the comprehensive review analyses described previously, and go well beyond. While all the categories shown are problematical and harmful, the most researched categories with perhaps the most serious adverse effects are ***cancer/tumors, neurodegenerative diseases, reproduction problems, and genotoxicity***. Thus, even confining these results to the non-classified open literature, many of which are based on single stressor experiments that tend to downplay greatly real-life adverse effects, there is more than enough hard evidence that wireless radiation 1) ***can be extremely harmful in real-life environments***, and 2) ***needs to be subjected to orders-of-magnitude harsher exposure limitations*** than is the case today. In Appendix 2, the categories in Table A2-1 are hyperlinked to their respective record title sections.

Table A2-1 – Manual Taxonomy

CATEGORY	KEY PHRASES
Cancer/Tumors	cancer, leukemia, glioma, lymphoma, melanoma, Hodgkin's disease, tumor, acoustic neuroma, meningioma
Neurodegenerative	memory, central nervous system, learning, neurodegenerative, Alzheimer's disease, cognition, amyotrophic lateral sclerosis, dementia, epilepsy, multiple sclerosis, cognitive impairment, seizures, autism
Reproduction	pregnancy, reproductive, sperm, embryos, testicular, fertility, embryo, testosterone, infertility
Genotoxicity	DNA damage, genotoxic, micronuclei, mutagenic, strand breaks, chromatin, mutation, chromosome aberrations,
Cardiovascular	Cardiac, cardiovascular, pacemaker, implanted, Cardiovascular disease, arrhythmia, arterial blood pressure, ventricular fibrillation
Immunity	lymphocytes, immune system, immunity, leukocytes, antibodies, neutrophils, autoimmune, macrophage,
Biomarkers	apoptosis, oxidative stress, Malondialdehyde, reactive oxygen species, superoxide dismutase, lipid peroxidation, inflammation, oxidation, ornithine decarboxylase, barrier permeability, atrophy, C-reactive protein, oxidative damages
Sensory Disorders	auditory, acoustic, hypersensitivity, electromagnetic hypersensitivity, cataract, tinnitus, dermatitis, cataractogenic, pain sensitivity, pain threshold
Discomfort Symptoms	depression, anxiety, headache, dizziness, depressed, vertigo, nausea, low back pain
Congenital Abnormalities	malformations, teratogenic, congenital malformations, cleft palate,
Circadian Rhythym and Melatonin	melatonin, sleep, circadian, insomnia, pineal function
Chronic Conditions	metabolism, glucose, endocrine, cholesterol, Diabetes, calcium homeostasis, obesity

2D3b1. Adverse effects of wireless radiation on food chain

The above taxonomy (and its associated records) focuses on the direct linkage between wireless radiation exposure and biomarkers, symptoms, and diseases. As such, these effects can be viewed as direct effects. Equally important, but usually overlooked in any discussions of adverse effects of wireless radiation, are the indirect effects, especially those on the ecological infrastructure that supports human life.

An analogy to war and conflict may be instructive. When one examines the great wars and battles of human history, especially those that persisted for more than very short periods, the critical role of logistics in determining the outcome becomes obvious. Many wars/battles have been won or lost by the adequacy and timeliness of logistical supplies and support.

The struggle for survival of human life on Earth is similarly dependent on the logistical food supply chain. At the foundation of this supply chain (before the farmers become involved in harvesting its bounty) are the insects, seeds, flora, trees, etc, that enable the bountiful growth of the myriad potential foods. If the integrity of this foundational logistical supply chain is threatened in any way, then both the animals and plant products we consume become unavailable.

There is a substantial literature on the adverse impacts of wireless radiation on this foundational logistical supply chain. These adverse effects are from the pre-5G exposures, and would include enhanced coupling from the higher frequency harmonics. Many of these supply chain elements (e.g., insects, seeds, larvae, etc) are very small, and we could expect enhanced resonance/energy coupling from the shorter-wavelength 5G radiation when implemented. This indirect impact of wireless radiation may turn out to be at least as important (if not more important) as the direct impact of wireless radiation on human survival! At the end of Chapter 3 are a few references showing the harmful effects of wireless radiation on the foundational food supply chain. They are the tip of the iceberg of a much larger literature on adverse effects of wireless radiation on the foundational food supply chain.

From a broader perspective, most of the laboratory experiment component of the wireless radiation adverse effects literature can be viewed as related to the foundational food supply chain. Much of this research is focused on mice, rats, insects, small birds, small fish, etc. These species tend to be prey of larger animals/fowl/fish, and eventually make their way to the human food table. Any environmental factor that affects the health of these species adversely will eventually impacts the humans who are at the end of that chain. In reality, we have accumulated a massive literature describing the adverse impacts of wireless radiation on myriad contributing components to our food supply, and the results do not bode well for our future ability to feed the existing world's population, much less the growing world's population!

2D3b2. Implants and Appendages

The adverse impacts of wireless radiation on myriad medical implants don't get much discussion in the literature, especially passive implants (defined below), and especially with regard to radiofrequency radiation. A number of articles in the database addressed non-organic implants, which are foreign bodies inserted into humans and animals for medical purposes. Non-organic implants addressed in the present database are typically not rejected by the immune system like organic foreign substances (although some adjuvants such as metal could induce autoimmune responses [Loyo et al, 2013]). Non-rejection does not mean they are safe, especially from exposure to wireless radiation.

There were two major types of implants covered by the database articles showing adverse effects: active implants that produced electrical signals mainly for controlling heart irregularities (e.g., pacemakers, defibrillators) and hearing deficiencies (e.g., cochlear implants), and passive metallic implants for structural support (e.g., dental implants, bone pins, plates, etc). Additionally, there are articles addressing adverse effects from wireless radiation in the vicinity of metallic appendages (e.g., metallic eyeglasses, metallic jewelry, etc).

The external EMF from microwaves (and other sources) could 1) impact the electrical operation of the active implants adversely, 2) increase the Specific Absorption Rate (SAR) values of tissue in the vicinity of the passive implants substantially because of resonance effects, and 3) increase the flow and acidity of saliva in the vicinity of dental structures. While the EMF effects on the cochlear implants could adversely affect auditory capability, EMF effects on the heart-related implants could potentially be life-threatening. The increased SAR values around the passive metal implants could result in increased tissue temperatures, and could adversely impact integration and longevity of the passive metallic implants.

In the mouth, the combination of 1) increased tissue temperatures in proximity to the implant or other orthodontic structures and 2) increased flow rate and acidity of saliva could lead to 3) increased leaching of heavy metals. Exposure to heavy metals is a major contributor to myriad chronic diseases [Kostoff, 2015]. The question then becomes: what other adverse health effects from the exposure of both the active and passive implants to increasing levels of wireless radiation have not been identified or addressed?

Appendix 7 addresses this issue of wireless radiation adverse effects related to medical implants and appendages in more detail, and additionally addresses potential wireless radiation adverse effects on tissues imbedded (deliberately or inadvertently) with exogenous-based nanoparticles that effectively act as micro/nano-implants. These nanoparticle-imbedded tissues may have the potential for enhanced energy absorption from the incoming RF signal, and may exhibit potentially harmful thermal transients (over and above the potential thermal transients resulting from the pulsed high peak-to-average power of the RF signal) that would be camouflaged under the wide averaging time periods in the FCC Guidelines.

2D3c. Factor analysis taxonomy results

The 5,311 records in the retrieved and ***partially*** filtered adverse health effects database were imported into the VP software [VP, 2019], and a factor analysis was performed. Thousands of MeSH Headings extracted by the VP software were inspected visually, and those directly applicable to adverse health effects were selected. The software then used these selected MeSH Headings to generate a factor matrix, which identified the main adverse health effects themes of the database. Appendix 3 presents this taxonomy (Table A3-1), and the titles of the records that were assigned to each category in the taxonomy. The titles give a better appreciation for the contents of each category than the brief category heading.

Table A3-1 (reproduced from Appendix 3) follows. It presents the factors/categories in the taxonomy, and the key MeSH Headings used to define the factor/category and link to the record titles shown in Appendix 3. In Appendix 3, the factors in Table A3-1 are hyperlinked to their respective record titles.

Table A3-1 - Factor Analysis Taxonomy

FACTOR THEME	MESH HEADINGS
1 Electromagnetic hypersensitivity and inflammation	C-Reactive Protein, Liver Diseases, Thyroid Diseases, Inflammation, Tonsillitis, Hypersensitivity
2 Coronary artery disease	Plaque, Atherosclerotic, Coronary Artery Disease, Diabetes Mellitus, Carotid Artery Diseases, Inflammation, Hypertension
3A Congenital abnormalities	Cleft Lip, Cleft Palate, Calcification, Physiologic, Congenital Abnormalities
3B Mammary tumors	Fibroadenoma, Adenoma, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma
4 Male infertility	Sperm Count, Spermatozoa, Sperm Motility, Semen, Testis, Infertility, Male, Spermatogenesis, Testosterone, Fertility
5 Brain neoplasms	Meningioma, Glioma, Meningeal Neoplasms, Neuroma, Acoustic, Brain Neoplasms, Glioblastoma, Neoplasms, Radiation-Induced, Neuroma, Cranial Nerve Neoplasms, Parotid Neoplasms, Central Nervous System Neoplasms
6 Sensory disorders	Burning Mouth Syndrome, Taste Disorders, Skin Diseases, Mouth Diseases, Dizziness, Vision Disorders, Hypersensitivity, Delayed, Fatigue
7 Breast neoplasms	Carcinoma, Lobular, Carcinoma, Ductal, Breast, Breast Neoplasms, Male, Adenoma
8 Oxidative stress	Oxidative Stress, Malondialdehyde, Glutathione Peroxidase, Lipid Peroxidation, Reactive Oxygen Species, Apoptosis, DNA Damage, Nitric Oxide, Protein Carbonylation
9 Neurodegenerative diseases	Parkinson Disease, Neurodegenerative Diseases, Alzheimer Disease, Amyotrophic Lateral Sclerosis, Motor Neuron Disease, Occupational Diseases, Dementia, Brain Diseases, Dementia, Vascular
10 Cerebrovascular disorders	Cerebrovascular Disorders, Dementia, Migraine Disorders, Tinnitus, Headache, Sleep Wake Disorders, Carotid Artery Diseases, Alzheimer Disease, Dementia, Vascular

11 Congenital abnormalities and glandular-based tumors	Cleft Lip, Cleft Palate, Fibroadenoma, Adenoma, Calcification, Physiologic, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma
12 Skin neoplasms	Carcinoma, Basal Cell, Carcinoma, Squamous Cell, Skin Neoplasms, Cocarcinogenesis, Neoplasms, Experimental, Neoplasms, Radiation-Induced, Colonic Neoplasms
13 Leukemia	Leukemia, Myeloid, Acute, Leukemia, Lymphocytic, Chronic, B-Cell, Leukemia, Myelogenous, Chronic, BCR-ABL Positive, Leukemia, Myeloid, Leukemia, Multiple Myeloma, Lymphoma, Leukemia, Radiation-Induced, Acute Disease, Liver Neoplasms, Experimental, Central Nervous System Neoplasms
14 Precancerous conditions	Atrophy, Precancerous Conditions, Hyperplasia, Hypersensitivity, Delayed, Thymus Gland, Capillary Permeability, Lymphoma
15 Circadian Rhythm	Melatonin, Circadian Rhythm, Pineal Gland
16 Eye diseases	Eye Diseases, Cataract, Vision Disorders, Sensation Disorders, Neurotic Disorders, Lens, Crystalline, Corneal Diseases, Edema, Hematologic Diseases
17 Electromagnetic interference in implanted electronic devices	Tachycardia, Ventricular, Ventricular Fibrillation, Death, Sudden, Cardiac, Arrhythmias, Cardiac
18 Liver Neoplasms	Liver Neoplasms, Carcinoma, Hepatocellular, Neoplasm Recurrence, Local, Lymphatic Metastasis
19 Symptoms of discomfort	Headache, Dizziness, Fatigue, Depression, Anxiety, Tremor, Sleep Wake Disorders, Neurotic Disorders, Stress, Psychological, Anxiety Disorders, Nervous System Diseases
20 Neoplasms	Lung Neoplasms, Ovarian Neoplasms, Pituitary Neoplasms, Lymphoma, Prostatic Neoplasms, Colonic Neoplasms, Carcinoma, Breast Neoplasms, Hematologic Neoplasms, Neoplasms, Liver Neoplasms, Cell Transformation, Neoplastic, Nervous System Neoplasms

2D3d. Text clustering taxonomy results

The 5,311 records in the retrieved and ***partially*** filtered adverse health effects database were imported into the CLUTO software [CLUTO, 2019], and a text clustering was performed. Forty-eight lowest level clusters were selected, based on theme resolution desired (average ~100 records per lowest level category). Appendix 4 presents this taxonomy (Table A4-1, Table A4-2), and the titles of the records that were assigned to each lowest-level category in the taxonomy. The titles give a better appreciation for the contents of each category than the brief category theme shown.

Table A4-1 (reproduced from the Appendix) presents the high-level clusters in the taxonomy, and the cluster themes. In Appendix 4, the fourth-level clusters in Table A4-2 (repeated from the fourth level shown in Table A4-1) are hyperlinked to their respective record titles.

Table A4-1 - CLUTO-Based Text Clustering Taxonomy – Top Levels

SECOND LEVEL	FOURTH LEVEL
Cluster 92 (2561) – Adverse effects of wireless radiation at cellular level, including radiation absorption at different frequencies	Cluster 78 (912) - Adverse impacts of wireless radiation, especially on cataracts, cells, and cognitive functions
	Cluster 79 (428) - Microwave radiation absorption at different frequencies
	Cluster 82 (529) - Adverse effects of mobile phone radiation, especially oxidative stress
	Cluster 84 (692) - Genotoxic effects of radiofrequency radiation
Cluster 93 (2750) – Adverse health effects of EMF on humans, especially cancer and neurodegenerative diseases, and on implanted electronic devices	Cluster 81 (673) - Adverse impacts of power-line EMF
	Cluster 85 (540) - Adverse impacts of low-frequency EMF, emphasizing cancer and neurodegenerative diseases
	Cluster 83 (668) – Adverse effects of mobile phone use, especially brain tumors, and brain and neural function
	Cluster 89 (869) - Human health risks from electromagnetic radiation, including adverse effects on implanted electronic devices, and possible protections

Note: Numbers in parentheses reflect numbers of records in cluster

2D3e. Wireless radiation adverse health effects in closed literatures

It should be re-emphasized at this point that almost all of the wireless radiation findings reported above reflect what is published in the open literature. That tends to emphasize basic research, and tends to be produced by academia, with its strong incentives for publication.

There's a much larger world of effort centered around wireless radiation technology and engineering development (for surveillance, communications, and weaponry) performed in organizations that have 1) few incentives to publish and 2) many prohibitions against publication due to classification and/or proprietary issues. Publication of adverse effects of these wireless systems could have severe financial consequences for all the stakeholders involved, and could result in potential military operational constraints as well.

The Federal government and industry who sponsor and many times conduct these advanced wireless radiation technology studies and demonstrations have 1) strong incentives to classify and proprietarize any results detrimental to their promotional activities and 2) no incentives to release results showing serious adverse health effects from wireless radiation to the public!

Consider the example shown in section 2D4 concerning the Zalyubovskaya [1977] reference, derived from Kostoff [2019a]. It shows some 1970s Soviet studies on EMF effects, including millimeter-wave effects, that were classified for 35 years until declassification in 2012. If relatively benign studies like those were classified for 35 years, one can only imagine the more serious studies that remain classified until this day. Or, Soviet studies that were not presented in an open forum because of their sensitivity. Or, USA studies that were performed decades ago (or recently), and remain classified to this day.

Also, consider the following example, which came to light relatively recently.

On 30 October 2019, an article was published suggesting the presence of cancer clusters among military pilots [https://www.mcclatchydc.com/news/nation-world/national/national-security/article236413708.html]. This may be the tip of the iceberg, since there are latency periods preceding the emergence of these cancers. It is unclear how well the health conditions of these pilots are tracked once they leave the service (according to the article), or, more specifically, ***how well the public is informed*** as to how well the health conditions of these pilots are tracked once they leave the service, and, if they are tracked, what the results of this tracking are. If there is tracking, who is funding the tracking, and what is its objectivity?

Severe recruiting consequences would result if it were shown that these serious diseases are in fact associated with exposures to on-board avionics and other stressors unique to the aircraft environment (EMF in combinations with other unique stressors [chemicals, psychological stress, high and low-G forces, etc] that performance aircraft crews face). It would be valuable to get EMF exposure data (***using an independent assessment***) under myriad flight conditions for many different military aircraft, with all the onboard avionics in full operation.

A similar article generated by the same organization addressing RF exposures of military pilots [https://www.mcclatchydc.com/news/nation-world/national/national-security/article237797304.html] complements the information contained in the above example, as shown in the following:

The largest Grumman measurement reported in the article translates to ***300 million microwatts/square meter***! This is thirty times today's FCC general public exposure limit, which itself is three-four orders-of-magnitude above levels shown by the cell tower studies to increase cancer incidence substantially. In parallel, the pilots are also being exposed to myriad other toxic stimuli, including EMF of other frequencies, cosmic radiation, perhaps fuel odors, etc, increasing the possibility of adverse effect synergies.

These may be the tip of the iceberg of RF exposure measurements done in the aircraft cabin, and there is no evidence that these were the highest occurring exposures. These types of exposure measurements rarely, if ever, see the light of day in the open literature, and are not advertised (for obvious purposes) by government-industry.

Additionally, while the gold coating mentioned may have kept a substantial amount of external RF from entering the cabin, it also would have delayed RF (that was internally generated or entered the cabin through non-gold coated non-metallic avenues) from leaving the cockpit, mirroring a hohlraum effect.

This cockpit problem reflects a disturbing trend. The military services became network-centric decades ago. They are almost completely dependent on wireless communications and wireless detection/surveillance for all their operations. If they were to allow their labs and contractors to report the possible damage from the levels of exposures happening in the field and at their facilities, potentially resulting in much lower wireless radiation exposure limits, they would be forced to eliminate many decades of so-called advances in their weaponry and operations. It could also impact their recruitment efforts adversely. No different in kind from their civilian counterparts, although the military may be operating at higher exposure levels because of their ultra-high-performance requirements.

So, while the adverse health effects of wireless radiation listed above in the monograph are very serious in their own right, they may be just the tip of the iceberg of the totality of adverse health effects that have actually been demonstrated if the non-published or classified studies had been taken into account.

2D4. Adverse Wireless Radiation Health Effects from Former USSR Literature Analysis

2D4a. Overview

The Former Soviet Union/USSR was a major player in biomedical research on health effects of non-ionizing radiation (both adverse and therapeutic) since at least the 1950s, and perhaps well before. Some/much of the work was published in the Soviet open literature, and available in Russian. Some/much of it was translated by USA intelligence agencies, and later declassified. Some may still be classified. The major difference between the USA and Soviet research on adverse effects of wireless radiation appears to be emphasis on thermal (USA) vs athermal (Soviet) effects. This difference is reflected in the different wireless radiation exposure limits imposed by each government.

2D4b. Glaser and Dodge review of East European radiofrequency literature

Glaser and Dodge addressed this issue within a comprehensive review of East European radiofrequency and microwave radiation literature [Glaser and Dodge, 1976], as follows:

THERMAL VS ATHERMAL EFFECTS – USA-USSR

"The most significant difference between East and West relative to biological mechanisms of effects of microwaves concerns the question of thermogenic versus nonthermogenic (or athermal) effects…..The traditional Soviet and East European view from the earliest publications of bio-studies has been that microwave and radio frequency fields can functionally, and even morphologically in some cases, alter the organism at field flux or power densities below those which cause measureable heating in tissues or biological substrates. Thus, reversible changes in behavior, physiological function, and microstructures are frequently reported at power densities of microwatts per square centimeter (muW/cm2), well below the Western world's "safe" exposure level of 10 milliwatts per square centimeter (10 mW/cm2)…..In contrast, the prevailing Western view, particularly in the United States, is that the effects of microwave and radio frequency fields are attributable only to the heating mechanism of those fields which are generally encountered at power densities in excess of 10 mW/cm2…..

The disparity between Eastern and Western views in this respect finds its most eloquent expression in daily occupational exposure standards for microwaves. In the Soviet Union and some East European countries, the standard for an occupational exposure day is 0.01 mW/cm2…..In the United States and some Western European countries, the value for continuous exposure is 10 mW/cm2.

Prior to 1953, it was believed that 100 mW/cm2 was the lowest level at which significant biological damage would occur…..Thus, 10 mW/cm2 is approximately one tenth the level calculated to cause significant heating in human tissues, and agrees with physiologic and metabolIc calculations . Intermediate standards between these values are practiced by some European countries….."

This conclusion, presented 43 years ago in print, is particularly disheartening. Despite all the evidence of adverse athermal effects of wireless radiation that was generated prior to 1976 (especially in the USSR, but in the USA as well), and the voluminous evidence (of adverse athermal effects of wireless radiation) that has been reported from global research since 1976, the USA government (along with many others) has refused to recognize the credibility of these athermal wireless radiation effects in the setting of regulatory exposure standards.

2D4c. Glaser review of global radiofrequency literature circa 1972

What was the state of the open literature on adverse health effects of wireless radiation in the 1970s, including what was known about Soviet and East European research? One partial answer can be gleaned from a very comprehensive review of the global radiofrequency and microwave biomedical effects literature published as a DTIC report in 1972 [Glaser, 1972]. The abstract of this report states in part:

> "More than 2300 references on the biological responses to radio frequency and microwave radiation, published up to April 1972, are included in this bibliography of the world literature. Particular attention has been paid to the effects on man on non-ionizing radiation at these frequencies. The citations are arranged alphabetically by author, and contain as much information as possible so as to assure effective retrieval of the original documents. ***Soviet and East European literature is included in detail***. An outline of the effects which have been attributed to radio frequency and microwave radiation is included as Chapter 1."

The effects mentioned in the last sentence have been converted to a more readable form by Dr. Magda Havas on her outstanding Web site (describing decades of global research on wireless radiation health effects) [Havas, 2019]. As stated on her Web site, Dr. Havas has obtained hard copies of Dr. Glaser's references from Dr. Glaser, and is in the process of scanning them and making them available to a wider audience. Dr. Havas' summary of the effects mentioned in the last sentence of the box above is repeated in the following table:

CATEGORY	ADVERSE EFFECTS
A. Heating of Organs* [Applications: Diathermy, Electrosurgery, Electrocoagulation, Electrodesiccation, Electrotomy]	This includes heating of the whole body or part of the body like the skin, bone and bone marrow, lens of the eye with cataracts and damage to the cornea; genitalia causing tubular degeneration of testicles; brains and sinuses; metal implants causing burns near hip pins etc. These effects are reversible except for damage to the eye.

B. Changes in Physiologic Function	This includes contraction of striated muscles; altered diameter of blood vessels (increased vascular elasticity), dilation; changes in oxidative processes in tissues and organs; liver enlargement; altered sensitivity to drugs; decreased spermatogenesis leading to decreased fertility and to sterility; altered sex ratio of births in favor of girls; altered menstrual activity; altered fetal development; decreased lactation in nursing mothers; reduction in diuresis resulting in sodium excretion via urine output; altered renal function; changes in conditioned reflexes; decreased electrical resistance of skin; changes in the structure of skin receptors; altered rate of blood flow; altered biocurrents in cerebral cortex in animals; changes in the rate of clearance of tagged ions from tissues; reversible structural changes in the cerebral cortex and diencephalon; changes in electrocardiographs; altered sensitivity to light, sound, and olfactory stimuli; functional and pathological changes in the eyes; myocardial necrosis; hemorrhage in lungs, liver, gut and brain and generalized degeneration of body tissue at fatal levels of radiation; loss of anatomical parts; death; dehydration; altered rate of tissue calcification.
C. Central Nervous System Effects	This includes headaches; insomnia; restlessness (daytime and during sleep); changes in brain wave activity (EEG); cranial nerve disorders; pyramidal tract lesions; disorders of conditioned reflexes; vagomimetic and sympathomimetic action of the heart; seizure and convulsions.
D. Autonomic Nervous System Effects	Altered heart rhythm; fatigue, structural alterations in synapses of the vagus nerve; stimulation of the parasympathetic nervous system leading to Bradycardia and inhibition of the sympathetic nervous system.
E. Peripheral Nervous System Effects	Effects on locomotor nerves.
F. Psychological Disorders	Symptoms include neurasthenia (general bad feeling); depression; impotence; anxiety; lack of concentration; hypochondria; dizziness; hallucinations; sleepiness or insomnia; irritability; decreased appetite; loss of memory; scalp sensations; fatigue; chest pain, tremors.
G. Behavioral Changes in Animals Studies	Effects include changes in reflexive, operant, avoidance and discrimination behaviors
H. Blood Disorders	Effects include changes in blood and bone marrow; increased phagocytic and bactericidal functions; increased rate of hemolysis (shorter lifespan of cells); increased blood sedimentation rate;

	decreased erythrocytes; increased blood glucose concentrations; altered blood histamine content; changes in lipids and cholesterol; changes in Gamma Globulin and total protein concentration; changes in number of eosinophils; decrease in albumin/globulin ratio; altered hemopoiesis (rate of blood corpuscles formation); leukopenia (increased number of white blood cells and leukocytosis; reticulocytosis (increase in immature red blood cells).
I. Vascular Disorders	This includes thrombosis and hypertension.
J. Enzyme and Other Biochemical Changes (in vitro)	Changes in the activity of cholinesterase (also in vivo); phosphatase; transaminase; amylase, carboxydismutase; denaturation of proteins; inactivation of fungi, viruses, and bacteria; killed tissue cultures; alterated rate of cell division; increased concentration of RNA in lymphocytes and decreased concentration of RNA in brain, liver and spleen; changes in pyruvic acid, lactic acid and creatinine excretions; changes in concentration of glycogen in liver (hyperglycemia); altered concentrationsof 17-ketosteroids in urine.
K. Metabolic Disorders	Effects include glycosuria (sugar in urne); increase in urinary phenols; altered processing of metabolic enzymes; altered carbohydrate metabolism.
L. Gastro-Intestinal Disorders	Effects include anorexia; epigastric pan; constipation; altered secretion of stomach digestive juices.
M. Endocrine Gland Changes	Effects include altered functioning of pituitary gland, thyroid gland (hyper-thyroidism and enlarged thyroid, increased uptake of radioactive iodine), and adrenal cortex; decreased corticosteroids in blood; decreased glucocorticoidal activity; hypogonadism (with decreased production of testosterone).
N. Histological Changes	Changes in tubular epithelium of testicles and gross changes.
O. Genetic and Chromosomal Changes	Effects include chromosomal aberrations (shortening, pseudochiasm, diploid structures, amitotic divisions, bridging, “stickiness”; irregularities in chromosomal envelope); mutations; mongolism; somatic alterations (not involving nucleus or chromosomes); neoplastic diseases (tumors).
P. Pearl Chain Effect	This refers to intracellular orientation of subcellular particles and orientation of cellular and other (non-biologic particles, i.e. mini magnetics) affecting orientation of animals, birds, and fish in electromagnetic fields.
Q. Miscellaneous Effects	These include sparking between dental fillings; metallic taste in mouth; changes in optical activity of colloidal solutions; treatment for

	syphilis, poliomyelitis, skin diseases; loss and brittleness of hair; sensations of buzzing, vibrations, pulsations, and tickling about head and ears; copious perspiration, salivation, and protrusion of tongue; changes in the operation of implanted cardiac pacemakers; changes in circadian rhythms.

Thus, much was known about the adverse health effects of both thermal and athermal high-frequency wireless radiation even in the early 1970s (Glaser's review did not address lower frequency radiation effects, although we now know these lower frequency effects could be equally damaging as those from high frequency), but this long-standing knowledge has not translated into adequate protections for the public from wireless radiation, both in the USA and the rest of the world.

2D4d. Joint Publications Research Service translations of East European research

Another avenue of insight into Soviet and East European research in the 1970s era was provided by the Joint Publications Research Service (JPRS). A description of this organization follows [https://guides.library.harvard.edu/jprs]:

> The United States Joint Publications Research Service is a government agency which translates foreign language books, newspapers, journals, unclassified foreign documents and research reports. Approximately 80% of the documents translated are serial publications. JPRS is the largest single producer of English language translations in the world. More than 80,000 reports have been issued since 1957, and currently JPRS produces over 300,000 pages of translations per year.
>
> **In its early years JPRS concentrated heavily on scientific and technical material from communist countries. Gradually coverage has broadened to include more non-scientific materials.**

2D4d1. Maritime occupational radiofrequency exposures in USSR

One of the Soviet technical books translated by the JPRS is listed on Dr. Havas' Web site [https://magdahavas.com/pick-of-the-week-15-russian-translations-on-biological-effects-of-magnetic-fields-and-radio-frequency-radiation/]. This book [Kulikovskaya, 1970] is important because it shows the levels of wireless radiation to which Soviets in some occupations were exposed fifty years ago, numbers that many wireless radiation proponent countries do not readily advertise. Whether these exposures are greater or less today is unclear; powers may be higher, but shielding may be better.

In the introductory section of Chapter IV (Biological Effect of Radio Waves – p.70), the following statement is made:

"Foreign researchers are giving basic attention to the effect of electromagnetic radio waves beginning with the thermal effect, that is, heating the animate organism by the field energy.

The research performed in our country, in contrast to foreign research, is based on a complex of dynamic studies of the reactions of the organism to the effect of low irradiation intensities, and, especially, in the superhighfrequency range, recognition of the cumulative biological effect in the case of chronic explosure to low power flux densities."

This quoted statement confirms the statement of Glaser and Dodge in section 2D4b above. Since the bulk of the references in Kulikovskaya's book are from the 1950s and 1960s, one can surmise that *a decision was made by the Western powers (especially the USA, who led the Western powers at that time) seventy years ago to downplay the adverse effects of athermal wireless radiation, and promote the false concept that only the thermal effects of wireless radiation are responsible for biomedical damage*. The decision-makers from the Western powers recognized seventy years ago that *wide-ranging wireless communications and surveillance were not possible if biologically protective exposure limits were promulgated*. Through countless Administrations and Legislatures since the days of President Eisenhower, all USA (and most foreign) decision-makers have presented a consistent and unified front promoting increased exposure to wireless radiation at the expense of the health of the nation's citizens!

The following table shows examples (from Kulikovskaya [1970]) of maximum levels of exposure to wireless radiation for Soviet citizens working in the marine environment. The maximum electric field exposure levels exceed the Soviet regulatory limits at that time (which were up to an order-of-magnitude lower than the USA regulatory limits) by up to two orders-of-magnitude!

To place these numbers in perspective, the Building Biologists' recommendations for safe long-term exposure limits in these frequency ranges is less than one volt per meter (https://mdsafetech.org/conversion-and-exposure-limits-emr-emf/). Thus, the reported exposures exceed safe levels by two-three orders of magnitude.

The research was performed at the Laboratory of Physical Factors of the State Scientific Research Insitute of Labor Hygiene and Professional Diseases. The exposure levels reported are what the Soviet government was willing to release to the public. Whether they were the most severe exposures experienced by members of the civilian and military fleets remains unknown. In terms of personnel recruitment for these jobs, it was/is not in the government's (Soviet or otherwise, including USA) best interests to release to the public exposure levels that would show these jobs to be highly dangerous to health. The book attempts to make the point that most exposures experienced by maritime personnel are much lower than the maximum, probably to assuage the public. The results are disturbing nevertheless, and should be viewed as the 'floor' of exposures to be expected relative to measurements made by an 1) ***independent objective group*** 2) ***on location during operations*** 3) ***without having given advanced notice***!

REGION	FREQ RANGE MHz	MAX EXPOS V/m	EXP LIMIT V/m
Electromagnetic Fields Near Tube Generators for High-Frequency Heating of Metals (P.23)	.06-.8	1,000+	20
Electromagnetic Waves Near Tube Generators for High-Frequency Heating of Dielectrics (P.26)	10-30	500+	20
Electromagnetic Fields in the Radio Rooms of Ships (P. 29)	.3-23		
Passenger Ships (P. 32)	.4-.8	2,000	20
Ships of the Tanker Fleet (P. 36)	.4-.8	2,000	20
Dry Cargo Ships (P. 37)	.4-.8	1,600	20
Ships of the Auxiliary Fleet (P. 40)	.4-.8	420	20
Electromagnetic Fields of Radio Communications Antennas on the Decks and Superstructures of Ships (P. 44)	.3-3	880	20
"In conclusion, it can be stated that the highest intensity of an electric field up to hundreds and sometimes ***thousands and more volts per meter*** occurs near the antenna drops and metal masses on the top bridges and decks during operation of a medium wave radio. Here, the magnetic component of the field can reach ***ten and even fifteen amps/meter***." P. 52)			
Superhigh Frequency Electromagnetic Fields of Radar Antennas on the Decks of Ships (P. 52)	3,000-15,000		
"Studies of the conditions of irradiation of the deck crew with superhigh-frequency fields performed on ships for various purposes show that when the radar antennas are installed on co1umns 1.2-2.5 meters above the deck of the top bridge, the power flux density can be hundreds and sometimes ***thousands of microwatts per square centimeter***." (P. 54)			
Some Adverse Health Effects of Marine Radio Operators (P. 80) "The conditions of labor of marine radio operators are least favorable….. a relatively large number of people with various diseases appear among radio operators. Thus, out of 215 radio operators, 50 had chronic diseases (23.2 percent)…..The primary disruption of the state of health of ship radio operators is damage to the organs of sight…..Among the diseases of the cardiovascular system occurring in ship radio operators, hypertonic disease. myocardial distrophy and disruption of the blood circulation in the brain play the leading role. All radio operators suffering from diseases of the cardiovascular system are young (from 30 to 35 years old) with five to 10 years of service. Among the diseases of the nervous system encountered in them, functional disorders of the central nervous system, vegetative neurosis, and neurasthenic syndrome are noted…..Thus, it is possible to consider it established that the largest number of people with health impairments occur among ship radio operators as compared to other marine professions."			

2D4d2. Biomedical effects of millimeter-wave exposures in some USSR research

Additionally, consider the following USSR reference [Zalyubovskaya, 1977] translated by the JPRS and published as a classified document in 1977.

SYSTEMIC ADVERSE EFFECTS FROM MILLIMETER-WAVE RADIATION

This is one of many translations of articles produced in the Former Soviet Union on wireless radiation (also, see reviews of Soviet research on this topic by McRee [1979, 1980], Glazer and Dodge [1976], Kositsky et al [2001]). On p. 57 of the pdf link, the article by Zalyubovskaya addresses biological effects of millimeter radiowaves. Zalyubovskaya ran experiments using power fluxes of 10,000,000 microwatts/square meter (the FCC guideline limit for the general public today), and frequencies on the order of 60 GHz. Not only was skin impacted adversely, but also heart, liver, kidney, spleen tissue as well, and blood and bone marrow properties. These results reinforce the conclusion of Russell (see section 2E) that systemic results may occur from millimeter-wave radiation. And, to re-emphasize, for Zalyubovskaya's experiments, the incoming signal was unmodulated carrier frequency only, and the experiment was single stressor only. Thus, the expected real-world results (when human beings are impacted, the signals are pulsed and modulated, and there is exposure to many toxic stimuli) would be far more serious and would be initiated at lower (perhaps much lower) power fluxes.

The Zalyubovskaya paper was published in 1977. What national security concerns caused it (and the other papers in the linked pdf reference) to be classified in the first place, and then kept classified for 35 years until declassification in 2012? What other papers on this topic with similar findings were published in the USSR (and the USA) at that time, or even earlier, and how many such papers never saw the light of day in the USSR (and the USA) at that time? It appears that we have known about the potentially damaging effects of millimeter-wave radiation on the skin (and other major systems in the body) for well over forty years, yet the discourse today only revolves around the possibility of modest potential effects on the skin and perhaps cataracts from millimeter-wave radiation.

2D4d3. Health effects from millimeter-wave exposures in Russian and Ukrainian literature

The review by Kositsky referenced in section 2D4d2 [Kositsky et al, 2001] appears to be based on 1) open literature publication of 2) wireless radiation biological effects 3) by Russian and Ukrainian researchers, covering the publication time period of 1968-2000. It appears to be quite comprehensive, and addresses both wireless radiation 1) adverse health effects and 2) therapeutics. It covers millimeter-wave frequencies almost exclusively. Some important takeaways from the Kositsky review are shown in the following box.

BIOLOGICAL EFFECTS FROM MILLIMETER-WAVE RADIATION

"there is a large probability of harmful effects from incidental generalized exposure, as confirmed in experiments on animals"

"Since living organisms have evolved under conditions of low natural background EHF EMR, they lack a ready-made mechanism of evolutionary adaptation to heightened levels of radiation resulting from technogenic factors"

"The results of clinical research showed that prolonged contact with EMF in the SHF band can lead to development of diseases, the clinical profile of which is determined above all by changes in the functional condition of the nervous and cardiovascular systems"

"Under EFD of 60 μW/cm2, disturbance of female cycles; reduction in fertility, number and weight of offspring; increase in postnatal deaths of the rat pups by a factor of 2.5; and dystrophic changes in the reproductive organs of the animals were noted"

"The results obtained give evidence that a single exposure to low-intensity EHF EMR without modulation, and with modulation at low frequencies of 5-10 Hz, induce opposite effects in red bone marrow (RBM). In the former case, we have pronounced stimulation of proliferative processes in the RBM, which are reversible. In the latter case—progressive depression of the process of blood production, right down to the formation of hypo- and aplastic conditions in the RBM on the sixth day of observation."

"biological effects of millimeter waves (BEF MMW):…They do not depend on the intensity of EMR, starting from the threshold to noticeable heating of tissue…..Irreversible BEF occur only during prolonged or cyclical exposure…..During amplitude or frequency modulation of MMW, bioeffects are maintained or strengthened as the power of exposure is significantly reduced…..The body "remembers" the effect of EMR for a relatively long time……In some cases, EMR influences sensitivity to other factors (chemicals, ionizing radiation, etc.), and the effects may persist through time."

"In epidemiological studies of the population of Ukraine, a connection was established between leukemia in children and cancer in adults, and exposure to EMF at industrial frequencies."

"Specific injuries under radiowave exposure are development of cataracts, instability in leukocyte make-up of peripheral blood, and vegeto-vascular disorder"

"the likelihood of cancer was three times greater under SHF exposure"

"It can be proposed that the current increase in electromagnetic pollution of the environment exceeds human adaptational capacities"

"The danger of mobile telephones consists of the fact that in addition to direct effects on the brain, the whole body is irradiated via the biologically active points of the concha of the ear"

"Observed higher resonance frequencies of a living cell coincide with frequencies of radiation of communications satellites. The power densities and duration of irradiation created by these satellites will significantly exceed…..the energetic doses inducing changes in living cells…… there will be a likelihood of changes (including negative changes) in the genetic apparatus of living cells during prolonged exposure to low-energy electromagnetic radiation from communications satellites"

"Combination with other deleterious factors: ionizing radiation, toxic substances, geomagnetic anomalies and stress significantly increase the effects of HF EMR."

"Occurrence of a narcotic-type dependency (by stimulating production of endorphins) is possible under regular irradiation with HF EMR."

"in animals irradiated with EMF, the nature of the infectious process changes—the course of the infectious process is aggravated"

"Absorption of EMF in biologically active points is many times more effective than in other parts of the skin, and this energy influences the internal organs and the body as a whole through the system of Chinese meridians."

In summary, these excerpts show that

- adverse effects can be initiated with very low doses of EMR,
- millimeter-wave radiation can impact regions below the skin, and
- adverse effects may be exacerbated when the EMR is combined with other toxic stimuli.

Given Kositsky's statement in section 2D4 about the potential of a narcotic-type dependency from exposure to EMR through stimulating production of endorphins, could EMR be effectively serving as one of the gateway 'drugs' to the increased opioid use we observe today? Appendix 5 addresses the potential impact of wireless radiation exposure on the opioid crisis, and shows that wireless radiation could indeed be a contributing factor to the overuse of opioids we are seeing today!

Particularly troubling are Kositsky's statements about the potential adverse effects of communications satellites. He bases his conclusions on the matching of communications satellites' frequencies with living cell resonances, as follows:

> "Observed higher resonance frequencies of a living cell coincide with frequencies of radiation of communications satellites. The power densities and duration of irradiation created by these satellites will significantly exceed (by ten or more orders of magnitude—such irradiation is possible over the course of a whole lifetime) the energetic doses inducing changes in living cells."

From some perspectives, the concept is counter-intuitive. Hormetic behavior of toxic substances and vaccines tends to be observed at extremely low doses of toxic stimuli. The average power fluxes from communications satellites are extremely low at the Earth's surface, and one would not expect adverse effects based on these low numbers. In the NTP experiments that many people cite as the wireless radiation experimental Gold Standard [Melnick, 2019], serious adverse effects were not observed until the power fluxes approached the FCC limit.

While his statements may seem counter-intuitive to some people, that does not mean they are incorrect. The issue needs to be resolved, sooner rather than later. At this time, 5G satellites are in fact being launched, and there are projections that tens of thousands of these satellites will eventually be launched to complete the global terrestrial and space 5G network. Launching of this number of satellites without the demonstrated evidence of safety would add to the unethical and harmful nature of the mobile networking experiment already observed.

2D4d4. "Confirmation" of Soviet microwave effects studies forty years later

The Soviet studies on adverse health effects from athermal radiofrequency exposures performed 40++ years ago showed clearly the dangers to human health from this toxic stimulus. Even though there was voluminous non-Soviet research showing a wide spectrum of adverse health effects from radiofrequency during that 40++ year period, some researchers undertook studies under 'similar' conditions to purportedly 'confirm' or validate the results from the Soviet studies [e.g., de Gannes et al, 2009; Repacholi et al, 2011; Grigoriev et al, 2010; Grigoriev, 2011]. This would require "validation" of health and safety research findings that were generated forty years ago in a ***completely different sponsorship and motivational context*** than has existed in the past decade. As one would expect, given the history of wireless radiation health and safety research, the results were mixed.

What type of independence and objectivity would one expect from 'confirmation' research sponsored by the promoters of 2G, 3G, 4G and now 5G mobile networking technology? Trillions of dollars in revenues are at stake in maintaining the fiction of wireless radiation safety under current exposure limit regulations. While the results could be correct, they should be interpreted with this context in mind.

2E. Potential Adverse Health Effects Expected from 5G Mobile Networking Technology

The potential 5G adverse health effects derive from the intrinsic nature of the radiation, and how this radiation interacts with tissue and other target structures. 4G networking technology was associated mainly with carrier frequencies in the range of ~1-2.5 GHz (cell phones, WiFi). The wavelength of 1 GHz radiation is 30 cm, and the penetration depth in human tissue is a few centimeters. The highest performance 5G networking technology is mainly associated with carrier frequencies at least an order of magnitude above the 4G frequencies, although, as stated previously, "ELFs (0–3000Hz) are always present in all telecommunication EMFs in the form of pulsing and modulation". Penetration depths for the high-performance carrier frequency component of 5G radiation will be on the order of a few millimeters.

For much of the early implementation of 5G, and perhaps later, 5G will be integrated with 4G. Some vendors will start out/have started out with 'low-band' 5G (~600-900 MHz); some will start out with 'mid-band' 5G (~2.5 GHz-4.2 GHz); and some will start out with 'high band' 5G (~24-47 GHz). All these modes are associated with potentially severe adverse health effects, and none have been tested for safety in any credible manner.

At the millimeter carrier wavelengths characteristic of high-band high performance 5G, one can expect resonance phenomena with small-scale human structures [Betzalel, 2018; Appendix 7B-3], as well as resonances with insects/insect components [Thielens et al, 2018].

The common 'wisdom' being presented in the literature and the broader media is that, if there are adverse impacts resulting from millimeter-wave 5G, the main impacts will be focused on near-surface phenomena, such as skin cancer, cataracts, and other skin conditions, because of shallow RF penetration depths. However, there is evidence that biological responses to millimeter-wave irradiation can be initiated within the skin, and the subsequent systemic signaling in the skin can result in physiological effects on the nervous system, heart, and immune system [Russell, 2018]. There is additional evidence that adverse effects from millimeter-wave radiation can occur in organs and tissue well below the skin surface (e.g., consider the example shown in section 2D4d2 in the box titled SYSTEMIC ADVERSE EFFECTS FROM MILLIMETER-WAVE RADIATION, or the example shown in section 2D4d3 in the box titled BIOLOGICAL EFFECTS FROM MILLIMETER-WAVE RADIATION) This should not be surprising, since there are myriad signaling conduits connecting the skin to deeper structures in the body.

2F. Why is there not Full Consensus on Adverse Effects from Wireless Radiation?

2F1. Reasons for Lack of Full Consensus

Not all studies of wireless radiation have shown adverse effects. There are many possibilities to explain this [Kostoff et al, 2020].

1) There could be 'windows' in parameter space where adverse effects occur, and the studies/experiments were conducted outside these 'windows'. Operation outside these windows could show i) no effects or ii) hormetic effects or iii) therapeutic effects.

For example, assume information content of the signal is a strong contributor to adverse health effects [Panagopoulus, 2019]. Experiments that involve only the carrier frequencies may be outside the 'window' where adverse health effects occur, and no adverse effects would be identified. Alternatively, in this specific example, the carrier signal and the information signal could be viewed as a combination of potentially toxic stimuli, where the adverse effects of each component are enabled because of the synergistic effects of the combination. If only one of the members of the combination were studied, again, adverse effects would not be identified.

As another example, an adverse health impact on one strain of rodent was shown for a combination of 50 Hz EMF and DMBA, while no adverse health impact was shown on another rodent strain for the same toxic stimuli combination [Fedrowitz et al, 2004]. From a higher-order combination perspective, if genetics are viewed conceptually as potentially equivalent to a toxic stimulus for combination purposes, then a synergistic three-constituent combination of 50 Hz EMF, DMBA, and genetics was required to elicit adverse health impacts in the above experiment. If these results can be extrapolated across species, then human beings could exhibit different responses to the same electromagnetic stimuli based on their genetic predispositions.

This particular experiment may be one of the most important conducted in wireless radiation toxicology. It shows that adverse effects from wireless radiation could depend on species/strain selection for the test subjects. This raises the question: which species or strain is most representative of human populations with respect to mirroring the adverse effects of wireless radiation. Is it rats; if so, is it Sprague-Dawley rats; if so, which strain of Sprague-Dawley rats? Or, are myriad strains of rats required to simulate effects on human populations with different genetic and other makeups? If not rats, is it dogs; if so, which species/strains of dogs. For setting regulatory exposure limits, should laboratory tests be conducted on a wide variety of species and strains, to determine which are the most representative of human responses to wireless radiation? Would the optimal species differ for different types of wireless radiation (e.g., high-frequency/low-frequency; high-power/low-power; pulsed/continuous, etc) and/or different types of other toxic stimuli?

The single stressor studies that constitute most of wireless radiation laboratory health research, and indeed constitute most of the laboratory medical research literature, essentially yield very narrow windows. Adverse effects are shown over very limited parameter ranges. As the above examples show, as well as the examples in Kostoff and Lau [2017] and Kostoff et al [2018], adverse effects shown by many combinations of stressors are not revealed when these stressors are tested in isolation over the same parametric ranges.

One could conclude that, whether by design or accident, ***the real-world impact of single stressor studies is to conceal, rather than reveal, many of the more serious adverse health effects of wireless radiation***.

The stressor variables to be used for health studies should not be limited to single stressors in isolation, but should include to the extent possible combinations of toxic stimuli stressors, since these combinations reflect more accurately real-life exposures.

2) Research quality could be poor, and adverse effects were overlooked.

3) Or, the research team could have had a preconceived agenda

where finding no adverse effects from wireless radiation was the main objective of the research.

2F2. The Role of Conflicts-of-Interest in Lack of Full Consensus

At this point, the reader would be well-advised to re-read section 2A1 on conflicts-of-interest relative to wireless radiation health and safety studies.

These conflicts pollute the well of knowledge relevant to health and safety, and are the ***largest contributor to mis-informing the public about the serious adverse health and safety impacts from wireless radiation***.

For example, studies have shown that industry-funded research of wireless radiation adverse health effects is far more likely to show no effects than funding from non-industry sources [Huss et al, 2007; Slesin, 2006; Carpenter, 2019]. Studies in disciplines other than wireless radiation have shown that, for products of high military, commercial, and political sensitivity, 'researchers'/organizations are hired to publish articles that conflict with the credible science (aka 'product defense' companies (https://www.fastcompany.com/1139299/manufacturing-doubt-product-defense), 'hired guns', etc), and therefore create doubt as to whether the product of interest is harmful [Michaels, 2008, 2020; Oreskes and Conway, 2011; Ong and Glantz, 2000; McGarity and Wagner, 2008; Walker, 2017].

Section 3.2.2 in a 2016 article on under-reporting of adverse effects of myriad substances in the biomedical literature [Kostoff, 2016] shows clearly the collusion of the USA government

and industry (and academia in some cases) in concealing harm of toxic substances (whose continued use is of importance to one or both organizations). These examples, and many others in the large USA government-industry candidate pool from which they were selected, show that

government-industry collusion to suppress adverse effects from technologies is endemic across technologies; ***it is not an aberration, but may be closer to the norm for technologies that are sensitive commercially, militarily, and politically.***

A comprehensive article in The New Yorker magazine (https://www.newyorker.com/magazine/2014/02/10/a-valuable-reputation?verso=true) details the travails that Prof. Tyrone Hays had to endure from industry in his quest to show that the herbicide Atrazine contributes to severe adverse effects. While the European Union banned the use of Atrazine almost two decades ago, the EPA has allowed its use to continue in the USA.

Finally, Appendix 6 lists study references showing effects of industry funding on research outcomes for myriad research disciplines (mainly within biomedical). What these references don't show (for the most part) is how industry convinced the regulators to incorporate the results of these studies in setting the lax regulations we see in practice today [e.g., Kostoff, 2018a]. Given that the sponsor and performer incentives of those studies are no different from the sponsor-performer incentives of wireless radiation health effects studies, there is little reason for expecting less concealment of adverse effects in the wireless radiation studies. Given the magnitude of revenues at stake for wireless radiation technology implementation, there is much reason for expecting more concealment and/or neutralization of adverse effects in the wireless radiation studies!

2F3. Interpreting Wireless Radiation Health Study Findings

Wireless radiation can play two roles as a contributor to adverse health effects: **initiator** and/or **promoter/accelerator**. The **initiator** role is reflected by single stressor studies (EMF alone) that show adverse health effects. The **promoter/accelerator** role is reflected by 1) combination studies that show no adverse effects from any of the constituents when tested in isolation, but show adverse effects (synergies) when tested in combination or 2) accelerating emergence of serious diseases. There can also be **initiator and promoter/accelerator** roles shown by combination studies, where each constituent tested in isolation shows a modest adverse effect, but the combination shows a much larger (i.e., synergistic) effect [Kostoff and Lau, 2013, 2017; Kostoff et al, 2018; Kostoff, 2018b].

So, if a study shows an adverse health effect from wireless radiation, and if it passes the criteria for high quality research, then that specific adverse effect for the parameter range shown could be accepted as credible. If a study shows no adverse health effects from wireless radiation in a single stressor experiment, the study MAY reflect no **initiator** role ***in the parameter window selected***, if the study is deemed to be of high research quality. However, such an experiment

offers little insight as to the **promoter/accelerator** role of the wireless radiation *in the parameter range selected*. The same would hold true for no adverse effects shown in combination experiments; there is no reason to believe that, even if wireless radiation serves as a promoter/accelerator for some combinations, it would therefore serve as a promoter/accelerator for all combinations.

In summary, the adverse effects of wireless radiation that result from credible high-quality studies published in the biomedical literature form the 'floor' for total adverse impacts of this wireless radiation. Given the insights of synergies from toxic stimuli combination studies evidenced in [Kostoff and Lau, 2013, 2017, Kostoff et al, 2018b, Juutiliin, 2006, 2008], many more adverse impacts from wireless radiation can be expected if the parameter range of single stressor studies is expanded and the numbers of combination studies are greatly expanded.

Further, there is little doubt that the biological effects of wireless radiation studies that have been classified (by the organization promoting the expansion of this technology, the Federal government, for alleged 'national security' purposes) show substantially more harmful effects from this technology in real-life situations.

Even the Gold Standard for research credibility – **independent replication of research results** – is questionable in politically, commercially, and militarily sensitive areas like wireless radiation safety. Suppose there are two research groups (funded by the same government agency) who both arrive at the same conclusion that just coincidentally coincides with what the government sponsor wanted. Would this be considered independent? Or, these two research groups received funding from different agencies of the same government. Would that be considered independent? Or, these two research groups received funding from two different governments that both had the same accelerated development objectives for the technology of interest. Review articles tend to treat these types of cases as independent, and don't make the distinction as long as the validation doesn't arise from within the performer group/organization.

Given the broad support exhibited today by the USA Federal government, military, and industry for the rapid implementation of 5G (and, indeed, the governments of most, if not all, the major developed countries globally), all these organizations must present a united front in declaring 5G (and previous generations of mobile networking technology) to be safe. If one government lab, or one highly-funded performer, were to perform a credible real-life simulation of wireless radiation effects and show the potential damage that might result, then the government's and industry's current fast-track effort to ***implement 5G before the full extent of the damage becomes known*** would be derailed.

It is unrealistic that any government would allow this to happen!

Even reporting of conflict-of-interest in wireless radiation research papers or evaluation panels leaves much to be desired. Currently, potential research performer conflicts of interest are identified by listing of funding sources in the published papers, or other formal documented evidence of conflicts of interest. However, there are many potential conflicts of interest that may not be as formal, but could be at least as influential as the formal conflicts in determining the outcome of the research or proposal. To ascertain these other less formal conflicts of interest would require vetting:

1) any elements of the researchers'/evaluators' investment portfolio that would profit from operation and expansion of the mobile telecommunications network, including impacts on related industries;

2) any elements of their present business endeavors that would profit from operation and expansion of this network, including impacts on related industries;

3) any elements of present or future pensions that would profit from operation and expansion of this network, including impacts on related industries;

4) any proposals or future employment offers in the pipeline or being considered that would profit from operation and expansion of this network, including impacts on related industries;

5) any other conflicts of interest by which they could profit from operation and expansion of the mobile telecommunications network, including impacts on related industries.

2G. Conclusions

Wireless radiation offers the promise of improved 1) remote sensing, 2) communications and data transfer, and 3) connectivity. Unfortunately, there is a large body of data from laboratory and epidemiological studies showing that previous generations of wireless networking technology have significant adverse health impacts. Much of this data was obtained under conditions not reflective of the real-world. When real-world considerations are added, such as 1) including the information content of signals along with the carrier frequencies, and 2) including other toxic stimuli in combination with the wireless radiation, ***the adverse effects are increased substantially***. Superimposing 5G mobile networking technology on an imbedded toxic wireless radiation environment (4G, 3G, etc) will exacerbate the myriad adverse health effects already shown to exist. Far more research and testing of potential 5G health effects is required before further rollout can be justified. Without this additional testing and demonstrated safety of potential 5G health effects, we will be even further along in **The Largest Unethical Medical Experiment in Human History!**

Chapter 3 - References

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Chapter 4 – Appendices

Appendix 1 – Unethical Medical Experiments

A1-A. Overview

The biomedical literature reflects much good research. However, the world today is also awash in unethical medical experiments. There are two major types. The first type is classical unethical medical experiments, where test subjects are explicitly/proactively selected for experiments on biological effects of drugs or potentially harmful substances, and participate in these experiments without having given 'informed consent'. The second type may be far more prevalent. Here, potentially harmful substances are introduced into commercial, military, or other government practice without adequate demonstration of safety. Then, test subjects are implicitly/reactively selected 'a posteriori' to participate in these de facto experiments, again without having given informed consent. These latter studies are usually epidemiological studies.

In parallel with the burgeoning conduct of unethical medical experiments is production of a literature that addresses the ethics of, and in many cases bemoans the prevalence and conduct of, these myriad unethical medical experiments. The experiments and the accompanying ethics literature form a symbiosis, where the literature feeds off the experiments, and the experiments spawn an additional literature. It is not clear how much, if any, impact the ethics literature has had/does have/will have on the conduct of the unethical medical experiments, especially those unethical medical experiments of the second type defined above.

Appendix 1A provides a few examples of mainly classical unethical medical experiments, and Appendix 1B provides a few references that reflect the medical experiment ethics literature.

Appendix 1A – Unethical Medical Experiments - Examples

This Sub-Appendix provides examples of unethical medical experiments, conducted mainly 1) over the last 100 years and 2) within the USA or under its auspices. The list is not exhaustive, since an abbreviated search approach was used, covering both Medline and the Web. Some of the more useful Web sources of information are shown in the following table:

https://en.wikipedia.org/wiki/Unethical_human_experimentation;
https://en.wikipedia.org/wiki/Human_subject_research;
https://en.wikipedia.org/wiki/Unethical_human_experimentation_in_the_United_States;
https://en.wikipedia.org/wiki/Medical_torture;
https://abuse.wikia.org/wiki/Unethical_human_experimentation_in_the_United_States;
https://www.amazon.com/s?k=human+experimentation&i=stripbooks&page=2&gclid=Cj0KCQiA89zvBRDoARIsAOIePbBy8acwX6tfMZcGkZyi_UTov1I7_PxcFYDAgDWiAgHVc7anOyx57slaAgtNEALw_wcB&hvadid=241915884190&hvdev=c&hvlocphy=9007578&hvnetw=g&hvpos=2o1&hvqmt=b&hvrand=12610529676369552 69&hvtargid=kwd-1053626641&hydadcr=22561_10346245&qid=1576539483&ref=sr_pg_2;
https://www.bibliotecapleyades.net/ciencia/ciencia_industryweapons173.htm.

It should be noted that information of this type is not easy to obtain. The research performers and their sponsors are not motivated to reveal such odious experiments to any oversight organizations, and therefore tend to conceal these experiments to the largest extent possible. There are three main routes by which this information eventually gets to the public: whistle-blowers; discovery in legal lawsuits; inadvertent access by researchers examining other topics. While we don't know the extent of these types of experiments that have not been reported, it is probably a good assumption that there are huge numbers.

Following are some of the books and journal/magazine articles that describe these experiments. It is by no means a complete list, and the interested reader would be well-advised to read the articles with the Web links provided in the box.

Examples of Unethical Medical Experiments

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Appendix 2 – Manual Taxonomy of Adverse EMF Health Effects Database

A2-A. Category Themes

A query to retrieve Medline records showing adverse health effects of wireless radiation was generated. The query was entered into the Medline search engine, and ~15,000 records were retrieved. Filtering was applied to the retrieval to remove records not associated with adverse health effects of wireless radiation, and 5311 records remained. Thousands of the highest frequency title and abstract phrases were read, the categories from the factor analysis and text clustering approaches of Appendices 3 and 4 were evaluated, and a manual taxonomy of main categories in the database was generated. The ~10,000 highest frequency abstract phrases were visually inspected, and assigned to the appropriate categories in the taxonomy.

The following table (A2-1) shows the categories in the taxonomy, and the phrases associated with each category. For each category, the records associated with the phrases identified were highlighted, and the titles of those records were extracted. Following the table, each category and representative record titles are shown in order to display the breadth of coverage of the category.

In the process of selecting the record titles to represent the category's theme, a second level of filtering was done (visual inspection). Strong emphasis was placed on 1) records associated with microwave frequencies; 2) exposures not exceeding FCC and ICNIRP-based limits; 3) records that clearly showed adverse effects. This meant that ***large numbers of records showing adverse health effects from especially i) power/ELF frequencies and ii) high microwave power exposures that had both thermal and athermal effects were not shown***.

> In the latter case (high microwave power exposures), where thermal effects exist, the assumption is usually made that any adverse effects shown are due to thermal effects. This may or may not be correct. Adverse effects could be due to thermal effects, they could be due to higher radiation intensity athermal effects, or they could be due to some (potentially synergistic) combination of thermal and athermal effects. In the record selection process, I used the conservative approach of not selecting records where the radiation flux was associated with increased temperatures.

The categories are not orthogonal; there is some overlap, especially among categories that cover different levels of detail (e.g., cancer-genotoxicity, reproduction-biomarkers, etc). Therefore, some representative record titles may appear in more than one category.

The major adverse effects are listed in the first column (Category), and the phrases associated with the theme are listed in the second column (Key Phrases). These adverse effects are self-explanatory. Each category in the taxonomy is hyper-linked to its respective record titles. To obtain the full record, insert title into Pubmed, or similar Medline search engine.

Table A2-1 – Manual Taxonomy

CATEGORY	KEY PHRASES
Cancer/Tumors	cancer, leukemia, cancers, carcinogenic, breast cancer, malignant, leukaemia, cancer risk, glioma, brain cancer, carcinogenesis, brain tumours, lymphoma, carcinogen, childhood cancer, childhood leukaemia, carcinoma, brain tumor, cancer incidence, carcinogenicity, lymphoblastic, acute lymphoblastic, melanoma, gliomas, neoplasms, acute lymphoblastic leukemia, breast cancer risk, carcinogens, lymphoblastic leukemia, neoplastic, glioblastoma, leukemia risk, malignancy, leukemias, malignancies, neuroblastoma, cancer risks, lung cancer, childhood cancers, lymphomas, astrocytoma, malignant brain, Acute leukemia, mammary gland, brain cancers, glioma risk, Malignant melanoma, malignant neoplasms, neoplasia, hyperplasia, myeloid leukemia, carcinomas, neuroblastoma cells, testicular cancer, leukaemias, neoplasm, mammary cancer, myeloma, nervous system cancers, adenocarcinoma, cocarcinogenic, colorectal, glioblastoma multiforme, Hodgkin's disease, multiple myeloma, non-Hodgkin's lymphoma, seminoma, breast carcinoma, colon cancer, glioma meningioma, larynx, neoplastic transformation, Non-Hodgkin lymphoma, tumor, tumors, tumours, brain tumors, tumour, neuroma, acoustic neuroma, meningioma, brain tumour, tumor risk, tumor growth, tumor incidence, mammary tumors, tumor promotion, intracranial tumors, tumor promoter, gland tumors, meningiomas, tumorigenesis, tumour risk, benign tumors, nervous system tumors, neuromas, acoustic neuromas, breast tumors, gland tumours, parotid gland tumors, tumor-promoting
Neurodegenerative	memory, cognitive, central nervous system, learning, neurodegenerative, Alzheimer's, learning and memory, Alzheimer's disease, cognition, amyotrophic lateral sclerosis, neurodegenerative diseases, cognitive function, cognitive functions, neurobehavioral, dementia, spatial learning, acetylcholine, Parkinson's disease, epilepsy, Glial fibrillary, motor activity, multiple sclerosis, cognitive impairment, spatial learning and memory, neurodegenerative disease, neuronal damage, Alzheimer disease, cognitive effects, seizure, seizures, autism, cognitive functioning, cognitive processing, memory function, memory impairment, memory loss, neurological diseases, neuronal excitability, cognitive dysfunction, memory deficit, memory functions, neurocognitive, neuronal degeneration, spatial working memory
Reproduction	pregnancy, reproductive, pregnant, sperm, embryos, testicular, fertility, embryo, testis, embryonic, fetuses, testosterone, motility, infertility, reproduction, testes, semen, spermatozoa,

	spermatogenesis, reproductive system, sperm motility, male fertility, sperm count, embryogenesis, abortion, male reproductive, spermatogenic, embryonic development, mating, male infertility, birth defects, serum testosterone, adverse reproductive, miscarriage, reproductive organs, semen parameters, sperm concentration, sperm parameters, testicular function, testosterone level, epididymis, male reproductive system, spermatogenic cells, spermatogonia, fertilized eggs, ovaries, reproductive capacity, reproductive outcomes, sperm cells, sperm morphology, fertile, pregnancies, reproductive function, testicular tissue, rat testes, rat testis, reproductive functions, reproductive systems, sperm DNA, spermatogonial, testis tissue, embryogeny, reproductive health, sperm cell, miscarriages, offsprings, oocyte, oogenesis, preterm birth, seminal vesicles, Sperm head, spermatids, sperms, testicles, fetal loss, genital, gonads, reproductive hormones, semen analysis
Genotoxicity	DNA damage, genotoxic, micronuclei, chromosomal, micronucleus, chromosome, genotoxicity, genotoxic effects, mutagenic, strand breaks, chromatin, mutation, DNA strand, Chromatid, mutations, chromosome aberrations, chromosomes, DNA fragmentation, double-strand, chromosomal aberrations, DNA repair, DNA strand breaks, micronucleus (MN), genetic damage, micronuclei (MN), Sister Chromatid, genome, blood leukocytes, double-strand breaks, oxidative DNA, chromosomal damage, DNA synthesis, mutant, cellular stress, chromosome aberration, oxidative DNA damage, Purkinje cells, DNA breaks, cell cycle arrest, clastogenic, genotoxic potential, keratinocytes, micronucleated, single strand, cell division, chromatid exchange, Chromatid Exchanges, genetic material, micronucleus test, Mutagenesis, cell cycle progression, cellular DNA, Cytochrome c, double strand, genetic effects, genomic instability, micronucleus frequency, DNA single-strand, DNA-damaging, Mutagen, mutagenicity, single strand breaks, chromatin condensation, chromosomal aberration, double-strand breaks (DSBs), strand breakage, cell cycle distribution, cell DNA, genetically, strand DNA
Cardiovascular	Cardiac, cardiovascular, pacemaker, pacemakers, implanted, blood pressure, implantable, vascular, heart rate variability, myocardial, heart rate variability (HRV), implants, cardiac pacemakers, implantation, defibrillators, implant, cardioverter, myocardium, cardiovascular system, implantable cardioverter, Cardiovascular disease, defibrillator, fibrillation, arrhythmia, arterial blood pressure, autonomic nervous system, cardioverter defibrillators, implanted pacemakers, cardiac pacemaker, hypertension, arrhythmias, cardioverter-defibrillators, implantable cardioverter defibrillators, implantable cardioverter-defibrillators, pacemaker function, heart disease, implanted cardiac, tachycardia, cardiac devices, circulatory system, microcirculation, blood vessels,

	cardiomyocytes, cardiovascular effects, vascular permeability, atherosclerosis, cardiovascular diseases, ventricular fibrillation, arterial pressure, Atrial fibrillation, cardiac output, cardiovascular function, Implantable cardioverter defibrillator (ICD), implantable devices, arrhythmic, carotid artery, pacemaker dysfunction, pacemaker malfunction
Immunity	lymphocytes, immune, lymphocyte, immune system, immunity, blood lymphocytes, leukocytes, antibodies, immune response, human lymphocytes, antibody, peripheral blood lymphocytes, immunological, leukocyte, neutrophils, lymphocytic, immune functions, immunoreactivity, autoimmune, immunization, monocytes, neutrophil, antigens, macrophage, immune parameters, immune responses, immunocompetent, natural killer cells, spleen lymphocytes, immunologic, immunoreactive, micronucleated cells, monoclonal antibodies, spleen cells, splenocytes, T lymphocytes, antibody production, antibody-forming, monoclonal antibody
Biomarkers	apoptosis, oxidative stress, Malondialdehyde, reactive oxygen species, apoptotic, superoxide dismutase, lipid peroxidation, permeability, catalase, MDA, ROS, ROS), reactive oxygen species (ROS), Malondialdehyde (MDA), SOD), cell death, glutathione peroxidase, inflammatory, erythrocytes, oxidative damage, SOD, caspase-3, free radical, nitric oxide, free radicals, biomarkers, bcl-2, catalase (CAT), inflammation, corticosterone, edema, glutathione peroxidase (GSH-Px), cytokine, cytokines, alkaline phosphatase, cell apoptosis, protein kinase, ATP, glutathione (GSH), oxidation, TNF-alpha, Bax, Ca2+, estrogen, ornithine decarboxylase, red blood cells, intracellular calcium, cell damage, apoptotic cell, hemoglobin, lactate dehydrogenase, cerebral blood flow, glutamate, hydrogen peroxide, IL-1beta, Purkinje, serotonin, apoptotic cell death, barrier permeability, carbonyl, hormone levels, ornithine decarboxylase (ODC), acetylcholinesterase, calcium ion, Calcium ions, endothelial cells, GABA, MDA levels, ODC, xanthine oxidase, creatinine, intracellular ROS, cholinesterase, lipid peroxidation levels, pro-inflammatory, protein kinase C, adrenocorticotropic hormone, alanine aminotransferase, aspartate aminotransferase, caspase 3, caspase-9, catalase activity, glutathione levels, NF-kappaB, atrophy, nitric oxide synthase, cAMP, acid phosphatase, adenosine deaminase, adrenocorticotropic hormone (ACTH), blood cell count, blood platelets, Ca++, adrenaline, C-reactive protein, oxidative damages, Reactive Oxygen Species), vascular endothelial growth factor
Sensory Disorders	auditory, acoustic, ear, hypersensitivity, EHS), EHS, electromagnetic hypersensitivity, otoacoustic, vestibular, hypersensitive, cataract, cochlea, auditory system, inner ear, lens epithelial, corneal, tinnitus, vision, lenses, otoacoustic emissions, hearing loss, otoacoustic emission, epidermis, rabbit lens,

	dermatitis, auditory stimuli, cataractogenic, Auditory brainstem response (ABR), auditory evoked, electrohypersensitive, electrosensitivity, vestibular system, cochlear implants, dermatological, hearing function, hearing thresholds, pain sensitivity, pain threshold, skin complaints
Discomfort Symptoms	depression, anxiety, headache, headaches, dizziness, depressed, depressive, vertigo, cataracts, behavioral effects, nausea, headache dizziness, low back pain, behavioural effects,
Congenital Abnormalities	malformations, teratogenic, congenital, congenital malformations, teratogenicity, malformation, teratogens, teratologic, cleft palate, congenital anomalies, malformed, teratological
Circadian Rhythym and Melatonin	melatonin, sleep, circadian, melatonin production, sleep disturbances, insomnia, melatonin levels, melatonin secretion, sleep disorders, sleep EEG, poor sleep, pineal function
Chronic Conditions	metabolism, metabolic, glucose, endocrine, cholesterol, Diabetes, calcium homeostasis, glucose levels, homeostatic, metabolic activity, metabolic heat production, Diabetes Mellitus, diabetic, glucose metabolism, obesity

All the records shown in this Appendix, and their relevant citing papers, were analyzed further for most frequent keywords relating to serious symptoms/disease. In order of frequency, they are: oxidative stress; Apoptosis; DNA damage; melatonin; Reactive oxygen species; glioma; Testis; cancer; liver; Malondialdehyde; Brain cancer; testosterone; Anxiety; Depression; Lipid peroxidation; ROS; Chromosomal aberrations; Learning and memory; oxidative damage; sperm; testes; Infertility; spermatogenesis; Breast cancer; Cell cycle; Genotoxicity; Kidney; Leukemia; Male infertility; micronuclei; Pregnancy; Sleep; sperm motility; acoustic neuroma; carcinogenesis; carcinogenicity; Cognitive function; fertility; Heart rate variability; Micronucleus; Reproduction; Spatial memory; Stress; Alzheimer's disease; astrocytoma; Autophagy; Cognition; Cytotoxicity; free radicals.

These match well with the prior results shown for this strongly filtered database.

A2-B. Category Record Titles

CANCER/TUMORS

Keywords – cancer, leukemia, cancers, carcinogenic, breast cancer, malignant, leukaemia, cancer risk, glioma, brain cancer, carcinogenesis, brain tumours, lymphoma, carcinogen, childhood cancer, childhood leukaemia, carcinoma, brain tumor, cancer incidence, carcinogenicity, lymphoblastic, acute lymphoblastic, melanoma, gliomas, neoplasms, acute lymphoblastic leukemia, breast cancer risk, carcinogens, lymphoblastic leukemia, neoplastic, glioblastoma, leukemia risk, malignancy, leukemias, malignancies, neuroblastoma, cancer risks, lung cancer, childhood cancers, lymphomas, astrocytoma, malignant brain, Acute leukemia, mammary gland, brain cancers, glioma risk, Malignant melanoma, malignant neoplasms, neoplasia, hyperplasia, myeloid leukemia, carcinomas, neuroblastoma cells, testicular cancer, leukaemias, neoplasm, mammary cancer, myeloma, nervous system cancers, adenocarcinoma, cocarcinogenic, colorectal, glioblastoma multiforme, Hodgkin's disease, multiple myeloma, non-Hodgkin's lymphoma, seminoma, breast carcinoma, colon cancer, glioma meningioma, larynx, neoplastic transformation, Non-Hodgkin lymphoma, tumor, tumors, tumours, brain tumors, tumour, neuroma, acoustic neuroma, meningioma, brain tumour, tumor risk, tumor growth, tumor incidence, mammary tumors, tumor promotion, intracranial tumors, tumor promoter, gland tumors, meningiomas, tumorigenesis, tumour risk, benign tumors, nervous system tumors, neuromas, acoustic neuromas, breast tumors, gland tumours, parotid gland tumors, tumor-promoting

Titles

2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic Ca(2)(+) influx in human leukemia cancer cells.

A case-case study of mobile phone use and acoustic neuroma risk in Japan.

A cluster of male breast cancer in office workers.

A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.

A new electromagnetic exposure metric: high frequency voltage transients associated with increased cancer incidence in teachers in a California school.

A population-based case-control study of radiofrequency exposure in relation to childhood neoplasm.

Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation.

Adult and childhood leukemia near a high-power radio station in Rome, Italy.

Association between exposure to pulsed electromagnetic fields and cancer in electric utility workers in Quebec, Canada, and France.

Association between number of cell phone contracts and brain tumor incidence in nineteen U.S. States.

Association between radiation from mobile phones and tumour risk in adults].

Association between vestibular schwannomas and mobile phone use.

Biological effects from electromagnetic field exposure and public exposure standards.

Brain cancer and occupational exposure to magnetic fields among men: results from a Canadian population-based case-control study.

Cancer in radar technicians exposed to radiofrequency/microwave radiation: sentinel episodes.

Cancer incidence and mortality and proximity to TV towers.

Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter.

Cancer incidence vs. FM radio transmitter density.

Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation.

Cancer versus FM radio polarization types.

Case-control study of the association between malignant brain tumours diagnosed between 2007 and 2009 and mobile and cordless phone use.

Case-control study on the use of cellular and cordless phones and the risk for malignant brain tumours.

Causes of death among Belgian professional military radar operators: a 37-year retrospective cohort study.

Cell phone radiation exposure on brain and associated biological systems.

Cell phone use and acoustic neuroma: the need for standardized questionnaires and access to industry data.

Cell phone use and risk of thyroid cancer: a population-based case-control study in Connecticut.

Cell phones and brain tumors: a review including the long-term epidemiologic data.

Cellular and cordless telephone use and the association with brain tumors in different age groups.

Cellular and cordless telephones and the risk for brain tumours.

Cellular neoplastic transformation induced by 916 MHz microwave radiation.

Cellular phone use and risk of benign and malignant parotid gland tumors--a nationwide case-control study.

Cellular telephones and their relay stations: a health risk?].

Commentary on the utility of the National Toxicology Program study on cell phone radiofrequency radiation data for assessing human health risks despite unfounded criticisms aimed at minimizing the findings of adverse health effects.

Connection between Cell Phone use, p53 Gene Expression in Different Zones of Glioblastoma Multiforme and Survival Prognoses.

Current Understanding of the Health Effects of Electromagnetic Fields.

Danger of cellular telephones and their relay stations].

Decreased survival for childhood leukemia in proximity to television towers.

Decreased survival of glioma patients with astrocytoma grade IV (glioblastoma multiforme) associated with long-term use of mobile and cordless phones.

Delayed biological effect of electromagnetic fields action].

Determining health policy for sensible mobile phone use--current world status].

Dirty electricity, chronic stress, neurotransmitters and disease.

Does cell phone use increase the chances of parotid gland tumor development? A systematic review and meta-analysis.

Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea.

Effect of cell-phone radiofrequency on angiogenesis and cell invasion in human head and neck cancer cells.

Effect of Exposure to 900 MHz GSM Mobile Phone Radiofrequency Radiation on Estrogen Receptor Methylation Status in Colon Cells of Male Sprague Dawley Rats.

Effect of Mobile Phone-Induced Electromagnetic Field on Brain Hemodynamics and Human Stem Cell Functioning: Possible Mechanistic Link to Cancer Risk and Early Diagnostic Value of Electronphotonic Imaging.

Effects of Mobile Phones on Children's and Adolescents' Health: A Commentary.

Effects of the microwave radiation from the cellular phones on humans and animals].

Electromagnetic field exposure and male breast cancer risk: a meta-analysis of 18 studies.

Electromagnetic field exposures and childhood cancers in New Zealand.

Electromagnetic field induced biological effects in humans.

Electromagnetic fields and cancer: the cost of doing nothing.

Enzymatic alterations in developing rat brain cells exposed to a low-intensity 16.5 GHz microwave radiation.

Epidemiologic evidence relevant to radar (microwave) effects.

Epidemiological evidence for a health risk from mobile phone base stations.

EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses.

Evaluation of genotoxic effects in male Wistar rats following microwave exposure.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Evaluation of Mobile Phone and Cordless Phone Use and Glioma Risk Using the Bradford Hill Viewpoints from 1965 on Association or Causation.

Evaluation of the cytogenotoxic damage in immature and mature rats exposed to 900 MHz radiofrequency electromagnetic fields.

Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure.

Evidence for microwave carcinogenesis in vitro.

Exposure to low-intensive superhigh frequency electromagnetic field as a factor of carcinogenesis in experimental animals.

Follow-up of radio and telegraph operators with exposure to electromagnetic fields and risk of breast cancer.

Further aspects on cellular and cordless telephones and brain tumours.

Genotoxic and carcinogenic effects of non-ionizing electromagnetic fields.

Human disease resulting from exposure to electromagnetic fields.

Incidence of cancer in the vicinity of Korean AM radio transmitters.

Incidence of Seminoma Cancer in Staffs that Worked in Electromagnetic Waves Station; Three Cases Report.

Increased incidence of cancer in a cohort of office workers exposed to strong magnetic fields.

Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies.

Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model.

Inferring the 1985-2014 impact of mobile phone use on selected brain cancer subtypes using Bayesian structural time series and synthetic controls.

Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations.

Leukemia mortality and incidence of infantile leukemia near the Vatican Radio Station of Rome].

Long-term exposure to microwave radiation provokes cancer growth: evidences from radars and mobile communication systems.

Long-term use of cellular phones and brain tumours: increased risk associated with use for > or =10 years.

Melanoma incidence and frequency modulation (FM) broadcasting.

Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells.

Meta-analysis of association between mobile phone use and glioma risk.

Meta-analysis of long-term mobile phone use and the association with brain tumours.

Microwaves from Mobile Phones Inhibit 53BP1 Focus Formation in Human Stem Cells More Strongly Than in Differentiated Cells: Possible Mechanistic Link to Cancer Risk.

Mitochondrial DNA damage and oxidative damage in HL-60 cells exposed to 900MHz radiofrequency fields.

Mobile phone radiation causes brain tumors and should be classified as a probable human carcinogen (2A) (review).

Mobile phone use and brain tumours in the CERENAT case-control study.

Mobile phone use and glioma risk: A systematic review and meta-analysis.

Mobile phone use and location of glioma: a case-case analysis.

Mobile phone use and risk for intracranial tumors and salivary gland tumors - A meta-analysis.

Mobile phone use and risk of brain tumours: a systematic review of association between study quality, source of funding, and research outcomes.

Mobile phone use and risk of tumors: a meta-analysis.

Mobile phone use and the risk for malignant brain tumors: a case-control study on deceased cases and controls.

Mobile phone use and the risk of acoustic neuroma.

Mobile Phone Use and the Risk of Parotid Gland Tumors: A Retrospective Case-Control Study.

Mobile phones and head tumours. The discrepancies in cause-effect relationships in the epidemiological studies - how do they arise?

Mobile phones and head tumours: it is time to read and highlight data in a proper way].

Mobile phones, cordless phones and the risk for brain tumours.

Mobile phones: time to rethink and limit usage.

Mobile telephones and cancer--a review of epidemiological evidence.

Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin.

Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais state, Brazil.

Mutagenic response of 2.45 GHz radiation exposure on rat brain.

Neoplastic transformation of C3H/10T1/2 cells following exposure to 120-Hz modulated 2.45-GHz microwaves and phorbol ester tumor promoter.

Neuroblastoma and paternal occupation. A case-control analysis.

New Zealand adolescents' cellphone and cordless phone user-habits: are they at increased risk of brain tumours already? A cross-sectional study.

Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: molecular mechanism for cancer- and blood-brain barrier-related effects.

Occupational exposure to high-frequency electromagnetic fields and brain tumor risk in the INTEROCC study: An individualized assessment approach.

Occupational exposures and brain cancer mortality: a preliminary study of east Texas residents.

Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation.

Oxidative and mutagenic effects of low intensity GSM 1800 MHz microwave radiation.

Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation.

Parental occupational exposures to electromagnetic fields and radiation and the incidence of neuroblastoma in offspring.

Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones.

Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects.

Pooled analysis of Swedish case-control studies during 1997-2003 and 2007-2009 on meningioma risk associated with the use of mobile and cordless phones.

Power-frequency magnetic fields and childhood brain tumors: a case-control study in Japan.

Probabilistic Multiple-Bias Modeling Applied to the Canadian Data From the Interphone Study of Mobile Phone Use and Risk of Glioma, Meningioma, Acoustic Neuroma, and Parotid Gland Tumors.

Proteomic analysis of continuous 900-MHz radiofrequency electromagnetic field exposure in testicular tissue: a rat model of human cell phone exposure.

Radio frequency radiation-related cancer: assessing causation in the occupational/military setting.

Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer.

Radiofrequency-induced carcinogenesis: cellular calcium homeostasis changes as a triggering factor.

Real versus Simulated Mobile Phone Exposures in Experimental Studies.

Real-world cell phone radiofrequency electromagnetic field exposures.

Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8GHz GSM base station environmental emission.

Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries.

Risks of carcinogenesis from electromagnetic radiation of mobile telephony devices.

Risks to Health and Well-Being From Radio-Frequency Radiation Emitted by Cell Phones and Other Wireless Devices.

Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action.

Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells.

Setting prudent public health policy for electromagnetic field exposures.

Simulation of the incidence of malignant brain tumors in birth cohorts that started using mobile phones when they first became popular in Japan.

Synergism between sinusoidal-50Hz magnetic field and formaldehyde in triggering carcinogenic effects in male Sprague-Dawley rats.

Terahertz radiation increases genomic instability in human lymphocytes.

The effect of electromagnetic radiation on the rat brain: an experimental study.

The electromagnetic fields of cellular phones and the health of children and of teenagers (the situation requiring to take an urgent measure)].

The Intracranial Distribution of Gliomas in Relation to Exposure From Mobile Phones: Analyses From the INTERPHONE Study.

The possible role of radiofrequency radiation in the development of uveal melanoma.

The probability of developing brain tumours among users of cellular telephones (scientific information to the decision of the International Agency for Research on Cancer (IARC) announced on May 31, 2011)].

Thermal and non-thermal health effects of low intensity non-ionizing radiation: An international perspective.

Towards 5G communication systems: Are there health implications?

Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma.

Use of cellular telephones and brain tumour risk in urban and rural areas.

Use of electric bedding devices and risk of breast cancer in African-American women.

Use of electric blankets and association with prevalence of endometrial cancer.

Use of mobile and cordless phones and survival of patients with glioma.

Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones.

Wi-Fi technology--an uncontrolled global experiment on the health of mankind.

Wireless Phone Use and Risk of Adult Glioma: Evidence from a Meta-Analysis.

X-rays, microwaves and vinyl chloride monomer: their clastogenic and aneugenic activity, using the micronucleus assay on human lymphocytes.

NEURODEGENERATIVE

Keywords – memory, cognitive, central nervous system, learning, neurodegenerative, Alzheimer's, learning and memory, Alzheimer's disease, cognition, amyotrophic lateral sclerosis, neurodegenerative diseases, cognitive function, cognitive functions, neurobehavioral, dementia, spatial learning, acetylcholine, Parkinson's disease, epilepsy, Glial fibrillary, motor activity, multiple sclerosis, cognitive impairment, spatial learning and memory, neurodegenerative disease, neuronal damage, Alzheimer disease, cognitive effects, seizure, seizures, autism, cognitive functioning, cognitive processing, memory function, memory impairment, memory loss, neurological diseases, neuronal excitability, cognitive dysfunction, memory deficit, memory functions, neurocognitive, neuronal degeneration, spatial working memory

Titles

2.45 GHz Microwave Radiation Impairs Learning and Spatial Memory via Oxidative/Nitrosative Stress Induced p53-Dependent/Independent Hippocampal Apoptosis: Molecular Basis and Underlying Mechanism.

A case-control study on the risk factors of Alzheimer's disease in military elderly men].

A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.

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Effect of Low Level Subchronic Microwave Radiation on Rat Brain.

Effect of Low-Intensity Microwave Radiation on Monoamine Neurotransmitters and Their Key Regulating Enzymes in Rat Brain.

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Effect of whole-body exposure to high-frequency electromagnetic field on the brain electrogeny in neurodefective and healthy mice.

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Exposure to radio-frequency electromagnetic waves alters acetylcholinesterase gene expression, exploratory and motor coordination-linked behaviour in male rats.

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REPRODUCTION

Keywords – pregnancy, reproductive, pregnant, sperm, embryos, testicular, fertility, embryo, testis, embryonic, fetuses, testosterone, motility, infertility, reproduction, testes, semen, spermatozoa, spermatogenesis, reproductive system, sperm motility, male fertility, sperm count, embryogenesis, abortion, male reproductive, spermatogenic, embryonic development, mating, male infertility, birth defects, serum testosterone, adverse reproductive, miscarriage, reproductive organs, semen parameters, sperm concentration, sperm parameters, testicular function, testosterone level, epididymis, male reproductive system, spermatogenic cells, spermatogonia, fertilized eggs, ovaries, reproductive capacity, reproductive outcomes, sperm cells, sperm morphology, fertile, pregnancies, reproductive function, testicular tissue, rat testes, rat testis, reproductive functions, reproductive systems, sperm DNA, spermatogonial, testis tissue, embryogeny, reproductive health, sperm cell, miscarriages, offsprings, oocyte, oogenesis, preterm birth, seminal vesicles, Sperm head, spermatids, sperms, testicles, fetal loss, genital, gonads, reproductive hormones, semen analysis

Titles

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

2.45 GHz microwave radiation induced oxidative and nitrosative stress mediated testicular apoptosis: Involvement of a p53 dependent bax-caspase-3 mediated pathway.

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Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study.

Effect of early pregnancy electromagnetic field exposure on embryo growth ceasing].

Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.

Effect of Electromagnetic Waves from Mobile Phones on Spermatogenesis in the Era of 4G-LTE.

Effect of exposure to radio frequency radiation emitted by cell phone on the developing dorsal root ganglion of chick embryo: a light microscopic study.

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Effect of long-term exposure of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on testes functions.

Effect of low power microwave on the mouse genome: a direct DNA analysis.

Effect of low-intensity extremely high frequency radiation on reproductive function in wistar rats.

Effect of Mobile Phone Radiation on Cardiovascular Development of Chick Embryo.

Effect of mobile telephones on sperm quality: a systematic review and meta-analysis.

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Effects of radiofrequency electromagnetic fields on mammalian spermatogenesis].

Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats.

Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study.

Effects of the exposure to mobile phones on male reproduction: a review of the literature.

Electromagnetic fields enhance chemically-induced hyperploidy in mammalian oocytes.

Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.

Epidemiologic evidence relevant to radar (microwave) effects.

EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses.

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Maternal cell phone use during pregnancy and child cognition at age 5years in 3 birth cohorts.

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GENOTOXICITY

Keywords – DNA damage, genotoxic, micronuclei, chromosomal, micronucleus, chromosome, genotoxicity, genotoxic effects, mutagenic, strand breaks, chromatin, mutation, DNA strand, Chromatid, mutations, chromosome aberrations, chromosomes, DNA fragmentation, double-strand, chromosomal aberrations, DNA repair, DNA strand breaks, micronucleus (MN), genetic damage, micronuclei (MN), Sister Chromatid, genome, blood leukocytes, double-strand breaks, oxidative DNA, chromosomal damage, DNA synthesis, mutant, cellular stress, chromosome aberration, oxidative DNA damage, Purkinje cells, DNA breaks, cell cycle arrest, clastogenic, genotoxic potential, keratinocytes, micronucleated, single strand, cell division, chromatid exchange, Chromatid Exchanges, genetic material, micronucleus test, Mutagenesis, cell cycle progression, cellular DNA, Cytochrome c, double strand, genetic effects, genomic instability, micronucleus frequency, DNA single-strand, DNA-damaging, Mutagen, mutagenicity, single strand breaks, chromatin condensation, chromosomal aberration, double-strand breaks (DSBs), strand breakage, cell cycle distribution, cell DNA, genetically, strand DNA

Titles

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

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Connection between Cell Phone use, p53 Gene Expression in Different Zones of Glioblastoma Multiforme and Survival Prognoses.

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Cytogenetic damage in human lymphocytes following GMSK phase modulated microwave exposure.

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DNA Damage of Lymphocytes in Volunteers after 4 hours Use of Mobile Phone.

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Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro].

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Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.

Effect of electromagnetic radiation of millimetric wave band on genome of somatic cells].

Effect of exposure to radio frequency radiation emitted by cell phone on the developing dorsal root ganglion of chick embryo: a light microscopic study.

Effect of GSTM1 and GSTT1 Polymorphisms on Genetic Damage in Humans Populations Exposed to Radiation From Mobile Towers.

Effect of Low Level Subchronic Microwave Radiation on Rat Brain.

Effect of low power microwave on the mouse genome: a direct DNA analysis.

Effect of low-intensity microwave radiation on proliferation of cultured epithelial cells of rabbit lens].

Effect of Mobile Phone Radiation on Cardiovascular Development of Chick Embryo.

Effect of Radiofrequency Radiation Emitted from 2G and 3G Cell Phone on Developing Liver of Chick Embryo - A Comparative Study.

Effect of Radiofrequency Radiation on Human Hematopoietic Stem Cells.

Effect of whole-body exposure to high-frequency electromagnetic field on the brain electrogeny in neurodefective and healthy mice.

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Effects of microwave radiation on thymocytes in mice at different power densities].

Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats.

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Electromagnetic fields enhance chemically-induced hyperploidy in mammalian oocytes.

Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells.

Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.

Epidemiologic evidence relevant to radar (microwave) effects.

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Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line.

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Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes to radiofrequency electromagnetic fields for 72 hours.

Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters.

Influence of 1.8 GHz microwave on DNA damage induced by 4 chemical mutagens].

Influence of electromagnetic fields on reproductive system of male rats.

Interference of vitamin E on the brain tissue damage by electromagnetic radiation of cell phone in pregnant and fetal rats].

Long-term microwave radiation affects male reproduction in rats].

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Terahertz radiation increases genomic instability in human lymphocytes.

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CARDIOVASCULAR

Keywords – Cardiac, cardiovascular, pacemaker, pacemakers, implanted, blood pressure, implantable, vascular, heart rate variability, myocardial, heart rate variability (HRV), implants, cardiac pacemakers, implantation, defibrillators, implant, cardioverter, myocardium, cardiovascular system, implantable cardioverter, Cardiovascular disease, defibrillator, fibrillation, arrhythmia, arterial blood pressure, autonomic nervous system, cardioverter defibrillators, implanted pacemakers, cardiac pacemaker, hypertension, arrhythmias, cardioverter-defibrillators, implantable cardioverter defibrillators, implantable cardioverter-defibrillators, pacemaker function, heart disease, implanted cardiac, tachycardia, cardiac devices, circulatory system, microcirculation, blood vessels, cardiomyocytes, cardiovascular effects, vascular permeability, atherosclerosis, cardiovascular diseases, ventricular fibrillation, arterial pressure, Atrial fibrillation, cardiac output, cardiovascular function, Implantable cardioverter defibrillator (ICD), implantable devices, arrhythmic, carotid artery, pacemaker dysfunction, pacemaker malfunction

Titles

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

5-HT contents change in peripheral blood of workers exposed to microwave and high frequency radiation].

Activation of VEGF/Flk-1-ERK Pathway Induced Blood-Brain Barrier Injury After Microwave Exposure.

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IMMUNITY

Keywords – lymphocytes, immune, lymphocyte, immune system, immunity, blood lymphocytes, leukocytes, antibodies, immune response, human lymphocytes, antibody, peripheral blood lymphocytes, immunological, leukocyte, neutrophils, lymphocytic, immune functions, immunoreactivity, autoimmune, immunization, monocytes, neutrophil, antigens, macrophage, immune parameters, immune responses, immunocompetent, natural killer cells, spleen lymphocytes, immunologic, immunoreactive, micronucleated cells, monoclonal antibodies, spleen cells, splenocytes, T lymphocytes, antibody production, antibody-forming, monoclonal antibody

Titles

954 MHz microwaves enhance the mutagenic properties of mitomycin C.

Cellphone electromagnetic radiation damages the testicular ultrastructure of male rats].

Electromagnetic fields may act via calcineurin inhibition to suppress immunity, thereby increasing risk for opportunistic infection: Conceivable mechanisms of action.

Exposure to 1.8 GHz electromagnetic fields affects morphology, DNA-related Raman spectra and mitochondrial functions in human lympho-monocytes.

Exposure to 900 MHz radiofrequency radiation induces caspase 3 activation in proliferating human lymphocytes.

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Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes to radiofrequency electromagnetic fields for 72 hours.

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Influence of 1.8 GHz microwave on DNA damage induced by 4 chemical mutagens].

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Study of low-intensity 2450-MHz microwave exposure enhancing the genotoxic effects of mitomycin C using micronucleus test and comet assay in vitro.

Studying the synergistic damage effects induced by 1.8 GHz radiofrequency field radiation (RFR) with four chemical mutagens on human lymphocyte DNA using comet assay in vitro.

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Effect of low-intensity microwave of on mitomycin C-induced genotoxicity in vitro].

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Effects of electromagnetic radiation on health and immune function of operators].

Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells].

BIOMARKERS

Keywords – apoptosis, oxidative stress, Malondialdehyde, reactive oxygen species, apoptotic, superoxide dismutase, lipid peroxidation, permeability, catalase, MDA, ROS, ROS), reactive oxygen species (ROS), Malondialdehyde (MDA), SOD), cell death, glutathione peroxidase, inflammatory, erythrocytes, oxidative damage, SOD, caspase-3, free radical, nitric oxide, free radicals, biomarkers, bcl-2, catalase (CAT), inflammation, corticosterone, edema, glutathione peroxidase (GSH-Px), cytokine, cytokines, alkaline phosphatase, cell apoptosis, protein kinase, ATP, glutathione (GSH), oxidation, TNF-alpha, Bax, Ca2+, estrogen, ornithine decarboxylase, red blood cells, intracellular calcium, cell damage, apoptotic cell, hemoglobin, lactate dehydrogenase, cerebral blood flow, glutamate, hydrogen peroxide, IL-1beta, Purkinje, serotonin, apoptotic cell death, barrier permeability, carbonyl, hormone levels, ornithine decarboxylase (ODC), acetylcholinesterase, calcium ion, Calcium ions, endothelial cells, GABA, MDA levels, ODC, xanthine oxidase, creatinine, intracellular ROS, cholinesterase, lipid peroxidation levels, pro-inflammatory, protein kinase C, adrenocorticotropic hormone, alanine aminotransferase, aspartate aminotransferase, caspase 3, caspase-9, catalase activity, glutathione levels, NF-kappaB, atrophy, nitric oxide synthase, cAMP, acid phosphatase, adenosine deaminase, adrenocorticotropic hormone (ACTH), blood cell count, blood platelets, Ca++, adrenaline, C-reactive protein, oxidative damages, Reactive Oxygen Species), vascular endothelial growth factor

Titles

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

2.45 GHz Microwave Radiation Impairs Learning and Spatial Memory via Oxidative/Nitrosative Stress Induced p53-Dependent/Independent Hippocampal Apoptosis: Molecular Basis and Underlying Mechanism.

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8-oxoG DNA glycosylase-1 inhibition sensitizes Neuro-2a cells to oxidative DNA base damage induced by 900 MHz radiofrequency electromagnetic radiation.

900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues.

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900-MHz microwave radiation promotes oxidation in rat brain.

915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons.

Activation of VEGF/Flk-1-ERK Pathway Induced Blood-Brain Barrier Injury After Microwave Exposure.

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Effect of Low-Intensity Microwave Radiation on Monoamine Neurotransmitters and Their Key Regulating Enzymes in Rat Brain.

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Effects of 2.45 GHz microwave exposures on the peroxidation status in Wistar rats.

Effects of 900-MHz electromagnetic field emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs.

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Electromagnetic fields (1.8 GHz) increase the permeability to sucrose of the blood-brain barrier in vitro.

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Electromagnetic fields at a mobile phone frequency (900 MHz) trigger the onset of general stress response along with DNA modifications in Eisenia fetida earthworms.

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Electromagnetic radiation (Wi-Fi) and epilepsy induce calcium entry and apoptosis through activation of TRPV1 channel in hippocampus and dorsal root ganglion of rats.

Electromagnetic radiation 2450 MHz exposure causes cognition deficit with mitochondrial dysfunction and activation of intrinsic pathway of apoptosis in rats.

Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.

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Long-term exposure to microwave radiation provokes cancer growth: evidences from radars and mobile communication systems.

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Microwave radiation induced oxidative stress, cognitive impairment and inflammation in brain of Fischer rats.

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Mobile phone (1800MHz) radiation impairs female reproduction in mice, Mus musculus, through stress induced inhibition of ovarian and uterine activity.

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Protective effects of Genistein on human renal tubular epithelial cells damage of microwave radiation].

Protective effects of luteolin on rat testis following exposure to 900 MHz electromagnetic field.

Pulse modulated 900 MHz radiation induces hypothyroidism and apoptosis in thyroid cells: a light, electron microscopy and immunohistochemical study.

Pulsed electromagnetic fields accelerate apoptotic rate in osteoclasts.

Pulsed or continuous electromagnetic field induce p53/p21-mediated apoptotic signaling pathway in mouse spermatogenic cells in vitro and thus may affect male fertility.

Radio frequency electromagnetic radiation (RF-EMR) from GSM (0.9/1.8GHz) mobile phones induces oxidative stress and reduces sperm motility in rats.

Radiofrequency electromagnetic radiation from cell phone causes defective testicular function in male Wistar rats.

Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice.

Radiofrequency radiation emitted from Wi-Fi (2.4 GHz) causes impaired insulin secretion and increased oxidative stress in rat pancreatic islets.

Radiofrequency-induced carcinogenesis: cellular calcium homeostasis changes as a triggering factor.

Reactive oxygen species formation and apoptosis in human peripheral blood mononuclear cell induced by 900 MHz mobile phone radiation.

Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field.

Reduction of phosphorylated synapsin I (ser-553) leads to spatial memory impairment by attenuating GABA release after microwave exposure in Wistar rats.

Role of Mitochondria in the Oxidative Stress Induced by Electromagnetic Fields: Focus on Reproductive Systems.

Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells.

Selenium supplementation ameliorates electromagnetic field-induced oxidative stress in the HEK293 cells.

Ten gigahertz microwave radiation impairs spatial memory, enzymes activity, and histopathology of developing mice brain.

Testicular apoptosis and histopathological changes induced by a 2.45 GHz electromagnetic field.

The antioxidant effect of Green Tea Mega EGCG against electromagnetic radiation-induced oxidative stress in the hippocampus and striatum of rats.

Ultrastructural change of rabbit lens epithelial cells induced by low power level microwave radiation].

Vitamin C protects rat cerebellum and encephalon from oxidative stress following exposure to radiofrequency wave generated by a BTS antenna model.

Zinc supplementation ameliorates electromagnetic field-induced lipid peroxidation in the rat brain.

SENSORY DISORDERS

Keywords – auditory, acoustic, ear, hypersensitivity, EHS, electromagnetic hypersensitivity, otoacoustic, vestibular, hypersensitive, cataract, cochlea, auditory system, inner ear, lens epithelial, corneal, tinnitus, vision, lenses, otoacoustic emissions, hearing loss, otoacoustic emission, epidermis, rabbit lens, dermatitis, auditory stimuli, cataractogenic, Auditory brainstem response (ABR), auditory evoked, electrohypersensitive, electrosensitivity, vestibular system, cochlear implants, dermatological, hearing function, hearing thresholds, pain sensitivity, pain threshold, skin complaints

Titles

A quantitative study on early changes in rabbit lens capsule epithelium induced by low power density microwave radiation].

A study on the effect of prolonged mobile phone use on pure tone audiometry thresholds of medical students of Sikkim.

Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation.

Alteration of glycine receptor immunoreactivity in the auditory brainstem of mice following three months of exposure to radiofrequency radiation at SAR 4.0 W/kg.

Assessment of intermittent UMTS electromagnetic field effects on blood circulation in the human auditory region using a near-infrared system.

Association between vestibular schwannomas and mobile phone use.

Audiologic disturbances in long-term mobile phone users.

Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields].

Cell phone use and acoustic neuroma: the need for standardized questionnaires and access to industry data.

Changes in gap junctional intercellular communication in rabbits lens epithelial cells induced by low power density microwave radiation.

Cognitive and neurobiological alterations in electromagnetic hypersensitive patients: results of a case-control study.

Contribution of physical factors to the complex anthropogenic load in an industrial town].

Decrease in the intensity of the cellular immune response and nonspecific inflammation upon exposure to extremely high frequency electromagnetic radiation].

DNA damage and repair induced by acute exposure of microwave from mobile phone on cultured human lens epithelial cells].

EEG Changes Due to Experimentally Induced 3G Mobile Phone Radiation.

Effect Of Electromagnetic Waves Emitted From Mobile Phone On Brain Stem Auditory Evoked Potential In Adult Males.

Effect of low-intensity microwave radiation on proliferation of cultured epithelial cells of rabbit lens].

Effect of superposed electromagnetic noise on DNA damage of lens epithelial cells induced by microwave radiation.

Effect of wide-band modulated electromagnetic fields on the workers of high-frequency telephone exchanges].

Effects of 2.45-GHz microwaves on primate corneal endothelium.

Effects of different dose microwave radiation on protein components of cultured rabbit lens].

Effects of exposure to 2100MHz GSM-like radiofrequency electromagnetic field on auditory system of rats.

Effects of GSM-like radiofrequency on distortion product otoacoustic emissions in pregnant adult rabbits.

Effects of intensive and moderate cellular phone use on hearing function.

Effects of low level electromagnetic field exposure at 2.45 GHz on rat cornea.

Effects of microwave radiation on the eye: the occupational health perspective.

Effects of mobile phones on oxidant/antioxidant balance in cornea and lens of rats.

Effects of pulsed electromagnetic fields on cognitive processes - a pilot study on pulsed field interference with cognitive regeneration.

Electromagnetic field induced biological effects in humans.

Electromagnetic hypersensitivity--an increasing challenge to the medical profession.

Experimental studies on the influence of millimeter radiation on light transmission through the lens].

Increased sensitivity of the non-human primate eye to microwave radiation following ophthalmic drug pretreatment.

Intraoperative observation of changes in cochlear nerve action potentials during exposure to electromagnetic fields generated by mobile phones.

Is human saliva an indicator of the adverse health effects of using mobile phones?

Low power density microwave radiation induced early changes in rabbit lens epithelial cells.

Mobile phone induced sensorineural hearing loss.

Mobile phone related-hazards and subjective hearing and vision symptoms in the Saudi population.

Non-thermal electromagnetic radiation damage to lens epithelium.

Occupational safety: effects of workplace radiofrequencies on hearing function.

Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder.

Replication of heart rate variability provocation study with 2.4-GHz cordless phone confirms original findings.

Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

Some ocular symptoms experienced by users of mobile phones.

The acute auditory effects of exposure for 60 minutes to mobile`s electromagnetic field.

The effect of radiofrequency radiation generated by a Global System for Mobile Communications source on cochlear development in a rat model.

The effect of very low dose pulsed magnetic waves on cochlea.

The effects of pulsed low-level EM fields on memory processes].

The electromagnetic fields of cellular phones and the health of children and of teenagers (the situation requiring to take an urgent measure)].

Tinnitus and cell phones: the role of electromagnetic radiofrequency radiation.

Tinnitus and mobile phone use.

Ultrastructural change of rabbit lens epithelial cells induced by low power level microwave radiation].

DISCOMFORT SYMPTOMS

Keywords – depression, anxiety, headache, headaches, dizziness, depressed, depressive, vertigo, cataracts, behavioral effects, nausea, headache dizziness, low back pain, behavioural effects

Titles

A study on the biological effects of exposure mobile-phone frequency EMF].

A survey study on some neurological symptoms and sensations experienced by long term users of mobile phones.

Adverse effects of excessive mobile phone use.

Anxiogenic effect of chronic exposure to extremely low frequency magnetic field in adult rats.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Chronic exposure to ELF fields may induce depression.

Clinical features of headache associated with mobile phone use: a cross-sectional study in university students.

Effect of electromagnetic radiations from mobile phone base stations on general health and salivary function.

Effect of high-frequency EMF on public health and its neuro-chemical investigations].

Effect of low intensity and very high frequency electromagnetic radiation on occupationally exposed personnel].

Effects of electromagnetic radiation from cellular telephone handsets on symptoms of neurasthenia].

Effects of electromagnetic radiation on health and immune function of operators].

Effects of GSM-Frequency Electromagnetic Radiation on Some Physiological and Biochemical Parameters in Rats.

Effects of low intensity radiofrequency electromagnetic fields on electrical activity in rat hippocampal slices.

Effects of microwave radiation on the eye: the occupational health perspective.

Effects of mobile phone radiation (900 MHz radiofrequency) on structure and functions of rat brain.

Exposure to mobile phone electromagnetic field radiation, ringtone and vibration affects anxiety-like behaviour and oxidative stress biomarkers in albino wistar rats.

Exposure to radio-frequency electromagnetic waves alters acetylcholinesterase gene expression, exploratory and motor coordination-linked behaviour in male rats.

Long-term exposure of 2450MHz electromagnetic radiation induces stress and anxiety like behavior in rats.

Magnetic fields of transmission lines and depression.

Microwave radiation and chlordiazepoxide: synergistic effects on fixed-interval behavior.

Microwave sickness: a reappraisal.

Mobile phone use and health symptoms in children.

Mobile Phone Use and The Risk of Headache: A Systematic Review and Meta-analysis of Cross-sectional Studies.

Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir.

Motor activity of rabbits in conditions of chronic low-intensity pulse microwave irradiation].

MRI magnetic field stimulates rotational sensors of the brain.

Neurobehavioral effects among inhabitants around mobile phone base stations.

Postnatal development and behavior effects of in-utero exposure of rats to radiofrequency waves emitted from conventional WiFi devices.

Preliminary report: symptoms associated with mobile phone use.

Prevalence of headache among handheld cellular telephone users in Singapore: a community study.

Radiofrequency electromagnetic radiation-induced behavioral changes and their possible basis.

Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action.

Self-reported symptoms associated with exposure to electromagnetic fields: a questionnaire study.

Self-reporting of symptom development from exposure to radiofrequency fields of wireless smart meters in victoria, australia: a case series.

Subjective complaints of people living near mobile phone base stations in Poland.

Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations.

CONGENITAL ABNORMALITIES

Keywords – malformations, teratogenic, congenital, congenital malformations, teratogenicity, teratogens, teratologic, cleft palate, congenital anomalies, malformed, teratological

Titles

Are microwaves a co-teratogen? Experimental model concept and its verification].

Effect of early pregnancy electromagnetic field exposure on embryo growth ceasing].

Effects of continuous low-level exposure to radiofrequency radiation on intrauterine development in rats.

Effects of GSM-like radiofrequency irradiation during the oogenesis and spermiogenesis of Xenopus laevis.

Effects of MR exposure at 1.5 T on early embryonic development of the chick.

First cell cycles of sea urchin Paracentrotus lividus are dramatically impaired by exposure to extremely low-frequency electromagnetic field.

Lethal and teratogenic effects of long-term low-intensity radio frequency radiation at 428 MHz on developing chick embryo.

Microwave radiation enhances teratogenic effect of cytosine arabinoside in mice.

Morinda officialis how extract improves microwave-induced reproductive impairment in male rats].

MRI effects on craniofacial size and crown-rump length in C57BL/6J mice in 1.5T fields.

Pathological study of testicular injury induced by high power microwave radiation in rats].

Pulsed magnetic field from video display terminals enhances teratogenic effects of cytosine arabinoside in mice.

Reproductive hazards among workers at high voltage substations.

Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. I. Morphologic analysis at term.

Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. II. Postnatal psychophysiologic evaluations.

Teratogenic effects of sinusoidal extremely low frequency electromagnetic fields on morphology of 24 hr chick embryos.

Teratogenic, biochemical, and histological studies with mice prenatally exposed to 2.45-GHz microwave radiation.

VDT pulse magnetic field enhances teratogenic effect of ara-c in mice.

CIRCADIAN RHYTHYM AND MELATONIN

Keywords – melatonin, sleep, circadian, melatonin production, sleep disturbances, insomnia, melatonin levels, melatonin secretion, sleep disorders, sleep EEG, poor sleep, pineal function

Titles

900-MHz microwave radiation promotes oxidation in rat brain.

A 50-Hz electromagnetic field impairs sleep.

Association between Excessive Use of Mobile Phone and Insomnia and Depression among Japanese Adolescents.

Association between overuse of mobile phones on quality of sleep and general health among occupational health and safety students.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Bedtime mobile phone use and sleep in adults.

Biological effects of continuous exposure of embryos and young chickens to electromagnetic fields emitted by video display units.

Breast cancer and electric power.

Cellular phones: are they detrimental?

Chronic exposure to ELF fields may induce depression.

Chronotoxicity of 1800 MHz microwave radiation on sex hormones and spermatogenesis in male mice].

Circadian rhythmicity of antioxidant markers in rats exposed to 1.8 GHz radiofrequency fields.

Direct suppressive effects of weak magnetic fields (50 Hz and 16 2/3 Hz) on melatonin synthesis in the pineal gland of Djungarian hamsters (Phodopus sungorus).

Do magnetic fields cause increased risk of childhood leukemia via melatonin disruption?

Effect of electromagnetic radiations from mobile phone base stations on general health and salivary function.

Effect of low intensity and very high frequency electromagnetic radiation on occupationally exposed personnel].

Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats.

Effects of electromagnetic fields exposure on plasma hormonal and inflammatory pathway biomarkers in male workers of a power plant.

Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats.

Effects of Mobile Phones on Children's and Adolescents' Health: A Commentary.

Effects of prenatal 900 MHz electromagnetic field exposures on the histology of rat kidney.

EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses.

Exposure to electromagnetic fields and suicide among electric utility workers: a nested case-control study.

Health effects of living near mobile phone base transceiver station (BTS) antennae: a report from Isfahan, Iran.

Individual differences in the effects of mobile phone exposure on human sleep: rethinking the problem.

Investigation on the health of people living near mobile telephone relay stations: I/Incidence according to distance and sex].

Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells.

Melatonin attenuates radiofrequency radiation (900 MHz)-induced oxidative stress, DNA damage and cell cycle arrest in germ cells of male Swiss albino mice.

Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain.

Melatonin protects rat thymus against oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes.

Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain.

Microwave frequency electromagnetic fields (EMFs) produce widespread neuropsychiatric effects including depression.

Mitochondrial DNA damage and oxidative damage in HL-60 cells exposed to 900MHz radiofrequency fields.

Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin.

Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir.

Mobile phones: time to rethink and limit usage.

Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin.

Neurobehavioral effects among inhabitants around mobile phone base stations.

Neuroprotective effects of melatonin and omega-3 on hippocampal cells prenatally exposed to 900 MHz electromagnetic fields.

Non-thermal biomarkers of exposure to radiofrequency/microwave radiation.

Non-thermal continuous and modulated electromagnetic radiation fields effects on sleep EEG of rats.

Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin.

CHRONIC CONDITIONS

Keywords - metabolism, metabolic, glucose, endocrine, cholesterol, Diabetes, calcium homeostasis, glucose levels, homeostatic, metabolic activity, metabolic heat production, Diabetes Mellitus, diabetic, glucose metabolism, obesity

Titles

Assessment of biological changes of continuous whole-body exposure to static magnetic field and extremely low frequency electromagnetic fields in mice.

Association of exposure to radio-frequency electromagnetic field radiation (RF-EMFR) generated by mobile phone base stations with glycated hemoglobin (HbA1c) and risk of Type 2 Diabetes Mellitus.

Biological accounts emerging from some kinds of electromagnetic waves in the environment.

Calreticulin attenuated microwave radiation-induced human microvascular endothelial cell injury through promoting actin acetylation and polymerization.

Cardiovascular risk in operators under radiofrequency electromagnetic radiation.

Cell oxidation-reduction imbalance after modulated radiofrequency radiation.

Changes in mitochondrial functioning with electromagnetic radiation of ultra high frequency as revealed by electron paramagnetic resonance methods.

Common behaviors alterations after extremely low-frequency electromagnetic field exposure in rat animal model.

Dirty electricity, chronic stress, neurotransmitters and disease.

Disordered redox metabolism of brain cells in rats exposed to low doses of ionizing radiation or UHF electromagnetic radiation.

Disturbances of glucose tolerance in workers exposed to electromagnetic radiation].

Dynamics of metabolic parameters in rats during repeated exposure to modulated low-intensity UHF radiation.

Effect of a 20 kHz sawtooth magnetic field exposure on the estrous cycle in mice.

Effect of coherent extremely high-frequency and low-intensity electromagnetic radiation on the activity of membrane systems in Escherichia coli].

Effect of discontinuous short-wave electromagnetic field irradiation on the state of the endocrine glands].

Effect of Exposure to 900 MHz GSM Mobile Phone Radiofrequency Radiation on Estrogen Receptor Methylation Status in Colon Cells of Male Sprague Dawley Rats.

Effects of continuous low-level exposure to radiofrequency radiation on intrauterine development in rats.

Effects of continuous-wave, pulsed, and sinusoidal-amplitude-modulated microwaves on brain energy metabolism.

Effects of electromagnetic fields on the immune systems of occupationally exposed humans and mice.

Effects of electromagnetic radiation exposure on bone mineral density, thyroid, and oxidative stress index in electrical workers.

Effects of exposure to electromagnetic field radiation (EMFR) generated by activated mobile phones on fasting blood glucose.

Effects of extremely low frequency electromagnetic field and its combination with lead on the antioxidant system in mouse].

Effects of microwave radiation on the eye: the occupational health perspective.

Effects of RF-EMF Exposure from GSM Mobile Phones on Proliferation Rate of Human Adipose-derived Stem Cells: An In-vitro Study.

Endocrine mechanism of placental circulatory disturbances induced by microwave in pregnant rats].

Evidence that dirty electricity is causing the worldwide epidemics of obesity and diabetes.

Exposure to GSM 900 MHz electromagnetic fields affects cerebral cytochrome c oxidase activity.

Functional activity and metabolism of blood neutrophils exposed to low-intensity microwaves].

Glucose administration attenuates spatial memory deficits induced by chronic low-power-density microwave exposure.

GSM mobile phone radiation suppresses brain glucose metabolism.

High-frequency electromagnetic field exposure on reproductive and endocrine functions of female workers].

Hippocampal lipidome and transcriptome profile alterations triggered by acute exposure of mice to GSM 1800 MHz mobile phone radiation: An exploratory study.

Long-term exposure to microwave radiation provokes cancer growth: evidences from radars and mobile communication systems.

Metabolic changes in cells under electromagnetic radiation of mobile communication systems].

Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer's disease.

Pulse modulated 900 MHz radiation induces hypothyroidism and apoptosis in thyroid cells: a light, electron microscopy and immunohistochemical study.

Radio frequency electromagnetic radiation (RF-EMR) from GSM (0.9/1.8GHz) mobile phones induces oxidative stress and reduces sperm motility in rats.

Radiofrequency radiation emitted from Wi-Fi (2.4 GHz) causes impaired insulin secretion and increased oxidative stress in rat pancreatic islets.

Towards 5G communication systems: Are there health implications?

Wi-Fi is an important threat to human health.

A2-C. Citing Papers

Essentially all the papers referenced in A2-B show adverse effects. The papers that cite these adverse effects papers (and some associated papers) were retrieved, and were filtered by visual inspection. The references to these citing papers that also show adverse effects from wireless radiation are presented in the following. The combination of relevant papers in A2-B and their citing papers in A2-C constitutes a representative sample of the wireless radiation adverse effects literature.

The actual literature is far larger. The query used to retrieve relevant papers for A2-B was quite simple, and mainly the citing papers component of the citation network (citing papers, cited papers, related records, etc) was used to expand the relevant papers.

CITING PAPERS

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Appendix 3 – Factor Analysis of Adverse EMF Effects Database

A3-A. Factor Themes

A query to retrieve Medline records showing adverse health effects of wireless radiation was generated. The query was entered into the Medline search engine, and ~15,000 records were retrieved. Filtering was applied to the retrieval to remove records not associated with adverse health effects of wireless radiation, and 5311 records remained. These records did not receive the further filtering as the database in Appendix 2.

Thousands of the highest frequency MeSH terms were read, and those strongly related to adverse health effects of wireless radiation were selected. A factor analysis was performed using these terms, and a 21-factor taxonomy was generated.

The following table (A3-1) shows the categories/factors in the taxonomy, and the highest weighted (most influential in determining the factor theme) MeSH Headings associated with each category/factor. For each category, the records associated with the highest weighted MeSH Headings identified were highlighted, and the titles of those records were extracted. Following the table, each category and associated record titles are shown in order to display the breadth of coverage of the category. The categories in Table A3-1 are hyperlinked to their respective titles. Because of the limitations on record filtering, most of the records show adverse health effects, but not all do. Some of the records go beyond the FCC exposure limits, and some address frequencies much lower than microwave. There is some overlap among factors, since some MeSH Headings may be influential in determining the themes of multiple factors.

Major themes from the table include cancer, breast cancer, liver cancer, skin cancer, brain cancer, leukemia, tumors, precancerous conditions, neurodegenerative diseases, cardiovascular disease, electronic implant dysfunction, cerebrovascular disorders, inflammation, oxidative stress, male infertility, electrohypersensitivity, sleep, congenital abnormalities, sensory disorders, symptoms of discomfort, eye diseases.

Table A3-1 - Factor Analysis Taxonomy

FACTOR THEME	MESH HEADINGS
1 **Electromagnetic hypersensitivity and inflammation**	C-Reactive Protein, Liver Diseases, Thyroid Diseases, Inflammation, Tonsillitis, Hypersensitivity
2 **Coronary artery disease**	Plaque, Atherosclerotic, Coronary Artery Disease, Diabetes Mellitus, Carotid Artery Diseases, Inflammation, Hypertension
3A **Congenital abnormalities**	Cleft Lip, Cleft Palate, Calcification, Physiologic, Congenital Abnormalities
3B **Mammary tumors**	Fibroadenoma, Adenoma, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma
4 **Male infertility**	Sperm Count, Spermatozoa, Sperm Motility, Semen, Testis, Infertility, Male, Spermatogenesis, Testosterone, Fertility
5 **Brain neoplasms**	Meningioma, Glioma, Meningeal Neoplasms, Neuroma, Acoustic, Brain Neoplasms, Glioblastoma, Neoplasms, Radiation-Induced, Neuroma, Cranial Nerve Neoplasms, Parotid Neoplasms, Central Nervous System Neoplasms
6 **Sensory disorders**	Burning Mouth Syndrome, Taste Disorders, Skin Diseases, Mouth Diseases, Dizziness, Vision Disorders, Hypersensitivity, Delayed, Fatigue
7 **Breast neoplasms**	Carcinoma, Lobular, Carcinoma, Ductal, Breast, Breast Neoplasms, Male, Adenoma
8 **Oxidative stress**	Oxidative Stress, Malondialdehyde, Glutathione Peroxidase, Lipid Peroxidation, Reactive Oxygen Species, Apoptosis, DNA Damage, Nitric Oxide, Protein Carbonylation
9 **Neurodegenerative diseases**	Parkinson Disease, Neurodegenerative Diseases, Alzheimer Disease, Amyotrophic Lateral Sclerosis, Motor Neuron Disease, Occupational Diseases, Dementia, Brain Diseases, Dementia, Vascular
10 **Cerebrovascular disorders**	Cerebrovascular Disorders, Dementia, Migraine Disorders, Tinnitus, Headache, Sleep Wake Disorders, Carotid Artery Diseases, Alzheimer Disease, Dementia, Vascular

11 **Congenital abnormalities and glandular-based tumors**	Cleft Lip, Cleft Palate, Fibroadenoma, Adenoma, Calcification, Physiologic, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma
12 **Skin neoplasms**	Carcinoma, Basal Cell, Carcinoma, Squamous Cell, Skin Neoplasms, Cocarcinogenesis, Neoplasms, Experimental, Neoplasms, Radiation-Induced, Colonic Neoplasms
13 **Leukemia**	Leukemia, Myeloid, Acute, Leukemia, Lymphocytic, Chronic, B-Cell, Leukemia, Myelogenous, Chronic, BCR-ABL Positive, Leukemia, Myeloid, Leukemia, Multiple Myeloma, Lymphoma, Leukemia, Radiation-Induced, Acute Disease, Liver Neoplasms, Experimental, Central Nervous System Neoplasms
14 **Precancerous conditions**	Atrophy, Precancerous Conditions, Hyperplasia, Hypersensitivity, Delayed, Thymus Gland, Capillary Permeability, Lymphoma
15 **Circadian Rhythm**	Melatonin, Circadian Rhythm, Pineal Gland
16 **Eye diseases**	Eye Diseases, Cataract, Vision Disorders, Sensation Disorders, Neurotic Disorders, Lens, Crystalline, Corneal Diseases, Edema, Hematologic Diseases
17 **Electromagnetic interference in implanted electronic devices**	Tachycardia, Ventricular, Ventricular Fibrillation, Death, Sudden, Cardiac, Arrhythmias, Cardiac
18 **Liver Neoplasms**	Liver Neoplasms, Carcinoma, Hepatocellular, Neoplasm Recurrence, Local, Lymphatic Metastasis
19 **Symptoms of discomfort**	Headache, Dizziness, Fatigue, Depression, Anxiety, Tremor, Sleep Wake Disorders, Neurotic Disorders, Stress, Psychological, Anxiety Disorders, Nervous System Diseases
20 **Neoplasms**	Lung Neoplasms, Ovarian Neoplasms, Pituitary Neoplasms, Lymphoma, Prostatic Neoplasms, Colonic Neoplasms, Carcinoma, Breast Neoplasms, Hematologic Neoplasms, Neoplasms, Liver Neoplasms, Cell Transformation, Neoplastic, Nervous System Neoplasms

A3-B. Factor Record Titles

FACTOR 1

Theme – Electromagnetic hypersensitivity and inflammation

Key MeSH Headings - C-Reactive Protein, Liver Diseases, Thyroid Diseases, Inflammation, Tonsillitis, Hypersensitivity

Titles

915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons.

A cognitive-behavioral treatment of patients suffering from "electric hypersensitivity". Subjective effects and reactions in a double-blind provocation study.

A systematic review of treatments for electromagnetic hypersensitivity.

Activation of TLR signalling regulates microwave radiation-mediated impairment of spermatogenesis in rat testis.

Analysis of the effect of a 60 Hz AC field on histamine release by rat peritoneal mast cells.

Are thyroid dysfunctions related to stress or microwave exposure (900 MHz)?

Bilateral symmetry of local inflammatory activation in human carotid atherosclerotic plaques.

Biological effects of low-level environmental agents.

Blood laboratory findings in patients suffering from self-perceived electromagnetic hypersensitivity (EHS).

Changes in antioxidant capacity of blood due to mutual action of electromagnetic field (1800 MHz) and opioid drug (tramadol) in animal model of persistent inflammatory state.

Changes in the chromatin structure of lymphoid cells under the influence of low-intensity extremely high-frequency electromagnetic radiation against the background of inflammatory process].

Clinical significance of tonsillar provocation test in diagnosis of tonsillar focal infection--by indirect irradiation of ultra-micro waves].

Controversies around electromagnetic fields and electromagnetic hypersensitivity. The construction of "low noise" public problems].

Decrease in the intensity of the cellular immune response and nonspecific inflammation upon exposure to extremely high frequency electromagnetic radiation].

Dependence of anti-inflammatory effects of high peak-power pulsed electromagnetic radiation of extremely high frequency on exposure parameters].

Description of persons with symptoms presumed to be caused by electricity or visual display units--oral aspects.

Development and evaluation of the electromagnetic hypersensitivity questionnaire.

Earthing: health implications of reconnecting the human body to the Earth's surface electrons.

Effect of high frequency electromagnetic wave stimulation on muscle injury in a rat model.

Effect of mobile phone use on salivary concentrations of protein, amylase, lipase, immunoglobulin A, lysozyme, lactoferrin, peroxidase and C-reactive protein of the parotid gland.

Effect of quinacrine on inflammatory reaction of blood system induced by microwave irradiation].

Effect of the pulsed electromagnetic field on the release of inflammatory mediators from adipose-derived stem cells (ADSCs) in rats.

Effects of low-intensity ultrahigh frequency electromagnetic radiation on inflammatory processes.

Effects of personalised exposure on self-rated electromagnetic hypersensitivity and sensibility - A double-blind randomised controlled trial.

Effects of RF fields emitted from smart phones on cardio-respiratory parameters: a preliminary provocation study.

Electrical hypersensitivity in humans--fact or fiction?

Electrohypersensitivity: a functional impairment due to an inaccessible environment.

Electromagnetic fields (EMF): do they play a role in children's environmental health (CEH)?

Electromagnetic fields and health outcomes.

Electromagnetic fields hypersensitivity].

Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication--a literature review published between 2000 and 2004.

Electromagnetic hypersensitivity--an increasing challenge to the medical profession.

Electromagnetic hypersensitivity: biological effects of dirty electricity with emphasis on diabetes and multiple sclerosis.

Electromagnetic hypersensitivity: fact or fiction?

Epidemiology and etiology of gliomas.

Extremely low-frequency electromagnetic field exposure enhances inflammatory response and inhibits effect of antioxidant in RAW 264.7 cells.

Features of anti-inflammatory effects of modulated extremely high-frequency electromagnetic radiation.

Functional brain MRI in patients complaining of electrohypersensitivity after long term exposure to electromagnetic fields.

Heavy metal exposure in patients suffering from electromagnetic hypersensitivity.

Hsp70 is an independent stress marker among frequent users of mobile phones.

Hypersensitivity symptoms associated with exposure to cellular telephones: no causal link.

Hypersensitivity syndrome].

Hypersensitivity to electricity: working definition and additional characterization of the syndrome.

Idiopathic environmental intolerance attributed to electromagnetic fields (formerly 'electromagnetic hypersensitivity'): An updated systematic review of provocation studies.

Increased mercury release from dental amalgam restorations after exposure to electromagnetic fields as a potential hazard for hypersensitive people and pregnant women.

Induction of macrophage migration inhibitory factor precedes the onset of acute tonsillitis.

Microwaves from GSM mobile telephones affect 53BP1 and gamma-H2AX foci in human lymphocytes from hypersensitive and healthy persons.

Mobile-phone-based home exercise training program decreases systemic inflammation in COPD: a pilot study.

Physiological variables and subjective symptoms by 60 Hz magnetic field in EHS and non-EHS persons.

Prevalence of self-reported hypersensitivity to electric or magnetic fields in a population-based questionnaire survey.

Provocation of electric hypersensitivity under everyday conditions.

Provocation study of persons with perceived electrical hypersensitivity and controls using magnetic field exposure and recording of electrophysiological characteristics.

Provocation with stress and electricity of patients with "sensitivity to electricity".

Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder.

Sensitivity of spiral ganglion neurons to damage caused by mobile phone electromagnetic radiation will increase in lipopolysaccharide-induced inflammation in vitro model.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

The amelioration of phagocytic ability in microglial cells by curcumin through the inhibition of EMF-induced pro-inflammatory responses.

The effect of melatonin on the liver of rats exposed to microwave radiation.

The implications of non-linear biological oscillations on human electrophysiology for electrohypersensitivity (EHS) and multiple chemical sensitivity (MCS).

The microwave syndrome or electro-hypersensitivity: historical background.

The role of fatty acids in anti-inflammatory effects of low-intensity extremely high-frequency electromagnetic radiation.

The role of microwave radiometry in carotid artery disease. Diagnostic and clinical prospective.

Thermal and non-thermal health effects of low intensity non-ionizing radiation: An international perspective.

Thermal Response of In Vivo Human Skin to Fractional Radiofrequency Microneedle Device.

Use of terahertz electromagnetic radiation at nitric oxide frequencies for the correction of thyroid functional state during stress].

Wireless communication fields and non-specific symptoms of ill health: a literature review.

Women growing older with environmental sensitivities: A grounded theory model of meeting one's needs.

FACTOR 2

Theme – Coronary artery disease

Key MeSH Headings - Plaque, Atherosclerotic, Coronary Artery Disease, Diabetes Mellitus, Carotid Artery Diseases, Inflammation, Hypertension

Titles

A study on the biological effects of exposure mobile-phone frequency EMF].

A survey on diabetes mellitus in the staff of electric power system in Baotou city].

Activation of TLR signalling regulates microwave radiation-mediated impairment of spermatogenesis in rat testis.

Analysis of the effect of a 60 Hz AC field on histamine release by rat peritoneal mast cells.

Bilateral symmetry of local inflammatory activation in human carotid atherosclerotic plaques.

Blood laboratory findings in patients suffering from self-perceived electromagnetic hypersensitivity (EHS).

Cardiovascular risk in operators under radiofrequency electromagnetic radiation.

Changes in antioxidant capacity of blood due to mutual action of electromagnetic field (1800 MHz) and opioid drug (tramadol) in animal model of persistent inflammatory state.

Changes in the chromatin structure of lymphoid cells under the influence of low-intensity extremely high-frequency electromagnetic radiation against the background of inflammatory process].

Decrease in the intensity of the cellular immune response and nonspecific inflammation upon exposure to extremely high frequency electromagnetic radiation].

Dependence of anti-inflammatory effects of high peak-power pulsed electromagnetic radiation of extremely high frequency on exposure parameters].

Development of hypertension after long-term exposure to static magnetic fields among workers from a magnetic resonance imaging device manufacturing facility.

Earthing: health implications of reconnecting the human body to the Earth's surface electrons.

Effect of electromagnetic irradiation of the millimetric range on hemodynamics in patients with arterial hypertension].

Effect of high frequency electromagnetic wave stimulation on muscle injury in a rat model.

Effect of quinacrine on inflammatory reaction of blood system induced by microwave irradiation].

Effect of the pulsed electromagnetic field on the release of inflammatory mediators from adipose-derived stem cells (ADSCs) in rats.

Effects of low-intensity ultrahigh frequency electromagnetic radiation on inflammatory processes.

Electromagnetic effects on people.

Electromagnetic hypersensitivity: biological effects of dirty electricity with emphasis on diabetes and multiple sclerosis.

Epidemiological risk assessment of pathology development in occupational exposure to radiofrequency electromagnetic fields].

Evaluation of occupational risk caused by exposure to electromagnetic rays].

Evidence that dirty electricity is causing the worldwide epidemics of obesity and diabetes.

Exacerbation of hypertension and disturbances of the geomagnetic field].

Exposure to radio-frequency radiation from an aircraft radar unit.

Extremely low-frequency electromagnetic field exposure enhances inflammatory response and inhibits effect of antioxidant in RAW 264.7 cells.

Features of anti-inflammatory effects of modulated extremely high-frequency electromagnetic radiation.

Health care utilisation and attitudes towards health care in subjects reporting environmental annoyance from electricity and chemicals.

Mobile-phone-based home exercise training program decreases systemic inflammation in COPD: a pilot study.

Psychological symptoms and intermittent hypertension following acute microwave exposure.

Radiofrequency Scanning for Retained Surgical Items Can Cause Electromagnetic Interference and Pacing Inhibition if an Asynchronous Pacing Mode Is Not Applied.

Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder.

Role of ultrasonic dopplerography in monitoring the effectiveness of treatment of patients who have sustained a stroke with decimeter-range electromagnetic waves].

Sensitivity of spiral ganglion neurons to damage caused by mobile phone electromagnetic radiation will increase in lipopolysaccharide-induced inflammation in vitro model.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

The amelioration of phagocytic ability in microglial cells by curcumin through the inhibition of EMF-induced pro-inflammatory responses.

The heliogeophysical aspects of circumpolar health.

The role of fatty acids in anti-inflammatory effects of low-intensity extremely high-frequency electromagnetic radiation.

The role of microwave radiometry in carotid artery disease. Diagnostic and clinical prospective.

Thermal Response of In Vivo Human Skin to Fractional Radiofrequency Microneedle Device.

FACTOR 3A

Theme – Congenital abnormalities

Key MeSH Headings - Cleft Lip, Cleft Palate, Calcification, Physiologic, Congenital Abnormalities

Titles

A confirmation study of Russian and Ukrainian data on effects of 2450 MHz microwave exposure on immunological processes and teratology in rats.

Adverse human reproductive outcomes and electromagnetic fields: a brief summary of the epidemiologic literature.

Age diseases depending on geomagnetic field activity inside the womb period].

Alternative functional relationships between ELF field exposure and possible health effects: report on an expert workshop.

An evaluation of the mutagenic, carcinogenic and teratogenic potential of microwaves.

An international project to confirm Soviet-era results on immunological and teratological effects of RF field exposure in Wistar rats and comments on Grigoriev et al. [2010].

Anesthesia as an effective agent against the production of congenital anomalies in mouse fetuses exposed to electromagnetic radiation.

Are microwaves a co-teratogen? Experimental model concept and its verification].

Case-control study on maternal residential proximity to high voltage power lines and congenital anomalies in France.

Chick embryo development can be irreversibly altered by early exposure to weak extremely-low-frequency magnetic fields.

Clinical teratology.

Congenital anomalies in the offspring of rats after exposure of the testis to an electrostatic field.

Contribution of physical factors to the complex anthropogenic load in an industrial town].

Development of chicken embryos in a pulsed magnetic field.

Development of preincubated chicken eggs following exposure to 50 Hz electromagnetic fields with 1.33-7.32 mT flux densities.

Developmental changes in Drosophila melanogaster following exposure to alternating electromagnetic fields.

Developmental toxicity interactions of salicylic acid and radiofrequency radiation or 2-methoxyethanol in rats.

Effects of 2.45 GHz CW microwave radiation on embryofetal development in mice.

Effects of gestational exposure to 1.95-GHz W-CDMA signals for IMT-2000 cellular phones: Lack of embryotoxicity and teratogenicity in rats.

Effects of noise and electromagnetic fields on reproductive outcomes.

Electromagnetic poles and reproduction].

EMF and health.

Epidemiological studies of work with video display terminals and adverse pregnancy outcomes (1984-1992).

Evaluation of the developmental toxicity of 60 Hz magnetic fields and harmonic frequencies in Sprague-Dawley rats.

Interaction of static and extremely low frequency electric and magnetic fields with living systems: health effects and research needs.

Joint actions of environmental nonionizing electromagnetic fields and chemical pollution in cancer promotion.

Maternal exposure to magnetic fields from high-voltage power lines and the risk of birth defects.

Maternal proximity to extremely low frequency electromagnetic fields and risk of birth defects.

Mouse early embryos obtained by natural breeding or in vitro fertilization display a differential sensitivity to extremely low-frequency electromagnetic fields.

Mutagenic, carcinogenic and teratogenic effects induced by radiofrequency electromagnetic field of mobile phone].

Neural and behavioral teratological evaluation of rats exposed to ultra-wideband electromagnetic fields.

Paternal work in the power industry: effects on children at delivery.

Possible effects of electric blankets and heated waterbeds on fetal development.

Prospective study of pregnancy outcomes after parental cell phone exposure: the Norwegian Mother and Child Cohort Study.

Pulsed magnetic field from video display terminals enhances teratogenic effects of cytosine arabinoside in mice.

Recent advances in research on radiofrequency fields and health.

Reproductive and teratologic effects of electromagnetic fields.

Risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields: a population based study.

Search for teratogenic risks with the aid of malformation registries.

Some effects of exposure of the Japanese quail embryo to 2.45-GHz microwave radiation.

Teratogenic effects of sinusoidal extremely low frequency electromagnetic fields on morphology of 24 hr chick embryos.

Teratology, survival, and reversal learning after fetal irradiation of mice by 2450-MHz microwave energy.

The effects of ionizing radiation, microwaves, and ultrasound on the developing embryo: clinical interpretations and applications of the data.

The influence of electromagnetic radiation generated by a mobile phone on the skeletal system of rats.

VDT pulse magnetic field enhances teratogenic effect of ara-c in mice].

Video display terminal use during pregnancy and reproductive outcome--a meta-analysis.

Video display terminals: risk of electromagnetic radiation.

FACTOR 3B

Theme – Mammary tumors

Key MeSH Headings - Fibroadenoma, Adenoma, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma

Titles

A histopathological study on alterations in DMBA-induced mammary carcinogenesis in rats with 50 Hz, 100 muT magnetic field exposure.

Acceleration of mammary tumorigenesis by exposure of 7,12-dimethylbenz[a]anthracene-treated female rats in a 50-Hz, 100-microT magnetic field: replication study.

Bioelectromagnetic field effects on cancer cells and mice tumors.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves.

Do cocarcinogenic effects of ELF electromagnetic fields require repeated long-term interaction with carcinogens? Characteristics of positive studies using the DMBA breast cancer model in rats.

Effect of 13 week magnetic field exposures on DMBA-initiated mammary gland carcinomas in female Sprague-Dawley rats.

Effect of 26 week magnetic field exposures in a DMBA initiation-promotion mammary gland model in Sprague-Dawley rats.

Effect of a 9 mT pulsed magnetic field on C3H/Bi female mice with mammary carcinoma. A comparison between the 12 Hz and the 460 Hz frequencies.

Effects of 50- or 60-hertz, 100 microT magnetic field exposure in the DMBA mammary cancer model in Sprague-Dawley rats: possible explanations for different results from two laboratories.

Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats.

Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats.

Effects of magnetic fields on mammary tumor development induced by 7,12-dimethylbenz(a)anthracene in rats.

Effects of mobile-phone microwave on dimethylbenz (a) anthracene induced mammary carcinoma development in rats].

Effects of weak alternating magnetic fields on nocturnal melatonin production and mammary carcinogenesis in rats.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Evaluation of the potential carcinogenicity of 60 Hz linear sinusoidal continuous-wave magnetic fields in Fischer F344 rats.

Low-frequency electromagnetic radiation enhances the induction of rat mammary tumors by nitrosomethyl urea.

Magnetic fields and mammary cancer in rodents: a critical review and evaluation of published literature.

Male breast tumors in railway engine drivers: investigation of 5 cases].

Microwave absorption by normal and tumor cells.

Modifying effect of light and electromagnetic field on development of mammary tumors induced by N-nitrosomethyl urea in female rats].

Non dietetic environmental risk factors in prostate cancer].

Occupational exposure to magnetic fields in relation to male breast cancer and testicular cancer: a Swedish case-control study.

On the role of the interactions of ions with external magnetic fields in physiologic processes and their importance in chronobiology.

Repeated exposure of C3H/HeJ mice to ultra-wideband electromagnetic pulses: lack of effects on mammary tumors.

Significant differences in the effects of magnetic field exposure on 7,12-dimethylbenz(a)anthracene-induced mammary carcinogenesis in two substrains of Sprague-Dawley rats.

Study on potential effects of "902-MHz GSM-type Wireless Communication Signals" on DMBA-induced mammary tumours in Sprague-Dawley rats.

The effect of low-frequency electromagnetic fields on the development of experimental mammary tumors].

Transferrin receptors and natural killer cell lysis. A study using Colo 205 cells exposed to 60 Hz electromagnetic fields.

FACTOR 4

Theme – Male infertility

Key MeSH Headings - Sperm Count, Spermatozoa, Sperm Motility, Semen, Testis, Infertility, Male, Spermatogenesis, Testosterone, Fertility

Titles

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

2.45 GHz microwave radiation induced oxidative and nitrosative stress mediated testicular apoptosis: Involvement of a p53 dependent bax-caspase-3 mediated pathway.

2.45-GHz microwave irradiation adversely affects reproductive function in male mouse, Mus musculus by inducing oxidative and nitrosative stress.

900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues.

A 50-Hz electromagnetic field impairs sleep.

Abnormal physical architecture of the lipophilic domains of human sperm membrane in oligospermia: a logical cause for low fertility profiles.

Action of UHF microwaves on the germ and somatic cells of mammals].

Activation of TLR signalling regulates microwave radiation-mediated impairment of spermatogenesis in rat testis.

Acute, whole-body microwave exposure and testicular function of rats.

Adolescent in-school cellphone habits: a census of rules, survey of their effectiveness, and fertility implications.

Alternating magnetic field damages the reproductive function of murine testes].

An evaluation of the effects of long-term cell phone use on the testes via light and electron microscope analysis.

An ultrastructural analysis of the testes in mice subjected to long-term exposure to a 17-kHz electrical field].

Analysis of Gene Expression in Mice Testes Exposed to 1.765 GHz Microwave in Utero.

Are men talking their reproductive health away?

Association between mobile phone use and semen quality: a systemic review and meta-analysis.

Biologic effects of prolonged exposure to ELF electromagnetic fields in rats: II. 50 Hz magnetic fields.

Biological and morphological effects on the reproductive organ of rats after exposure to electromagnetic field.

Biological effects of non-ionizing electromagnetic fields: Two sides of a coin.

Biophysical evaluation of radiofrequency electromagnetic field effects on male reproductive pattern.

Cell phones and male infertility: a review of recent innovations in technology and consequences.

Cell phones and male infertility: dissecting the relationship.

Cell phones: modern man's nemesis?

Cellphone electromagnetic radiation damages the testicular ultrastructure of male rats].

Challenging cell phone impact on reproduction: a review.

Changes of rat testicular germ cell apoptosis after high power microwave radiation].

Chronotoxicity of 1800 MHz microwave radiation on sex hormones and spermatogenesis in male mice].

Combined effects of traffic and electromagnetic fields on the immune system of fertile atopic women.

Combined effects of varicocele and cell phones on semen and hormonal parameters.

Comparative effectiveness of different tests to determine the mutagenicity of certain factors in mammals. II. Frequency of anomalous sperm head in mice exposed to different factors].

Comparison of native and microwave irradiated DNA.

Congenital anomalies in the offspring of rats after exposure of the testis to an electrostatic field.

Cytogenetic effects of microwave irradiation on male germ cells of the mouse.

Cytokines produced by microwave-radiated Sertoli cells interfere with spermatogenesis in rat testis.

DNA damage, cell kinetics and ODC activities studied in CBA mice exposed to electromagnetic fields generated by transmission lines.

Does exposure to computers affect the routine parameters of semen quality?

Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce DNA damage in various tissues of rats?

Does static electric field from ultra-high voltage direct-current transmission lines affect male reproductive capacity? Evidence from a laboratory study on male mice.

Dominant lethal studies in male mice after exposure to 2.45 GHz microwave radiation.

Dominant lethal studies in male mice after exposure to a 50 Hz magnetic field.

Dominant lethal studies in male mice after exposure to a 50-Hz electric field.

Dosimetry for a study of effects of 2.45-GHz microwaves on mouse testis.

Effect of 2.45 GHz microwave radiation on the fertility pattern in male mice.

Effect of 2450 MHz microwaves on the fertility of Swiss female mice].

Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study.

Effect of discontinuous short-wave electromagnetic field irradiation on the state of the endocrine glands].

Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.

Effect of Electromagnetic Waves from Mobile Phones on Spermatogenesis in the Era of 4G-LTE.

Effect of Guilingji Capsule on the fertility, liver functions, and serum LDH of male SD rats exposed by 900 mhz cell phone].

Effect of long-term exposure of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on testes functions.

Effect of low power microwave on the mouse genome: a direct DNA analysis.

Effect of low-intensity extremely high frequency radiation on reproductive function in wistar rats.

Effect of mobile telephones on sperm quality: a systematic review and meta-analysis.

Effect of Modified Wuzi Yanzong Pill () on Tip60-Mediated Apoptosis in Testis of Male Rats after Microwave Radiation.

Effect of rosmarinic acid on sertoli cells apoptosis and serum antioxidant levels in rats after exposure to electromagnetic fields.

Effect of whole-body 1800MHz GSM-like microwave exposure on testicular steroidogenesis and histology in mice.

Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats.

Effects of 2.45 GHz CW microwave radiation on embryofetal development in mice.

Effects of 2.45 GHz microwave radiation and heat on mouse spermatogenic epithelium.

Effects of 2.45 GHz microwaves on meiotic chromosomes of male CBA/CAY mice.

Effects of 60 Hz electromagnetic field exposure on testicular germ cell apoptosis in mice.

Effects of a unique electromagnetic field system on the fertility of rats.

Effects of cellular phone emissions on sperm motility in rats.

Effects of electromagnetic fields exposure on plasma hormonal and inflammatory pathway biomarkers in male workers of a power plant.

Effects of electromagnetic fields on fecundity in the chicken.

Effects of electromagnetic fields on the reproductive success of American kestrels.

Effects of electromagnetic pulses on apoptosis and TGF-beta3 expression of mouse testis tissue].

Effects of electromagnetic radiation from a cellular phone on human sperm motility: an in vitro study.

Effects of electromagnetic radiation on morphology and TGF-beta3 expression in mouse testicular tissue.

Effects of electromagnetic waves emitted from 3G+wi-fi modems on human semen analysis.

Effects of exposure to a mobile phone on sexual behavior in adult male rabbit: an observational study.

Effects of exposure to a mobile phone on testicular function and structure in adult rabbit.

Effects of exposure to electromagnetic field (1.8/0.9 GHz) on testicular function and structure in growing rats.

Effects of extremely low-frequency electromagnetic fields (ELF-EMF) exposure on B6C3F1 mice.

Effects of GSM-like radiofrequency irradiation during the oogenesis and spermiogenesis of Xenopus laevis.

Effects of high power microwave on the expressions of Bcl-2 and C-myc proteins in the rat testis].

Effects of microwaves (950 MHZ mobile phone) on morphometric and apoptotic changes of rabbit epididymis.

Effects of mobile phone radiation on serum testosterone in Wistar albino rats.

Effects of radiofrequency electromagnetic fields (UMTS) on reproduction and development of mice: a multi-generation study.

Effects of radiofrequency electromagnetic fields on mammalian spermatogenesis].

Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats.

Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study.

Effects of the exposure to mobile phones on male reproduction: a review of the literature.

Effects of whole-body 50-Hz magnetic field exposure on mouse Leydig cells.

Effects on rat testis of 1.95-GHz W-CDMA for IMT-2000 cellular phones.

Electric power, pineal function, and the risk of breast cancer.

Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.

Environmental risk factors in the history of male patients of an infertility clinic.

Evaluation of changes in electrophysiological and hormonal parameters in rabbits resulting from short-term low-intensity ultra-high-frequency irradiation].

Evaluation of testicular degeneration induced by low-frequency electromagnetic fields.

Evaluation of the effect of using mobile phones on male fertility.

Evidence for mobile phone radiation exposure effects on reproductive pattern of male rats: role of ROS.

Examination of electric field effects on tissues by using back propagation neural network.

Exercise testing in the evaluation of human responses to powerline frequency fields.

Experimental research on the biological action of the pulse-modulated microwave radiation created by shipboard radar stations].

Exposure to a 900 MHz electromagnetic field for 1 hour a day over 30 days does change the histopathology and biochemistry of the rat testis.

Exposure to magnetic fields and the risk of poor sperm quality.

Exposure to non-ionizing electromagnetic radiation of public risk prevention instruments threatens the quality of spermatozoids.

Extremely low frequency electromagnetic field exposure affects fertilization outcome in swine animal model.

Extremely low-frequency magnetic fields can impair spermatogenesis recovery after reversible testicular damage induced by heat.

Flow cytometric analysis of the effects of 50 Hz magnetic fields on mouse spermatogenesis].

Germ cell degeneration in normal and microwave-irradiated rats: potential sperm production rates at different developmental steps in spermatogenesis.

Growing concern over the safety of using mobile phones and male fertility.

Habits of cell phone usage and sperm quality - does it warrant attention?

Health problems among workers of iron welding machines: an effect of electromagnetic fields.

Histological and cytological examination of rat reproductive tissue after short-time intermittent radiofrequency exposure.

How does long term exposure to base stations and mobile phones affect human hormone profiles?

Human disease resulting from exposure to electromagnetic fields.

Hygienic standardization of electromagnetic radiation from two-channel meteorological radar stations].

Hypospermatogenesis and spermatozoa maturation arrest in rats induced by mobile phone radiation.

Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices.

Immunomorphologic changes in the testes upon exposure to a microwave electromagnetic field].

Impact of 2.45 GHz microwave radiation on the testicular inflammatory pathway biomarkers in young rats: The role of gallic acid.

Impact of cell phone radiation on male reproduction].

Impact of cell phone use on men's semen parameters.

Impact of microwave at X-band in the aetiology of male infertility.

Impact of mobile phone radiation on the quality and DNA methylation of human sperm in vitro].

Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline.

In vitro effect of pulsed 900 MHz GSM radiation on mitochondrial membrane potential and motility of human spermatozoa.

In vitro effects of radiofrequency electromagnetic waves on bovine spermatozoa motility.

In vitro fertilization of mouse ova by spermatozoa exposed isothermally to radio-frequency radiation.

Influence of a 50 hz extra low frequency electromagnetic field on spermatozoa motility and fertilization rates in rabbits.

Influence of electromagnetic fields emitted by GSM-900 cellular telephones on the circadian patterns of gonadal, adrenal and pituitary hormones in men.

Influence of electromagnetic fields on reproductive system of male rats.

Influence of in vitro microwave radiation on the fertilizing capacity of turkey sperm.

Influence of microwave exposure on fertility of male rats.

Influence of radiofrequency-electromagnetic waves from 3rd-generation cellular phones on fertilization and embryo development in mice.

Inhibition by Egb761 of the effect of cellphone radiation on the male reproductive system.

Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure.

Interaction of microwave radiation with turkey sperm.

Is there a relationship between cell phone use and semen quality?

Long-term effects of 900 MHz radiofrequency radiation emitted from mobile phone on testicular tissue and epididymal semen quality.

Long-term exposure of male and female mice to 50 Hz magnetic field: effects on fertility.

Long-term exposure to low intensity microwave radiation affects male reproductivity].

Long-term microwave radiation affects male reproduction in rats].

Low frequency electromagnetic waves increase human sperm motility - A pilot study revealing the potent effect of 43 kHz radiation.

Mechanisms of biological effects of radiofrequency electromagnetic fields: an overview.

Melatonin attenuates radiofrequency radiation (900 MHz)-induced oxidative stress, DNA damage and cell cycle arrest in germ cells of male Swiss albino mice.

Metabolic and ultrastructural adaptation mechanisms during the primary prophylactic action of low-intensity electromagnetic radiation under normal and radiation conditions].

Microwave emissions from police radar.

Microwave exposure affecting reproductive system in male rats.

Microwave radiation decreases the expressions of occludin and JAM-1 in rats].

Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro.

Mobile phone usage and male infertility in Wistar rats.

Morinda officialis how extract improves microwave-induced reproductive impairment in male rats].

Multigeneration reproductive toxicity assessment of 60-Hz magnetic fields using a continuous breeding protocol in rats.

Occupational exposures obtained by questionnaire in clinical practice and their association with semen quality.

Occupational hazards for the male reproductive system.

Occupational influences on male fertility and sexuality. I.

Oxidative effects of extremely low frequency magnetic field and radio frequency radiation on testes tissues of diabetic and healthy rats.

Oxidative stress-mediated alterations on sperm parameters in male Wistar rats exposed to 3G mobile phone radiation.

PARAMETERS OF SPERMATOGENESIS IN MEN EXPOSED TO DIFFICULT ENVIRONMENTS].

Pathological study of testicular injury induced by high power microwave radiation in rats].

Poly ADP ribosylation as a possible mechanism of microwave--biointeraction.

Prospective study of pregnancy outcomes after parental cell phone exposure: the Norwegian Mother and Child Cohort Study.

Protective effect of Liuweidihuang Pills against cellphone electromagnetic radiation-induced histomorphological abnormality, oxidative injury, and cell apoptosis in rat testes].

Protective effects of luteolin on rat testis following exposure to 900 MHz electromagnetic field.

Proteomic analysis of continuous 900-MHz radiofrequency electromagnetic field exposure in testicular tissue: a rat model of human cell phone exposure.

Pulsed or continuous electromagnetic field induce p53/p21-mediated apoptotic signaling pathway in mouse spermatogenic cells in vitro and thus may affect male fertility.

Quantitative changes in testicular structure and function in rat exposed to mobile phone radiation.

Radar radiation damages sperm quality].

Radiations and male fertility.

Radio frequency electromagnetic radiation (RF-EMR) from GSM (0.9/1.8GHz) mobile phones induces oxidative stress and reduces sperm motility in rats.

Radiofrequency electromagnetic fields; male infertility and sex ratio of offspring.

Radiofrequency electromagnetic radiation from cell phone causes defective testicular function in male Wistar rats.

Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice.

Rat fertility and embryo fetal development: influence of exposure to the Wi-Fi signal.

Reaction of Reproductive System and Epididymal Spermatozoa .of Rats to Electromagnetic Radiation from Mobile Phone (1745 MHz) of Various Duration].

Recent reports of Wi-Fi and mobile phone-induced radiation on oxidative stress and reproductive signaling pathways in females and males.

Reproduction in male Japanese quail exposed to microwave radiation during embryogeny.

Reproductive hazards among workers at high voltage substations.

Response of Caenorhabditis elegans to wireless devices radiation exposure.

Response of the seminiferous epithelium of the mouse exposed to low dose high energy (HZE) and electromagnetic radiation.

Scientometric study of the effects of exposure to non-ionizing electromagnetic fields on fertility: A contribution to understanding the reasons of partial failure.

Self-reported mobile phone use and semen parameters among men from a fertility clinic.

Semen analysis of military personnel associated with military duty assignments.

Sperm count and sperm abnormality in male mice after exposure to 2.45 GHz microwave radiation.

State of the reproductive systemin in male rats of 1st generation obtained from irradiated parents and exposed to electromagnetic radiation (897 MHz) during embryogenesis and postnatal development].

Status quo of the researches on the biological effect of electromagnetic radiation on the testis and epididymal sperm].

Structural and ultrastructural study of rat testes influenced by electromagnetic radiation.

Studies of the induction of dominant lethals and translocations in male mice after chronic exposure to microwave radiation.

Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. II. Postnatal psychophysiologic evaluations.

Study of bioeffects of ship-borne microwave navigation radar in chronic experiments].

Testicular apoptosis and histopathological changes induced by a 2.45 GHz electromagnetic field.

Testicular development evaluation in rats exposed to 60 Hz and 1 mT electromagnetic field.

Testicular function of rats following exposure to microwave radiation.

Tests of mutagenesis and reproduction in male rats exposed to 2,450-MHz (CW) microwaves.

The biological effects of radiofrequency radiation: a critical review and recommendations.

The combined action of drinking mineral water and low-intensity electromagnetic radiation under the immobilization stress conditions (an experimental study)].

The effect of acute far field exposure at 2.45 GHz on the mouse testis.

The effect of alternating electric field of industrial frequency on testicles of white mice].

The effect of low-intensity prolonged impulse electromagnetic irradiation in the UHF range on the testes and the appendages of the testis in rats].

The effect of male occupational exposure in infertile couples in Norway.

The effect of prenatal exposure to 900-MHz electromagnetic field on the 21-old-day rat testicle.

The effect of pulsed 900-MHz GSM mobile phone radiation on the acrosome reaction, head morphometry and zona binding of human spermatozoa.

The effects of an electromagnetic field on the boundary tissue of the seminiferous tubules of the rat: A light and transmission electron microscope study.

The effects of electromagnetic waves emitted by the cell phones on the testicular tissue.

The effects of extremely low frequency electromagnetic field exposure on the pH of the adult male semen and the motoricity parameters of spermatozoa in vitro].

The Effects of Melatonin on Oxidative Stress Parameters and DNA Fragmentation in Testicular Tissue of Rats Exposed to Microwave Radiation.

The effects of radiofrequency electromagnetic radiation on sperm function.

The effects of simultaneous combined exposure to CDMA and WCDMA electromagnetic fields on rat testicular function.

The genomic effects of cell phone exposure on the reproductive system.

The influence of electromagnetic radiation of industrial frequency on Daphnia magna (Straus)].

The influence of ultrasound and constant magnetic field on gametes, zygotes, and embryos of the sea urchin].

The interaction of changes in the genitalia in the pathogenesis of sterility in men].

The mobile phone decreases fructose but not citrate in rabbit semen: a longitudinal study.

The semen quality of the mobile phone users.

The specific features of the development of metabolic and regenerative processes under the action of low-intensity electromagnetic radiation in radiation exposure conditions (an experimental study)].

The therapeutic effect of a pulsed electromagnetic field on the reproductive patterns of male Wistar rats exposed to a 2.45-GHz microwave field.

The use of FDTD in establishing in vitro experimentation conditions representative of lifelike cell phone radiation on the spermatozoa.

Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats.

Whole-body microwave exposure emitted by cellular phones and testicular function of rats.

Wi-Fi (2.45 GHz)- and mobile phone (900 and 1800 MHz)-induced risks on oxidative stress and elements in kidney and testis of rats during pregnancy and the development of offspring.

FACTOR 5

Theme – Brain neoplasms

Key MeSH Headings - Meningioma, Glioma, Meningeal Neoplasms, Neuroma, Acoustic, Brain Neoplasms, Glioblastoma, Neoplasms, Radiation-Induced, Neuroma, Cranial Nerve Neoplasms, Parotid Neoplasms, Central Nervous System Neoplasms

Titles

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The problem of hygienic standardization of commercial electric and magnetic fields in Russia and other countries].

The question of health effects from exposure to electromagnetic fields.

The role of chemical and physical factors in cancer development].

The sensitivity of children to electromagnetic fields.

Thermal and non-thermal health effects of low intensity non-ionizing radiation: An international perspective.

Time trend in incidence of malignant neoplasms of the central nervous system in relation to mobile phone use among young people in Japan.

Time trends (1998-2007) in brain cancer incidence rates in relation to mobile phone use in England.

Trends in incidence of primary brain cancer in New Zealand, 1995 to 2010.

Uncertainty in the relation between exposure to magnetic fields and brain cancer due to assessment and assignment of exposure and analytical methods in dose-response modeling.

Use of cellular and cordless telephones and risk of testicular cancer.

Use of cellular telephones and brain tumour risk in urban and rural areas.

Use of cellular telephones and risk of cancer. A Danish cohort study].

Use of cellular telephones and the risk for brain tumours: A case-control study.

Use of mobile and cordless phones and survival of patients with glioma.

Use of mobile phones and cancer risk.

Use of mobile phones and risk of brain tumours: update of Danish cohort study.

Use of mobile phones in Norway and risk of intracranial tumours.

Use of wireless phones and the risk of salivary gland tumours: a case-control study.

Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones.

Validation of self-reported start year of mobile phone use in a Swedish case-control study on radiofrequency fields and acoustic neuroma risk.

Variation in cancer risk estimates for exposure to powerline frequency electromagnetic fields: a meta-analysis comparing EMF measurement methods.

Vestibular schwannoma and cell-phones. Results, limits and perspectives of clinical studies.

Wire codes, magnetic fields, and childhood cancer.

Wireless Phone Use and Risk of Adult Glioma: Evidence from a Meta-Analysis.

World Health Organization, radiofrequency radiation and health - a hard nut to crack (Review).

FACTOR 6

Theme – Sensory disorders

Key MeSH Headings - Burning Mouth Syndrome, Taste Disorders, Skin Diseases, Mouth Diseases, Dizziness, Vision Disorders, Hypersensitivity, Delayed, Fatigue

Titles

A method for in vivo detection of abnormal subepidermal tissues based on dielectric properties.

A survey study on some neurological symptoms and sensations experienced by long term users of mobile phones.

Adverse cutaneous effects of ionizing and non-ionizing electromagnetic radiation.

Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Bedtime mobile phone use and sleep in adults.

Can exposure to a terrestrial trunked radio (TETRA)-like signal cause symptoms? A randomised double-blind provocation study.

Cardiac devices and electromagnetic interference revisited: new radiofrequency technologies and implications for dermatologic surgery.

Description of persons with symptoms presumed to be caused by electricity or visual display units--oral aspects.

Effect of millimeter waves on cyclophosphamide induced suppression of the immune system.

Effect of stress and intesity of mobile phone using on the health and subjective symptoms in GSM workers].

Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication--a literature review published between 2000 and 2004.

Environmental illness: fatigue and cholinesterase activity in patients reporting hypersensitivity to electricity.

Health response of two communities to military antennae in Cyprus.

Health status of the workers exposed to strong, constant magnetic fields].

Human exposure to 4.0-Tesla magnetic fields in a whole-body scanner.

Immune function and host defense in rodents exposed to 60-Hz magnetic fields.

Immunomorphologic changes in the testes upon exposure to a microwave electromagnetic field].

Interference with cardiac pacemakers by cellular telephones.

Microwave sickness: a reappraisal.

Mobile communication: radiobiology problems and evaluation of danger].

Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones.

Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir.

Neurobehavioral effects among inhabitants around mobile phone base stations.

Non-specific physical symptoms and electromagnetic field exposure in the general population: can we get more specific? A systematic review.

Odontologic survey of referred patients with symptoms allegedly caused by electricity or visual display units.

Provocation with stress and electricity of patients with "sensitivity to electricity".

Psychologic aspects of patients with symptoms presumed to be caused by electricity or visual display units.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

Some ocular symptoms experienced by users of mobile phones.

Specific patterns of weak (1 microTesla) transcerebral complex magnetic fields differentially affect depression, fatigue, and confusion in normal volunteers.

Study of human neurovegetative and hematologic effects of environmental low-frequency (50-Hz) electromagnetic fields produced by transformers.

Subjective symptoms related to mobile phone use--a pilot study].

Symptoms experienced by people in vicinity of base stations: II/ Incidences of age, duration of exposure, location of subjects in relation to the antennas and other electromagnetic factors].

Symptoms of ill health ascribed to electromagnetic field exposure--a questionnaire survey.

Symptoms, personality traits, and stress in people with mobile phone-related symptoms and electromagnetic hypersensitivity.

The effects of cell phone use on peripheral vision.

The effects of multivitamin supplementation on mood and general well-being in healthy young adults. A laboratory and at-home mobile phone assessment.

The risk of subjective symptoms in mobile phone users in Poland--an epidemiological study.

Video display terminals: risk of electromagnetic radiation.

FACTOR 7

Theme – Breast neoplasms

Key MeSH Headings - Carcinoma, Lobular, Carcinoma, Ductal, Breast, Breast Neoplasms, Male, Adenoma

Titles

A cluster of male breast cancer in office workers.

A meta-analysis of epidemiologic studies of electric and magnetic fields and breast cancer in women and men.

Breast cancer, occupation, and exposure to electromagnetic fields among Swedish men.

Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art].

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Effect of 13 week magnetic field exposures on DMBA-initiated mammary gland carcinomas in female Sprague-Dawley rats.

Electromagnetic field exposure and male breast cancer risk: a meta-analysis of 18 studies.

Epidemiology and aetiological factors of male breast cancer: a ten years retrospective study in eastern Turkey.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Magnetic fields and breast cancer in Swedish adults residing near high-voltage power lines.

Magnetic fields and mammary cancer in rodents: a critical review and evaluation of published literature.

Male breast tumors in railway engine drivers: investigation of 5 cases].

Occupational exposure to magnetic fields in relation to male breast cancer and testicular cancer: a Swedish case-control study.

Risk for leukaemia and brain and breast cancer among Danish utility workers: a second follow-up.

The relationship between electromagnetic field and light exposures to melatonin and breast cancer risk: a review of the relevant literature.

FACTOR 8

Theme – Oxidative stress

Key MeSH Headings - Oxidative Stress, Malondialdehyde, Glutathione Peroxidase, Lipid Peroxidation, Reactive Oxygen Species, Apoptosis, DNA Damage, Nitric Oxide, Protein Carbonylation

Titles

14.6 mT ELF magnetic field exposure yields no DNA breaks in model system Salmonella, but provides evidence of heat stress protection.

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

2.1 GHz electromagnetic field does not change contractility and intracellular Ca2+ transients but decreases beta-adrenergic responsiveness through nitric oxide signaling in rat ventricular myocytes.

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

2.45 GHz Microwave Radiation Impairs Learning and Spatial Memory via Oxidative/Nitrosative Stress Induced p53-Dependent/Independent Hippocampal Apoptosis: Molecular Basis and Underlying Mechanism.

2.45 GHz microwave radiation induced oxidative and nitrosative stress mediated testicular apoptosis: Involvement of a p53 dependent bax-caspase-3 mediated pathway.

2.45-GHz microwave irradiation adversely affects reproductive function in male mouse, Mus musculus by inducing oxidative and nitrosative stress.

2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic Ca(2)(+) influx in human leukemia cancer cells.

50 Hz extremely low frequency electromagnetic fields enhance protein carbonyl groups content in cancer cells: effects on proteasomal systems.

50-Hertz electromagnetic fields induce gammaH2AX foci formation in mouse preimplantation embryos in vitro.

8-Oxo-7, 8-dihydro-2'-deoxyguanosine as a biomarker of DNA damage by mobile phone radiation.

8-oxoG DNA glycosylase-1 inhibition sensitizes Neuro-2a cells to oxidative DNA base damage induced by 900 MHz radiofrequency electromagnetic radiation.

900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues.

900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: protection by vitamins E and C.

900-MHz microwave radiation enhances gamma-ray adverse effects on SHG44 cells.

900-MHz microwave radiation promotes oxidation in rat brain.

915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons.

A 700 MHz 1H-NMR study reveals apoptosis-like behavior in human K562 erythroleukemic cells exposed to a 50 Hz sinusoidal magnetic field.

A comparative analysis of the biological action of microwaves and laser radiation].

A critical review of the genotoxic potential of electric and magnetic fields.

A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.

A cross-sectional study on oxidative stress in workers exposed to extremely low frequency electromagnetic fields.

A histopathological and biochemical evaluation of oxidative injury in the sciatic nerves of male rats exposed to a continuous 900-megahertz electromagnetic field throughout all periods of adolescence.

Acute exposure to 930 MHz CW electromagnetic radiation in vitro affects reactive oxygen species level in rat lymphocytes treated by iron ions.

Acute exposure to a 60 Hz magnetic field increases DNA strand breaks in rat brain cells.

Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells.

Adaptive response in mice exposed to 900 MHZ radiofrequency fields: bleomycin-induced DNA and oxidative damage/repair.

Adaptive response in mice exposed to 900 MHz radiofrequency fields: primary DNA damage.

Adaptive response in mouse bone-marrow stromal cells exposed to 900-MHz radiofrequency fields: Gamma-radiation-induced DNA strand breaks and repair.

Adverse and beneficial effects in Chinese hamster lung fibroblast cells following radiofrequency exposure.

Age diseases depending on geomagnetic field activity inside the womb period].

Age-related effects on induction of DNA strand breaks by intermittent exposure to electromagnetic fields.

An evaluation of genotoxicity in human neuronal-type cells subjected to oxidative stress under an extremely low frequency pulsed magnetic field.

An in vitro study of the effects of exposure to a GSM signal in two human cell lines: monocytic U937 and neuroblastoma SK-N-SH.

Analysis of proto-oncogene and heat-shock protein gene expression in human derived cell-lines exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field.

Antioxidants alleviate electric field-induced effects on lung tissue based on assays of heme oxygenase-1, protein carbonyl content, malondialdehyde, nitric oxide, and hydroxyproline.

Anxiety-like behavioural effects of extremely low-frequency electromagnetic field in rats.

Apoptosis in haemopoietic progenitor cells exposed to extremely low-frequency magnetic fields.

Apoptosis induced by microwave radiation in pancreatic cancer JF305 cells.

Apoptosis induced by ultraviolet radiation is enhanced by amplitude modulated radiofrequency radiation in mutant yeast cells.

Apoptosis is induced by radiofrequency fields through the caspase-independent mitochondrial pathway in cortical neurons.

Apoptosis of Lewis Lung Carcinoma Cells Induced by Microwave via p53 and Proapoptotic Proteins In vivo.

Apoptotic cell death during Drosophila oogenesis is differentially increased by electromagnetic radiation depending on modulation, intensity and duration of exposure.

Assessment of biological changes of continuous whole body exposure to static magnetic field and extremely low frequency electromagnetic fields in mice.

Assessment of cytogenetic damage and oxidative stress in personnel occupationally exposed to the pulsed microwave radiation of marine radar equipment.

Assessment of DNA sensitivity in peripheral blood leukocytes after occupational exposure to microwave radiation: the alkaline comet assay and chromatid breakage assay.

Assessment of genetic damage in peripheral blood of human volunteers exposed (whole-body) to a 200 muT, 60 Hz magnetic field.

Assessment of genotoxicity and genomic instability in rat primary astrocytes exposed to 872 MHz radiofrequency radiation and chemicals.

Assessment of nuclear abnormalities in exfoliated cells from the oral epithelium of mobile phone users.

Assessment of oxidant/antioxidant status in saliva of cell phone users.

Association of microwaves and ionizing radiation: potentiation of teratogenic effects in the rat.

Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 4. Manifestation of oxidative intracellular stress-reaction after long-term non-thermal EMF exposure of rats].

Biochemical and histological studies on adverse effects of mobile phone radiation on rat's brain.

Biochemical and pathological changes in the male rat kidney and bladder following exposure to continuous 900-MHz electromagnetic field on postnatal days 22-59<sup/>.

Biochemical modifications and neuronal damage in brain of young and adult rats after long-term exposure to mobile phone radiations.

Bioeffects of microwave--a brief review.

Bioelectromagnetic field effects on cancer cells and mice tumors.

Biological effects of non-ionizing electromagnetic fields: Two sides of a coin.

Biological responses of mobile phone frequency exposure.

Biological stress responses to radio frequency electromagnetic radiation: are mobile phones really so (heat) shocking?

Biophysical evaluation of radiofrequency electromagnetic field effects on male reproductive pattern.

Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields].

Calreticulin protects rat microvascular endothelial cells against microwave radiation-induced injury by attenuating endoplasmic reticulum stress.

Cell oxidation-reduction imbalance after modulated radiofrequency radiation.

Cell phone electromagnetic field radiations affect rhizogenesis through impairment of biochemical processes.

Cell phone radiation exposure on brain and associated biological systems.

Cell phones and cancer: what is the evidence for a connection?

Cell type-specific genotoxic effects of intermittent extremely low-frequency electromagnetic fields.

Cellphone electromagnetic radiation damages the testicular ultrastructure of male rats].

Cellular effects of electromagnetic fields.

Changes in antioxidant capacity of blood due to mutual action of electromagnetic field (1800 MHz) and opioid drug (tramadol) in animal model of persistent inflammatory state.

Changes of apoptosis, mitochondrion membrane potential and Ca2+ of hypothalamic neurons induced by high power microwave].

Changes of rat testicular germ cell apoptosis after high power microwave radiation].

Chromosomal damage in human diploid fibroblasts by intermittent exposure to extremely low-frequency electromagnetic fields.

Chronic exposure to 50Hz magnetic fields causes a significant weakening of antioxidant defence systems in aged rat brain.

Cognitive impairment and neurogenotoxic effects in rats exposed to low-intensity microwave radiation.

Combinative exposure effect of radio frequency signals from CDMA mobile phones and aphidicolin on DNA integrity.

Combined effects of 872 MHz radiofrequency radiation and ferrous chloride on reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells.

Combined exposure of ELF magnetic fields and x-rays increased mutant yields compared with x-rays alone in pTN89 plasmids.

Comments on "Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes" by Schwarz et al. (Int Arch Occup Environ Health 2008: doi: 10.1007/s00420-008-0305-5).

Comparative study of cell cycle kinetics and induction of apoptosis or necrosis after exposure of human Mono Mac 6 cells to radiofrequency radiation.

Comparison of biological effects between continuous and intermittent exposure to GSM-900-MHz mobile phone radiation: Detection of apoptotic cell-death features.

Comparison of cytotoxic and genotoxic effects of plutonium-239 alpha particles and mobile phone GSM 900 radiation in the Allium cepa test.

Correction of microcirculatory disturbances with terahertz electromagnetic radiation at nitric oxide frequencies in albino rats under conditions of acute stress.

Corrective effects of electromagnetic radiation in a millimeter wavelength range on the parameters of oxidative stress after standard anti-helicobacterial therapy in patients with ulcer disease].

Critical comments on DNA breakage by mobile-phone electromagnetic fields [Diem et al., Mutat. Res. 583 (2005) 178-183].

Cytogenetic effects of 18.0 and 16.5 GHz microwave radiation on human lymphocytes in vitro.

Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C.

Cytogenetic studies in human blood lymphocytes exposed in vitro to 2.45 GHz or 8.2 GHz radiofrequency radiation.

Cytogenetic studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using R-banded karyotyping.

Cytokines produced by microwave-radiated Sertoli cells interfere with spermatogenesis in rat testis.

Cytotoxic and genotoxic effect in RTG-2 cell line exposed to selected biocides used in the disinfection of cooling towers.

Cytotoxic and genotoxic effects of high-frequency electromagnetic fields (GSM 1800 MHz) on immature and mature rats.

Decreased DNA repair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure.

Dependence of microwave effect on the secondary structure of DNA on molecular weight of polynucleotide].

Developmental effects of perinatal exposure to extremely weak 7 Hz magnetic fields and nitric oxide modulation in the Wistar albino rat.

Different methods for evaluating the effects of microwave radiation exposure on the nervous system.

Disordered redox metabolism of brain cells in rats exposed to low doses of ionizing radiation or UHF electromagnetic radiation.

DNA and chromosomal damage in response to intermittent extremely low-frequency magnetic fields.

DNA damage and repair induced by acute exposure of microwave from mobile phone on cultured human lens epithelial cells].

DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia.

DNA damage induced in brain cells of CBA mice exposed to magnetic fields.

DNA damage, cell kinetics and ODC activities studied in CBA mice exposed to electromagnetic fields generated by transmission lines.

Do extremely low frequency magnetic fields enhance the effects of environmental carcinogens? A meta-analysis of experimental studies.

Do naturally occurring magnetic nanoparticles in the human body mediate increased risk of childhood leukaemia with EMF exposure?

Does MW Radiation Affect Gene Expression, Apoptotic Level, and Cell Cycle Progression of Human SH-SY5Y Neuroblastoma Cells?

Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce DNA damage in various tissues of rats?

Drosophila oogenesis as a bio-marker responding to EMF sources.

Effect of 0.2 T static magnetic field on human neurons: remodeling and inhibition of signal transduction without genome instability.

Effect of 2.45 GHz microwave radiation on the fertility pattern in male mice.

Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the hsp27/p38MAPK stress pathway in rat brain.

Effect of 7 mT static magnetic field and iron ions on rat lymphocytes: apoptosis, necrosis and free radical processes.

Effect of 900 MHz radio frequency radiation on beta amyloid protein, protein carbonyl, and malondialdehyde in the brain.

Effect of 900 MHz radiofrequency radiation on oxidative stress in rat brain and serum.

Effect of 900-, 1800-, and 2100-MHz radiofrequency radiation on DNA and oxidative stress in brain.

Effect of 950 MHz UHF electromagnetic radiation on biomarkers of oxidative damage, metabolism of UFA and antioxidants in the livers of young rats of different ages.

Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro].

Effect of American Ginseng Capsule on the liver oxidative injury and the Nrf2 protein expression in rats exposed by electromagnetic radiation of frequency of cell phone].

Effect of coexposure to 50 Hz magnetic fields and an aneugen on human lymphocytes, determined by the cytokinesis block micronucleus assay.

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Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1)--in vitro researches].

Effect of electromagnetic microwave radiation on the growth of Ehrlich ascites carcinoma.

Effect of exposure and withdrawal of 900-MHz-electromagnetic waves on brain, kidney and liver oxidative stress and some biochemical parameters in male rats.

Effect of exposure to the edge signal on oxidative stress in brain cell models.

Effect of extremely low frequency magnetic field on antioxidant activity in plasma and red blood cells in spot welders.

Effect of extremely low-frequency electromagnetic fields on antioxidant activity in the human keratinocyte cell line NCTC 2544.

Effect of GSTM1 and GSTT1 Polymorphisms on Genetic Damage in Humans Populations Exposed to Radiation From Mobile Towers.

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Effect of intermittent and continuous exposure to electromagnetic fields on cultured hippocampal cells.

Effect of long-term power frequency electromagnetic field exposure on proliferation and apoptosis of SRA01/04 cells].

Effect of low level microwave radiation exposure on cognitive function and oxidative stress in rats.

Effect of Low Level Subchronic Microwave Radiation on Rat Brain.

Effect of microwave radiation on primary cultured Sertoli cells].

Effect of mobile phone exposure on apoptotic glial cells and status of oxidative stress in rat brain.

Effect of Mobile Phone Radiation on Cardiovascular Development of Chick Embryo.

Effect of mobile phones on micronucleus frequency in human exfoliated oral mucosal cells.

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Effect of radiofrequency radiation on reproductive health.

Effect of rosmarinic acid on sertoli cells apoptosis and serum antioxidant levels in rats after exposure to electromagnetic fields.

Effect of selenium pre-treatment on plasma antioxidant vitamins A (retinol) and E (alpha-tocopherol) in static magnetic field-exposed rats.

Effect of static magnetic field on development toxicity of rat embryonic midbrain neurons cells].

Effect of superposed electromagnetic noise on DNA damage of lens epithelial cells induced by microwave radiation.

Effects of 10-GHz microwaves on hematological parameters in Swiss albino mice and their modulation by Prunus avium.

Effects of 1800 MHz RF-EMF exposure on DNA damage and cellular functions in primary cultured neurogenic cells.

Effects of 2,450 MHz microwave on DNA damage induced by three chemical mutagens in vitro].

Effects of 2.45 GHz electromagnetic fields with a wide range of SARs on bacterial and HPRT gene mutations.

Effects of 2.45 GHz microwave exposures on the peroxidation status in Wistar rats.

Effects of 50 Hz electromagnetic fields on the histology, apoptosis, and expression of c-Fos and beta-catenin on the livers of preincubated white Leghorn chicken embryos.

Effects of 50-Hz magnetic field exposure on hormone secretion and apoptosis-related gene expression in human first trimester villous trophoblasts in vitro.

Effects of 60 Hz electromagnetic field exposure on testicular germ cell apoptosis in mice.

Effects of 60-Hz fields, estradiol and xenoestrogens on human breast cancer cells.

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Effects of cell phone radiation on lipid peroxidation, glutathione and nitric oxide levels in mouse brain during epileptic seizure.

Effects of centimeter waves on the immune system of mice in endotoxic shock].

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Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits.

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Effects of electromagnetic radiation on RAF/MEK/ERK signaling pathway in rats hippocampus].

Effects of electromagnetic radiation produced by 3G mobile phones on rat brains: magnetic resonance spectroscopy, biochemical, and histopathological evaluation.

Effects of exposure to 2100MHz GSM-like radiofrequency electromagnetic field on auditory system of rats.

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FACTOR 9

Theme – Neurodegenerative diseases

Key MeSH Headings - Parkinson Disease, Neurodegenerative Diseases, Alzheimer Disease, Amyotrophic Lateral Sclerosis, Motor Neuron Disease, Occupational Diseases, Dementia, Brain Diseases, Dementia, Vascular

Titles

5-HT contents change in peripheral blood of workers exposed to microwave and high frequency radiation].

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Evaluation of occupational risk caused by exposure to electromagnetic rays].

Evaluation of various psychologic parameters in a group of workers occupationally exposed to radiofrequency].

Evaluation of vital activity of workers with obliterating diseases of lower extremities servicing electric transmission lines].

Exposure from occupational versus other sources.

Exposure to electromagnetic fields and risk of central nervous system diseases among employees at Danish electric companies].

Exposure to extremely-low-frequency electromagnetic fields and radiofrequency radiation: cardiovascular effects in humans.

Exposure to low-frequency electromagnetic fields--a health hazard?

Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County.

Exposure to VHF and UHF electromagnetic fields among workers employed in radio and TV broadcast centers. I. Assessment of exposure].

Exposure, health complaints and cognitive performance among employees of an MRI scanners manufacturing department.

Fifty Hertz electromagnetic field exposure stimulates secretion of beta-amyloid peptide in cultured human neuroglioma.

Follow-up of radio and telegraph operators with exposure to electromagnetic fields and risk of breast cancer.

Future needs of occupational epidemiology of extremely low frequency electric and magnetic fields: review and recommendations.

Health Effects of Electromagnetic Fields on Reproductive-Age Female Operators of Plastic Welding Machines in Fuzhou, China.

Health effects of electromagnetic fields].

Health effects relevant to the setting of EMF exposure limits.

Health of workers exposed to electric fields.

Health problems among operators of plastic welding machines and exposure to radiofrequency electromagnetic fields.

Health state and performance of operators in electric discharge facilities--sources of electromagnetic impulses].

HEALTH STATUS OF ELECTROTECHNICAL PERSONNEL EXPOSED TO THE COMBINED IMPACT OF ELECTROMAGNETIC FIELDS OF 50 HZ AND CHEMICALS].

Health status of railway workers using magnetic powder flaw detectors].

Health status of the workers exposed to strong, constant magnetic fields].

Hygienic assessment of working conditions and functional resistance in electric power station workers].

HYGIENIC ASSESSMENT OF WORKING CONDITIONS OF EMPLOYEES OF BROADCASTING CENTER].

Hygienic evaluation of work conditions for shielded compartments staff].

Hygienic optimization of the use of chemical protective means on railway transport].

Idiopathic environmental intolerance: 2 disabling entities to recognize].

Impact of electromagnetic fields on a computer user].

Incidence of breast cancer in a Norwegian cohort of women with potential workplace exposure to 50 Hz magnetic fields.

Incidence of cancer among workers in Norwegian hydroelectric power companies.

Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields.

Incidence of cancer in persons with occupational exposure to electromagnetic fields in Denmark.

Incidence of leukaemia and brain tumours in some "electrical occupations".

Increased incidence of cancer in a cohort of office workers exposed to strong magnetic fields.

Influence of power frequency electric and magnetic fields on human health.

Interactions between occupational exposure to extremely low frequency magnetic fields and chemicals for brain tumour risk in the INTEROCC study.

Interactive effect of chemical substances and occupational electromagnetic field exposure on the risk of gliomas and meningiomas in Swedish men.

Leukaemia, brain tumours and exposure to extremely low frequency magnetic fields: cohort study of Swiss railway employees.

Leukemia and occupational exposure to electromagnetic fields: review of epidemiologic surveys.

Leukemia following occupational exposure to 60-Hz electric and magnetic fields among Ontario electric utility workers.

Leukemia in electric utility workers: the evaluation of alternative indices of exposure to 60 Hz electric and magnetic fields.

Leukemia in telephone linemen.

Long term and excessive use of 900 MHz radiofrequency radiation alter microRNA expression in brain.

Low frequency electromagnetic fields in the working environment--exposure and health effects. Elevated risk of cancer, reproductive hazards or other unwanted health effects?].

Low-back pain among electric power supply workers and their attitude toward its prevention and the treatment.

Magnetic field exposure and neurodegenerative disease mortality among electric utility workers.

Magnetic field exposure and neurodegenerative diseases--recent epidemiological studies.

Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers.

Magnetic field exposure related to cancer subtypes.

Magnetic field on the deranged accommodation of visual detector terminal operators].

Magnetic fields and brain tumour risks in UK electricity supply workers.

Magnetic fields and leukaemia risks in UK electricity supply workers.

Magnetic fields exposure from high-voltage power lines and risk of amyotrophic lateral sclerosis in two Italian populations.

Male breast tumors in railway engine drivers: investigation of 5 cases].

Microwave antigen retrieval of beta-amyloid precursor protein immunoreactivity.

Microwave sickness: a reappraisal.

Mobile phone use and brain tumours in the CERENAT case-control study.

Modern concepts and methodology of means, methods of protection, and safety measures for servicemen affected by nonionizing radiation].

Mortality among workers in the geothermal power plants at Larderello, Italy.

Mortality from amyotrophic lateral sclerosis, other chronic disorders, and electric shocks among utility workers.

Mortality from brain cancer and leukaemia among electrical workers.

Mortality from neurodegenerative disease and exposure to extremely low-frequency magnetic fields: 31 years of observations on Swiss railway employees.

Mortality of personnel operating electric power objects with 500 kV voltage].

Myeloid leukemias and myelodysplastic syndromes: chemical exposure, histologic subtype and cytogenetics in a case-control study.

Neurodegenerative disease and magnetic field exposure in UK electricity supply workers.

Neurodegenerative diseases in welders and other workers exposed to high levels of magnetic fields.

Neurodegenerative diseases, suicide and depressive symptoms in relation to EMF.

Neurological effects of microwave exposure related to mobile communication.

Neurological effects of radiofrequency radiation.

News in occupational cancers].

Non-Hodgkin's lymphoma among electric utility workers in Ontario: the evaluation of alternate indices of exposure to 60 Hz electric and magnetic fields.

Non-ionizing radiation exposure causing ill-health and alopecia areata.

Novelties in hygienic evaluation of electromagnetic conditions on computerized workplaces].

Occupation and malignant lymphoma: a population based case control study in Germany.

Occupational assessment of computer placement in school areas].

Occupational electromagnetic field exposures associated with sleep quality: a cross-sectional study.

Occupational exposure and amyotrophic lateral sclerosis in a prospective cohort.

Occupational exposure of healthcare and research staff to static magnetic stray fields from 1.5-7 Tesla MRI scanners is associated with reporting of transient symptoms.

Occupational exposure to electromagnetic field and breast cancer risk in a large, population-based, case-control study in the United States.

Occupational exposure to electromagnetic fields and Alzheimer disease.

Occupational exposure to electromagnetic fields and its health effects in electric energy workers].

Occupational exposure to electromagnetic fields and sex-differential risk of uveal melanoma.

Occupational exposure to electromagnetic fields and the occurrence of brain tumors. An analysis of possible associations.

Occupational exposure to electromagnetic fields of extremely low frequency (with particular regard to power plants) and the health status of workers, based on a literature review].

Occupational exposure to extremely low frequency electric and magnetic fields and Alzheimer disease: a meta-analysis.

Occupational exposure to extremely low frequency magnetic fields and risk of Alzheimer disease: A systematic review and meta-analysis.

Occupational exposure to ionizing radiation and electromagnetic fields in relation to the risk of thyroid cancer in Sweden.

Occupational exposure to low frequency magnetic fields and dementia: a case-control study.

Occupational exposure to low frequency magnetic fields and the risk of low grade and high grade glioma.

Occupational exposure to magnetic fields in case-referent studies of neurodegenerative diseases.

Occupational exposure to non-ionizing radiation and an association with heart disease: an exploratory study.

Occupational exposure to physical agents: the new Italian database for risk assessment and control.

Occupational exposure to power frequency magnetic fields and risk of non-Hodgkin lymphoma.

Occupational exposures and brain cancer mortality: a preliminary study of east Texas residents.

Occupational Exposures and Neurodegenerative Diseases-A Systematic Literature Review and Meta-Analyses.

Occupational exposures and the risk of amyotrophic lateral sclerosis.

Occupational exposures obtained by questionnaire in clinical practice and their association with semen quality.

Occupational exposures to extremely low frequency magnetic fields and postmenopausal breast cancer.

Occupational factors of anxiety and depressive disorders in the French National Electricity and Gas Company. The Anxiety-Depression Group.

Occupational hazards for the male reproductive system.

Occupational health evaluation of electromagnetic fields in electric trains and subway technologic areas].

Occupational magnetic field exposure and neurodegenerative disease.

Occupational magnetic field exposure and site-specific cancer incidence: a Swedish cohort study.

Occupational risk and its prophylaxis for female workers engaged in radio-electronic instrument industry].

Occupational risk factors for acute leukaemia: a case-control study.

Occupational risk factors for cancer of the central nervous system: a case-control study on death certificates from 24 U.S. states.

Occupational risk factors in Alzheimer's disease: a review assessing the quality of published epidemiological studies.

Occupational risks in grocery stores].

Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer's disease.

Ocular medical surveillance on microwave and laser workers.

On prevention of a combined impact of electromagnetic radiation and climatic/weather factors on worker's organism].

On prevention of electromagnetic rays effects in workers exposed to extreme climate conditions].

On the microwave exposure.

Optimization of methods for measurement and assessment of occupational exposure to electromagnetic fields in physiotherapy (SW diathermy)].

Overview of epidemiologic research on electric and magnetic fields and cancer.

Perspectives on health effects of electric and magnetic fields.

Physical factors and stress].

Physicians appeals on the dangers of mobile communication--what is the evidence? Assessment of public health data.

Prevalence of depression among electrical workers.

Prevalence of musculoskeletal disorders and related occupational causative factors among electricity linemen: A narrative review.

Problem of studying influence of electric and magnetic fields on human health. Results and prospects].

Provocation of the electromagnetic distress syndrome.

Radiofrequency (RF) sickness in the Lilienfeld Study: an effect of modulated microwaves?

Radiofrequency electromagnetic fields; male infertility and sex ratio of offspring.

Radiofrequency fields, transthyretin, and Alzheimer's disease.

Relationship between amyloid beta protein and melatonin metabolite in a study of electric utility workers.

Relationships between occupational history and serum concentrations of organochlorine compounds in exocrine pancreatic cancer.

Remote effects of occupational and non-occupational exposure to electromagnetic fields of power-line frequency. Epidemiological studies].

Reports on electromagnetic field strength measurements issued for occupational health and safety needs in the opinion of radio communication station users].

Residence near power lines and mortality from neurodegenerative diseases: longitudinal study of the Swiss population.

Residential and occupational exposures to 50-Hz magnetic fields and breast cancer in women: a population-based study.

Residential distance to high-voltage power lines and risk of neurodegenerative diseases: a Danish population-based case-control study.

Review of the epidemiologic literature on EMF and Health.

Risk agents related to work and amyotrophic lateral sclerosis: An occupational medicine focus.

Risk factors for Alzheimer disease: a population-based case-control study in Istanbul, Turkey.

Risk factors, health risks, and risk management for aircraft personnel and frequent flyers.

Risk for leukaemia and brain and breast cancer among Danish utility workers: a second follow-up.

Risk of severe cardiac arrhythmia in male utility workers: a nationwide danish cohort study.

Searching for the perfect wave: the effect of radiofrequency electromagnetic fields on cells.

Setting prudent public health policy for electromagnetic field exposures.

Socioeconomic status, social mobility and cancer occurrence during working life: a case-control study among French electricity and gas workers.

State of peripheral blood of technical personnel exposed to constant magnetic fields].

Symptoms of the musculoskeletal system and exposure to magnetic fields in an aluminium plant.

Systematic analysis of the state of man exposed to radio wave irradiation for a long time].

The effect of various occupational exposures to microwave radiation on the concentrations of immunoglobulins and T lymphocyte subsets].

The evaluation of the consequences of electromagnetic irradiation of hands in operators of high-frequency welding devices].

The evaluation of the exposure of seamstresses to electromagnetic fields, emitted by sewing machines].

The health problems of computer operators].

The possible role of radiofrequency radiation in the development of uveal melanoma.

The potential hazard for the development of leukemia from exposure to electromagnetic radiation (a review of the literature)].

The psychosocial work environment and skin symptoms among visual display terminal workers: a case referent study.

The strategy of targetted health surveillance. II. Genetically determined susceptibility to chemical substances and other issues related to health surveillance.

Trends in nonionizing electomagnetic radiation bioeffects research and related occupational health aspects.

Various psychological parameters in subjects occupationally exposed to radiofrequencies].

Work environment and cardiovascular diseases. A short review of the literature.

Work related etiology of amyotrophic lateral sclerosis (ALS): a meta-analysis.

FACTOR 10

Theme - Cerebrovascular disorders

Key MeSH Headings - Cerebrovascular Disorders, Dementia, Migraine Disorders, Tinnitus, Headache, Sleep Wake Disorders, Carotid Artery Diseases, Alzheimer Disease, Dementia, Vascular

Titles

A 50-Hz electromagnetic field impairs sleep.

A case-control study on the risk factors of Alzheimer's disease in military elderly men].

A literature review of medical side effects from radio-frequency energy in the human environment: involving cancer, tumors, and problems of the central nervous system.

A study on the biological effects of exposure mobile-phone frequency EMF].

A survey study on some neurological symptoms and sensations experienced by long term users of mobile phones.

Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study.

Association between overuse of mobile phones on quality of sleep and general health among occupational health and safety students.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Association of tinnitus and electromagnetic hypersensitivity: hints for a shared pathophysiology?

Cell phones: modern man's nemesis?

Clinical features of headache associated with mobile phone use: a cross-sectional study in university students.

Cohort study on the effects of everyday life radio frequency electromagnetic field exposure on non-specific symptoms and tinnitus.

Dementia and occupational exposure to magnetic fields.

Do mobile phone base stations affect sleep of residents? Results from an experimental double-blind sham-controlled field study.

Effect of stress and intesity of mobile phone using on the health and subjective symptoms in GSM workers].

Effects of 60 Hz electromagnetic field exposure on APP695 transcription levels in differentiating human neuroblastoma cells.

Effects of Millimeter-Wave Electromagnetic Radiation on the Experimental Model of Migraine.

Effects of Sleep Quality on the Association between Problematic Mobile Phone Use and Mental Health Symptoms in Chinese College Students.

Electrical occupations and neurodegenerative disease: analysis of U.S. mortality data.

Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication--a literature review published between 2000 and 2004.

Electromagnetic pulse exposure induces overexpression of beta amyloid protein in rats.

Elevated risk of Alzheimer's disease among workers with likely electromagnetic field exposure.

Exposure to electromagnetic fields and risk of central nervous system diseases among employees at Danish electric companies].

Fifty Hertz electromagnetic field exposure stimulates secretion of beta-amyloid peptide in cultured human neuroglioma.

Headache and sferics.

Health of workers exposed to electric fields.

Individual variation in temporal relationships between exposure to radiofrequency electromagnetic fields and non-specific physical symptoms: A new approach in studying 'electrosensitivity'.

Investigation of sleep disorders in the vicinity of high frequency transmitters].

Long term and excessive use of 900 MHz radiofrequency radiation alter microRNA expression in brain.

Long-term and frequent cellular phone use and risk of acoustic neuroma.

Magnetic field exposure and neurodegenerative disease mortality among electric utility workers.

Magnetic field exposure and neurodegenerative diseases--recent epidemiological studies.

Microwave antigen retrieval of beta-amyloid precursor protein immunoreactivity.

Microwave sickness: a reappraisal.

Mobile communication: radiobiology problems and evaluation of danger].

Mobile phone headache: a double blind, sham-controlled provocation study.

Mobile phone use and health symptoms in children.

Mobile phone use and stress, sleep disturbances, and symptoms of depression among young adults--a prospective cohort study.

Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones.

Mobile Phone Use and The Risk of Headache: A Systematic Review and Meta-analysis of Cross-sectional Studies.

Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir.

Natural very-low-frequency sferics and headache.

Neurobehavioral effects among inhabitants around mobile phone base stations.

Neurodegenerative diseases in welders and other workers exposed to high levels of magnetic fields.

Neurodegenerative diseases, suicide and depressive symptoms in relation to EMF.

Neurological changes induced by a mobile phone.

Non-specific physical symptoms and electromagnetic field exposure in the general population: can we get more specific? A systematic review.

Occupational electromagnetic field exposures associated with sleep quality: a cross-sectional study.

Occupational exposure to electromagnetic fields and Alzheimer disease.

Occupational exposure to extremely low frequency electric and magnetic fields and Alzheimer disease: a meta-analysis.

Occupational exposure to extremely low frequency magnetic fields and risk of Alzheimer disease: A systematic review and meta-analysis.

Occupational exposure to low frequency magnetic fields and dementia: a case-control study.

Occupational exposure to magnetic fields in case-referent studies of neurodegenerative diseases.

Occupational Exposures and Neurodegenerative Diseases-A Systematic Literature Review and Meta-Analyses.

Occupational magnetic field exposure and neurodegenerative disease.

Occupational risk factors in Alzheimer's disease: a review assessing the quality of published epidemiological studies.

Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer's disease.

Physicians appeals on the dangers of mobile communication--what is the evidence? Assessment of public health data.

Preliminary report: symptoms associated with mobile phone use.

Prevalence of headache among handheld cellular telephone users in Singapore: a community study.

Psychological symptoms and intermittent hypertension following acute microwave exposure.

Radio and microwave frequency radiation and health--an analysis of the literature].

Radiofrequency fields, transthyretin, and Alzheimer's disease.

Relationship between amyloid beta protein and melatonin metabolite in a study of electric utility workers.

Residence near power lines and mortality from neurodegenerative diseases: longitudinal study of the Swiss population.

Residential distance to high-voltage power lines and risk of neurodegenerative diseases: a Danish population-based case-control study.

Risk factors for Alzheimer disease: a population-based case-control study in Istanbul, Turkey.

Role of ultrasonic dopplerography in monitoring the effectiveness of treatment of patients who have sustained a stroke with decimeter-range electromagnetic waves].

Subjective symptoms related to mobile phone use--a pilot study].

Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations.

Survey of mobile phone use and their chronic effects on the hearing of a student population.

Symptom prevalence and worry about high voltage transmission lines.

Symptoms experienced by people in vicinity of base stations: II/ Incidences of age, duration of exposure, location of subjects in relation to the antennas and other electromagnetic factors].

Symptoms of ill health ascribed to electromagnetic field exposure--a questionnaire survey.

Symptoms reported by mobile cellular telephone users].

The association between use of mobile phones after lights out and sleep disturbances among Japanese adolescents: a nationwide cross-sectional survey.

The effects of 884 MHz GSM wireless communication signals on headache and other symptoms: an experimental provocation study.

The prevalence of symptoms attributed to electromagnetic field exposure: a cross-sectional representative survey in Switzerland.

The relationship between adolescents' well-being and their wireless phone use: a cross-sectional study.

The risk of subjective symptoms in mobile phone users in Poland--an epidemiological study.

The role of microwave radiometry in carotid artery disease. Diagnostic and clinical prospective.

Time-dependent hematological changes in workers exposed to electromagnetic fields.

Tinnitus and cell phones: the role of electromagnetic radiofrequency radiation.

Tinnitus and mobile phone use.

FACTOR 11

Theme - Congenital abnormalities and glandular-based tumors

Key MeSH Headings - Cleft Lip, Cleft Palate, Fibroadenoma, Adenoma, Calcification, Physiologic, Mammary Neoplasms, Animal, Mammary Neoplasms, Experimental, Adenocarcinoma

Titles

A histopathological study on alterations in DMBA-induced mammary carcinogenesis in rats with 50 Hz, 100 muT magnetic field exposure.

Acceleration of mammary tumorigenesis by exposure of 7,12-dimethylbenz[a]anthracene-treated female rats in a 50-Hz, 100-microT magnetic field: replication study.

Are microwaves a co-teratogen? Experimental model concept and its verification].

Bioelectromagnetic field effects on cancer cells and mice tumors.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves.

Do cocarcinogenic effects of ELF electromagnetic fields require repeated long-term interaction with carcinogens? Characteristics of positive studies using the DMBA breast cancer model in rats.

Effect of 13 week magnetic field exposures on DMBA-initiated mammary gland carcinomas in female Sprague-Dawley rats.

Effect of 26 week magnetic field exposures in a DMBA initiation-promotion mammary gland model in Sprague-Dawley rats.

Effect of a 9 mT pulsed magnetic field on C3H/Bi female mice with mammary carcinoma. A comparison between the 12 Hz and the 460 Hz frequencies.

Effects of 50- or 60-hertz, 100 microT magnetic field exposure in the DMBA mammary cancer model in Sprague-Dawley rats: possible explanations for different results from two laboratories.

Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats.

Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats.

Effects of magnetic fields on mammary tumor development induced by 7,12-dimethylbenz(a)anthracene in rats.

Effects of mobile-phone microwave on dimethylbenz (a) anthracene induced mammary carcinoma development in rats].

Effects of weak alternating magnetic fields on nocturnal melatonin production and mammary carcinogenesis in rats.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Evaluation of the potential carcinogenicity of 60 Hz linear sinusoidal continuous-wave magnetic fields in Fischer F344 rats.

Low-frequency electromagnetic radiation enhances the induction of rat mammary tumors by nitrosomethyl urea.

Magnetic fields and mammary cancer in rodents: a critical review and evaluation of published literature.

Male breast tumors in railway engine drivers: investigation of 5 cases].

Microwave absorption by normal and tumor cells.

Modifying effect of light and electromagnetic field on development of mammary tumors induced by N-nitrosomethyl urea in female rats].

Non dietetic environmental risk factors in prostate cancer].

Occupational exposure to magnetic fields in relation to male breast cancer and testicular cancer: a Swedish case-control study.

On the role of the interactions of ions with external magnetic fields in physiologic processes and their importance in chronobiology.

Pulsed magnetic field from video display terminals enhances teratogenic effects of cytosine arabinoside in mice.

Repeated exposure of C3H/HeJ mice to ultra-wideband electromagnetic pulses: lack of effects on mammary tumors.

Search for teratogenic risks with the aid of malformation registries.

Significant differences in the effects of magnetic field exposure on 7,12-dimethylbenz(a)anthracene-induced mammary carcinogenesis in two substrains of Sprague-Dawley rats.

Study on potential effects of "902-MHz GSM-type Wireless Communication Signals" on DMBA-induced mammary tumours in Sprague-Dawley rats.

The effect of low-frequency electromagnetic fields on the development of experimental mammary tumors].

The influence of electromagnetic radiation generated by a mobile phone on the skeletal system of rats.

Transferrin receptors and natural killer cell lysis. A study using Colo 205 cells exposed to 60 Hz electromagnetic fields.

VDT pulse magnetic field enhances teratogenic effect of ara-c in mice].

FACTOR 12

Theme – Skin neoplasms

Key MeSH Headings - Carcinoma, Basal Cell, Carcinoma, Squamous Cell, Skin Neoplasms, Cocarcinogenesis, Neoplasms, Experimental, Neoplasms, Radiation-Induced, Colonic Neoplasms

Titles

50-Hz electromagnetic environment and the incidence of childhood tumors in Stockholm County.

A case-case study of mobile phone use and acoustic neuroma risk in Japan.

A histopathological study on alterations in DMBA-induced mammary carcinogenesis in rats with 50 Hz, 100 muT magnetic field exposure.

A literature review of medical side effects from radio-frequency energy in the human environment: involving cancer, tumors, and problems of the central nervous system.

A pooled analysis of extremely low-frequency magnetic fields and childhood brain tumors.

A population-based case-control study of radiofrequency exposure in relation to childhood neoplasm.

A review of in vitro studies: low-frequency electromagnetic fields.

A study on skin tumour formation in mice with 50 Hz magnetic field exposure.

A three-dimensional point process model for the spatial distribution of disease occurrence in relation to an exposure source.

Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation.

Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study.

Adverse cutaneous effects of ionizing and non-ionizing electromagnetic radiation.

Alternative functional relationships between ELF field exposure and possible health effects: report on an expert workshop.

An epidemiological review of mobile telephones and cancer.

Animal carcinogenicity studies on radiofrequency fields related to mobile phones and base stations.

Application criteria of the precautionary principle].

Assessing the potential carcinogenic activity of magnetic fields using animal models.

Assessment of cellular telephone and other radio frequency exposure for epidemiologic research.

Association between radiation from mobile phones and tumour risk in adults].

Bcl-2 and p53 immunoprofile in Kaposi's sarcoma.

Bioeffects of electromagnetic fields--safety limits of each frequency band, especially less than radio one].

Biological effects from electromagnetic field exposure and public exposure standards.

Biological effects of amplitude-modulated radiofrequency radiation.

Biological effects of electromagnetic fields and radiation.

Biological effects of extremely low-frequency electromagnetic fields: in vivo studies.

Biological effects on human health due to radiofrequency/microwave exposure: a synopsis of cohort studies.

Biological indicators in response to radiofrequency/microwave exposure.

Biological interactions and potential health effects of extremely-low-frequency magnetic fields from power lines and other common sources.

Biological responses to electromagnetic fields.

Biomarkers of induced electromagnetic field and cancer.

Biophysical estimation of the environmental importance of electromagnetic fields.

Biophysical mechanisms of electromagnetic fields interaction and health effects].

Brain tumor risk in offspring of men occupationally exposed to electric and magnetic fields.

Cancer in radar technicians exposed to radiofrequency/microwave radiation: sentinel episodes.

Cancer incidence among welders: possible effects of exposure to extremely low frequency electromagnetic radiation (ELF) and to welding fumes.

Cancer incidence and magnetic field exposure in industries using resistance welding in Sweden.

Cancer incidence in California flight attendants (United States).

Cancer incidence vs. FM radio transmitter density.

Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation.

Cancer promotion in a mouse-skin model by a 60-Hz magnetic field: I. Experimental design and exposure system.

Cancer promotion in a mouse-skin model by a 60-Hz magnetic field: II. Tumor development and immune response.

Cancer risks related to low-level RF/MW exposures, including cell phones.

Cancer versus FM radio polarization types.

Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art].

Carcinogenicity study of 217 Hz pulsed 900 MHz electromagnetic fields in Pim1 transgenic mice.

Carcinogenicity study of GSM and DCS wireless communication signals in B6C3F1 mice.

Carcinogenicity test in B6C3F1 mice after parental and prenatal exposure to 50 Hz magnetic fields.

Carcinogenicity test of 50 Hz sinusoidal magnetic fields in rats.

Case-control study on uveal melanoma (RIFA): rational and design.

Cell phone radiation exposure on brain and associated biological systems.

Cell phones and cancer: what is the evidence for a connection?

Cellular and cordless telephone use and the association with brain tumors in different age groups.

Childhood cancer and magnetic fields from high-voltage power lines in England and Wales: a case-control study.

Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study.

Childhood cancer in relation to indicators of magnetic fields from ground current sources.

Childhood cancer occurrence in relation to power line configurations: a study of potential selection bias in case-control studies.

Children's health and RF EMF exposure. Views from a risk assessment and risk communication perspective.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves.

Comparative health risk assessment of electromagnetic fields.

Concern that "EMF" magnetic fields from power lines cause cancer.

Danger of cellular telephones and their relay stations].

Delayed biological effect of electromagnetic fields action].

Do cocarcinogenic effects of ELF electromagnetic fields require repeated long-term interaction with carcinogens? Characteristics of positive studies using the DMBA breast cancer model in rats.

Do people understand IARC's 2B categorization of RF fields from cell phones?

Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea.

Effect of 13 week magnetic field exposures on DMBA-initiated mammary gland carcinomas in female Sprague-Dawley rats.

Effect of 26 week magnetic field exposures in a DMBA initiation-promotion mammary gland model in Sprague-Dawley rats.

Effect of magnetic field exposure on anchorage-independent growth of a promoter-sensitive mouse epidermal cell line (JB6).

Effect of radiofrequency radiation exposure on mouse skin tumorigenesis initiated by 7,12-dimethybenz[alpha]anthracene.

Effects of 2.45-GHz microwave radiation and phorbol ester 12-O-tetradecanoylphorbol-13-acetate on dimethylhydrazine-induced colon cancer in mice.

Effects of 2450 MHz electromagnetic fields with a wide range of SARs on methylcholanthrene-induced transformation in C3H10T1/2 cells.

Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats.

Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats.

Effects of low level microwave radiation on carcinogenesis in Swiss Albino mice.

Effects of mobile phone radiation on UV-induced skin tumourigenesis in ornithine decarboxylase transgenic and non-transgenic mice.

Electric blanket or mattress cover use and breast cancer incidence in women 50-79 years of age.

Electric Blanket Use and Risk of Thyroid Cancer in the Women's Health Initiative Observational Cohort.

Electrical field exposure and human health. Risk assessment and problems relative to bureaucratic procedures and to the role of instituitional organizations in control and prevention].

Electromagnetic fields and cancer risks.

Electromagnetic fields and cancer: the cost of doing nothing.

Electromagnetic fields and cells.

Electromagnetic fields and female breast cancer.

Electromagnetic fields and health effects--epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease.

Electromagnetic fields and public health.

Electromagnetic fields at mobile phone frequency induce apoptosis and inactivation of the multi-chaperone complex in human epidermoid cancer cells.

Electromagnetic fields of mobile telephone systems--thresholds, effects and risks for cochlear implant patients and healthy people].

Electromagnetic fields--effects on health].

Electromagnetic fields: a cancer promoter?

Electromagnetic radiations and cancer. Cause and prevention.

Electromagnetic-field exposure and cancer.

EMF and current cancer concepts.

EMF and health.

Epidemiologic evidence on mobile phones and tumor risk: a review.

Epidemiological studies of human exposures to radiofrequency radiation. A critical review.

Epidemiological studies of radio frequency exposures and human cancer.

Epidemiological study of power lines and childhood cancer in the UK: further analyses.

Estimates of Environmental Exposure to Radiofrequency Electromagnetic Fields and Risk of Lymphoma Subtypes.

Estimating exposure in studies of residential magnetic fields and cancer: importance of short-term variability, time interval between diagnosis and measurement, and distance to power line.

Evaluation of carcinogenic effects of electromagnetic fields (EMF).

Evaluation of potential confounders in planning a study of occupational magnetic field exposure and female breast cancer.

Evaluation of residential exposure to intermediate frequency magnetic fields.

Evaluation of the effects of electric and magnetic fields in humans].

Evaluation of the potential carcinogenicity of 60 Hz linear sinusoidal continuous-wave magnetic fields in Fischer F344 rats.

Evaluation of the potential in vitro antiproliferative effects of millimeter waves at some therapeutic frequencies on RPMI 7932 human skin malignant melanoma cells.

Evidence for microwave carcinogenesis in vitro.

Experimental data on radiofrequency].

Exposure to low electromagnetic fields and the carcinogenesis process].

Exposure to low-intensive superhigh frequency electromagnetic field as a factor of carcinogenesis in experimental animals.

Exposure to power-frequency magnetic fields and the risk of childhood cancer. UK Childhood Cancer Study Investigators.

Exposure to radio-frequency electromagnetic fields from broadcast transmitters and risk of childhood cancer: a census-based cohort study.

Extremely low frequency electromagnetic fields (EMF) and brain cancer in adults and children: review and comment.

Extremely low-frequency electromagnetic fields exposure and female breast cancer risk: a meta-analysis based on 24,338 cases and 60,628 controls.

Follow-up of radio and telegraph operators with exposure to electromagnetic fields and risk of breast cancer.

Further aspects on cellular and cordless telephones and brain tumours.

Future needs of occupational epidemiology of extremely low frequency electric and magnetic fields: review and recommendations.

Genetic, carcinogenic and teratogenic effects of radiofrequency fields.

GSM and DCS wireless communication signals: combined chronic toxicity/carcinogenicity study in the Wistar rat.

Health effects of microwave exposures: a review of the recent (1995-1998) literature.

Health risks from the use of mobile phones.

Health risks of electric and magnetic fields caused by high-voltage systems in Finland.

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Health risks of mobile phones].

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Incorporation of epidemiological findings into radiation protection standards.

Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies.

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Influence of extremely-low-frequency magnetic field on antioxidative melatonin properties in AT478 murine squamous cell carcinoma culture.

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Leukemia, brain tumors, and exposure to extremely low frequency electromagnetic fields in Swiss railway employees.

Long-term use of cellular phones and brain tumours: increased risk associated with use for > or =10 years.

Long-term, low-level microwave irradiation of rats.

Lost in laterality: interpreting "preferred side of the head during mobile phone use and risk of brain tumour" associations.

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Low-level exposure to radiofrequency electromagnetic fields: health effects and research needs.

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Magnetic fields and childhood cancer--a pooled analysis of two Scandinavian studies.

Magnetic fields and childhood cancer: an epidemiological investigation of the effects of high-voltage underground cables.

Magnetic fields of high voltage power lines and risk of cancer in Finnish adults: nationwide cohort study.

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Mobile phone base stations and early childhood cancers: case-control study.

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Mobile phone use and acoustic neuroma risk in Japan.

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Mobile phones and brain tumours: a review of epidemiological research.

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Mobile phones, cordless phones and the risk for brain tumours.

Mobile phones, mobile phone base stations and cancer: a review.

Mortality in workers exposed to electromagnetic fields.

Mortality indices for hemoblastoses in Rivno Province before and after the accident at the Chernobyl Atomic Electric Power Station].

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Nonionizing electromagnetic fields and cancer: a review.

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Possible health hazards from exposure to power-frequency electric and magnetic fields--a COMAR Technical Information Statement.

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Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: a nested case-control study.

Radio and microwave frequency radiation and health--an analysis of the literature].

Radio frequency electromagnetic fields: cancer, mutagenesis, and genotoxicity.

Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer.

Radiofrequency and microwave radiation in the microelectronics industry.

Radiofrequency electromagnetic fields emitted from base stations of DECT cordless phones and the risk of glioma and meningioma (Interphone Study Group, Germany).

Radiofrequency exposure and mammalian cell toxicity, genotoxicity, and transformation.

Radiofrequency field exposure and cancer: what do the laboratory studies suggest?

Radiofrequency-induced carcinogenesis: cellular calcium homeostasis changes as a triggering factor.

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Reanalysis of risks of childhood leukaemia with distance from overhead power lines in the UK.

Recent advances in research on radiofrequency fields and health: 2001-2003.

Recent advances in research on radiofrequency fields and health: 2004-2007.

Recent data from the literature on the biological and pathologic effects of electromagnetic radiation, radio waves and stray currents].

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Repeated exposure of C3H/HeJ mice to ultra-wideband electromagnetic pulses: lack of effects on mammary tumors.

Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8GHz GSM base station environmental emission.

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Risk of brain tumors from wireless phone use.

Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries.

Risk of cancer among Danish electricity workers. A cohort study].

Risk of neoplastic diseases in conditions of exposure to radio- and microwave fields--epidemiologic investigations].

Selection bias from differential residential mobility as an explanation for associations of wire codes with childhood cancer.

Self-reported electrical appliance use and risk of adult brain tumors.

Short-term exposure to 50 Hz ELF-EMF alters the cisplatin-induced oxidative response in AT478 murine squamous cell carcinoma cells.

Should the threshold limit value for power frequency (60 Hz) magnetic fields be changed? Perceptions among scientists and other risk experts.

Significant differences in the effects of magnetic field exposure on 7,12-dimethylbenz(a)anthracene-induced mammary carcinogenesis in two substrains of Sprague-Dawley rats.

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Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves.

Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats exposed to frequency-modulated microwave fields.

Studying the effects of mobile phone use on the auditory system and the central nervous system: a review of the literature and future directions.

Survival and cancer in laboratory mammals exposed to radiofrequency energy.

Systematic review of wireless phone use and brain cancer and other head tumors.

Testing electromagnetic fields for potential carcinogenic activity: a critical review of animal models.

The design, construction and calibration of a carefully controlled source for exposure of mammalian cells to extremely low-frequency electromagnetic fields.

The effect of 60-Hz magnetic fields on co-promotion of chemically induced skin tumors on SENCAR mice: a discussion of three studies.

The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats.

The effect of embryonic and fetal exposure to x-ray, microwaves, and ultrasound: counseling the pregnant and nonpregnant patient about these risks.

The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumors and other neoplasms in rats.

The effects of embryonic and fetal exposure to x-ray, microwaves, and ultrasound.

The effects of ionizing radiation, microwaves, and ultrasound on the developing embryo: clinical interpretations and applications of the data.

The effects of pulsed 860 MHz radiofrequency radiation on the promotion of neurogenic tumors in rats.

The epidemiology of electric and magnetic field exposures in the power frequency range and reproductive outcomes.

The Intracranial Distribution of Gliomas in Relation to Exposure From Mobile Phones: Analyses From the INTERPHONE Study.

The possible role of contact current in cancer risk associated with residential magnetic fields.

The possible role of radiofrequency radiation in the development of uveal melanoma.

The potential carcinogenic hazards of electromagnetic radiation: a review.

The precautionary principle and electric and magnetic fields.

The probability of developing brain tumours among users of cellular telephones (scientific information to the decision of the International Agency for Research on Cancer (IARC) announced on May 31, 2011)].

The problem of hygienic standardization of commercial electric and magnetic fields in Russia and other countries].

The question of health effects from exposure to electromagnetic fields.

The role of chemical and physical factors in cancer development].

The sensitivity of children to electromagnetic fields.

Time trend in incidence of malignant neoplasms of the central nervous system in relation to mobile phone use among young people in Japan.

Use of cellular and cordless telephones and risk of testicular cancer.

Use of cellular telephones and brain tumour risk in urban and rural areas.

Use of cellular telephones and risk of cancer. A Danish cohort study].

Use of cellular telephones and the risk for brain tumours: A case-control study.

Use of mobile phones and cancer risk.

Use of wireless phones and the risk of salivary gland tumours: a case-control study.

Variable E-cadherin expression in a MNU-induced colon tumor model in rats which exposed with 50 Hz frequency sinusoidal magnetic field.

Variation in cancer risk estimates for exposure to powerline frequency electromagnetic fields: a meta-analysis comparing EMF measurement methods.

Wire codes, magnetic fields, and childhood cancer.

World Health Organization, radiofrequency radiation and health - a hard nut to crack (Review).

FACTOR 13

Theme - Leukemia

Key MeSH Headings - Leukemia, Myeloid, Acute, Leukemia, Lymphocytic, Chronic, B-Cell, Leukemia, Myelogenous, Chronic, BCR-ABL Positive, Leukemia, Myeloid, Leukemia, Multiple Myeloma, Lymphoma, Leukemia, Radiation-Induced, Acute Disease, Liver Neoplasms, Experimental, Central Nervous System Neoplasms

Titles

60 Hertz magnetic field exposure assessment for an investigation of leukemia in telephone lineworkers.

A Bayesian approach to hazard identification. The case of electromagnetic fields and cancer.

A case-control pilot study of traffic exposures and early childhood leukemia using a geographic information system.

A case-control study of childhood leukemia in southern Ontario, Canada, and exposure to magnetic fields in residences.

A case-control study of risk of leukaemia in relation to mobile phone use.

A literature review of medical side effects from radio-frequency energy in the human environment: involving cancer, tumors, and problems of the central nervous system.

A pooled analysis of magnetic fields and childhood leukaemia.

A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia-EMF Study Group.

A population-based case-control study of radiofrequency exposure in relation to childhood neoplasm.

A precautionary public health protection strategy for the possible risk of childhood leukaemia from exposure to power frequency magnetic fields.

Acute childhood leukemias and exposure to magnetic fields generated by high voltage overhead power lines - a risk factor in Iran.

Acute effects of pulsed microwaves and 3-nitropropionic acid on neuronal ultrastructure in the rat caudate-putamen.

Acute leukaemia in workers exposed to electromagnetic fields.

Acute leukemia in electrical workers: a New Zealand case-control study.

Acute nonlymphocytic leukemia and residential exposure to power frequency magnetic fields.

Acute ocular injuries caused by 60-Ghz millimeter-wave exposure.

Adult and childhood leukemia near a high-power radio station in Rome, Italy.

Adult mortality from leukemia, brain cancer, amyotrophic lateral sclerosis and magnetic fields from power lines: a case-control study in Brazil.

Aetiology of childhood leukemia.

Aluminum, calcium ion and radiofrequency synergism in acceleration of lymphomagenesis.

An evaluation of exposure metrics in an epidemiologic study on radio and television broadcast transmitters and the risk of childhood leukemia.

An examination of underlying physical principles. The interaction of power-line electromagnetic fields with the human body.

Animal carcinogenicity studies on radiofrequency fields related to mobile phones and base stations.

Are occupational, hobby, or lifestyle exposures associated with Philadelphia chromosome positive chronic myeloid leukaemia?

Are the stray 60-Hz electromagnetic fields associated with the distribution and use of electric power a significant cause of cancer?

Assessment of cellular telephone and other radio frequency exposure for epidemiologic research.

Assessment of selection bias in the Canadian case-control study of residential magnetic field exposure and childhood leukemia.

Association of childhood cancer with residential traffic density.

Biological effects of environmental electromagnetic fields: molecular mechanisms.

Biophysical mechanisms of electromagnetic fields interaction and health effects].

Can disturbances in the atmospheric electric field created by powerline corona ions disrupt melatonin production in the pineal gland?

Cancer incidence among welders: possible effects of exposure to extremely low frequency electromagnetic radiation (ELF) and to welding fumes.

Cancer incidence and magnetic field exposure in industries using resistance welding in Sweden.

Cancer incidence and mortality and proximity to TV towers.

Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter.

Cancer incidence near radio and television transmitters in Great Britain. II. All high power transmitters.

Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art].

Carcinogenicity test of 50 Hz sinusoidal magnetic fields in rats.

Case-control study of childhood cancer and exposure to 60-Hz magnetic fields.

Case-only study of interactions between DNA repair genes (hMLH1, APEX1, MGMT, XRCC1 and XPD) and low-frequency electromagnetic fields in childhood acute leukemia.

Cell Phones and Risk of brain and acoustic nerve tumours: the French INTERPHONE case-control study].

Characterization of Children's Exposure to Extremely Low Frequency Magnetic Fields by Stochastic Modeling.

Childhood cancer and exposure to corona ions from power lines: an epidemiological test.

Childhood cancer and magnetic fields from high-voltage power lines in England and Wales: a case-control study.

Childhood cancer and residential proximity to power lines. UK Childhood Cancer Study Investigators.

Childhood cancer in relation to a modified residential wire code.

Childhood cancer in relation to indicators of magnetic fields from ground current sources.

Childhood incidence of acute lymphoblastic leukaemia and exposure to broadcast radiation in Sydney--a second look.

Childhood leukaemia and distance from power lines in California: a population-based case-control study.

Childhood leukaemia close to high-voltage power lines--the Geocap study, 2002-2007.

Childhood leukaemia in a residential area with a high-voltage power line: approach according to the Dutch Community Health Services' guideline 'Cancer Clusters'].

Childhood leukemia and electromagnetic fields: results of a population-based case-control study in Germany.

Childhood leukemia and magnetic fields in infant incubators.

Childhood leukemia and magnetic fields in Japan: a case-control study of childhood leukemia and residential power-frequency magnetic fields in Japan.

Childhood leukemia and personal monitoring of residential exposures to electric and magnetic fields in Ontario, Canada.

Childhood leukemia, electric and magnetic fields, and temporal trends.

Childhood leukemia: electric and magnetic fields as possible risk factors.

Children's exposure to magnetic fields produced by U.S. television sets used for viewing programs and playing video games.

Children's health and RF EMF exposure. Views from a risk assessment and risk communication perspective.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers.

Combined risk estimates for two German population-based case-control studies on residential magnetic fields and childhood acute leukemia.

Comparative analyses of the studies of magnetic fields and cancer in electric utility workers: studies from France, Canada, and the United States.

Comparative health risk assessment of electromagnetic fields.

Contact voltage measured in residences: implications to the association between magnetic fields and childhood leukemia.

Decreased survival for childhood leukemia in proximity to television towers.

Description of a new computer wire coding method and its application to evaluate potential control selection bias in the Savitz et al. childhood cancer study.

Designs and analyses for exploring the relationship of magnetic fields to childhood leukaemia: a pilot project for the Danish National Birth Cohort.

Determinants of power-frequency magnetic fields in residences located away from overhead power lines.

Developing policy in the face of scientific uncertainty: interpreting 0.3 microT or 0.4 microT cutpoints from EMF epidemiologic studies.

Distance from residence to power line and risk of childhood leukemia: a population-based case-control study in Denmark.

Distance to high-voltage power lines and risk of childhood leukemia--an analysis of confounding by and interaction with other potential risk factors.

Do magnetic fields cause increased risk of childhood leukemia via melatonin disruption?

Do naturally occurring magnetic nanoparticles in the human body mediate increased risk of childhood leukaemia with EMF exposure?

Do power frequency magnetic fields cause leukemia in children?

Do studies of wire code and childhood leukemia point towards or away from magnetic fields as the causal agent?

Effect of pulsed magnetic fields on leukemia-prone AKR mice. No-effect on mortality through five generations.

Effects of centimeter waves on the immune system of mice in endotoxic shock].

Effects of electromagnetic fields on health].

Effects of extremely low-frequency electromagnetic fields (ELF-EMF) exposure on B6C3F1 mice.

Effects of mobile phone type signals on calcium levels within human leukaemic T-cells (Jurkat cells).

Electric and magnetic fields (EMF): what do we know about the health effects?

Electric and magnetic fields and health outcomes--an overview.

Electric and magnetic fields at power frequencies.

Electrical field exposure and human health. Risk assessment and problems relative to bureaucratic procedures and to the role of instituitional organizations in control and prevention].

Electrical power lines and childhood leukemia: a study from Greece.

Electromagnetic field exposures and childhood cancers in New Zealand.

Electromagnetic field exposures and childhood leukaemia in New Zealand.

Electromagnetic fields (EMF): do they play a role in children's environmental health (CEH)?

Electromagnetic fields and cancer risks.

Electromagnetic fields from high-voltage installations and cancer in childhood].

Electromagnetic fields--effects on health].

Electromagnetic pollution (electrosmog)--potential hazards of our electromagnetic future].

Electrosmog as a health risk factor: sources of artificial electromagnetic fields, evaluation of health risk, prevention methods].

EMF and health.

EMFs: cutting through the controversy.

Environmental factors and childhood acute leukemias and lymphomas.

Epidemiologic evidence relevant to radar (microwave) effects.

Epidemiologic study of residential proximity to transmission lines and childhood cancer in California: description of design, epidemiologic methods and study population.

Epidemiological appraisal of studies of residential exposure to power frequency magnetic fields and adult cancers.

Epidemiological study of power lines and childhood cancer in the UK: further analyses.

Epidemiology of health effects of radiofrequency exposure.

Estimates of Environmental Exposure to Radiofrequency Electromagnetic Fields and Risk of Lymphoma Subtypes.

Estimating exposure in studies of residential magnetic fields and cancer: importance of short-term variability, time interval between diagnosis and measurement, and distance to power line.

Estimation of population attributable fractions from fitted incidence ratios and exposure survey data, with an application to electromagnetic fields and childhood leukemia.

Ethical values in the regulation of the exposure to electromagnetic fields].

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Experimental estimation of thermogenic levels of acute microwave exposure for different animal species].

Exposure of high resolution fetuses in advanced pregnant woman models at different stages of pregnancy to uniform magnetic fields at the frequency of 50 Hz.

Exposure to 50-Hz electric field and incidence of leukemia, brain tumors, and other cancers among French electric utility workers.

Exposure to electromagnetic fields and risk of leukemia.

Exposure to electromagnetic fields and the risk of leukemia.

Exposure to low frequency pulsed electromagnetic fields increases interleukin-1 and interleukin-6 production by human peripheral blood mononuclear cells.

Exposure to low-frequency electromagnetic fields--a health hazard?

Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County.

Exposure to magnetic fields and survival after diagnosis of childhood leukemia: a German cohort study.

Exposure to power frequency electric fields and the risk of childhood cancer in the UK.

Exposure to power-frequency magnetic fields and the risk of childhood cancer. UK Childhood Cancer Study Investigators.

Exposure to radio-frequency electromagnetic fields from broadcast transmitters and risk of childhood cancer: a census-based cohort study.

Exposure to residential electric and magnetic fields and risk of childhood leukemia.

Extra low frequency electric and magnetic fields in the bedplace of children diagnosed with leukaemia: a case-control study.

Extremely low frequency electromagnetic fields and cancer: the epidemiologic evidence.

Factors that explain the power line configuration wiring code-childhood leukemia association: what would they look like?

Geomagnetic field variation in early ontogenesis as a risk factor for oncopathology].

Health effects of electromagnetic fields].

Health effects of low-level electromagnetic fields: phantom or not-so-phantom risk?

Hematopoietic neoplasia in C57BL/6 mice exposed to split-dose ionizing radiation and circularly polarized 60 Hz magnetic fields.

High incidence of acute leukemia in the proximity of some industrial facilities in El Bierzo, northwestern Spain.

Hypothesis: the risk of childhood leukemia is related to combinations of power-frequency and static magnetic fields.

In vitro microwave effects on human neutrophil precursor cells (CFU-C).

Incidence of cancer in the vicinity of Korean AM radio transmitters.

Incidence of leukaemia and brain tumours in some "electrical occupations".

Incorporation of epidemiological findings into radiation protection standards.

Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies.

Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters.

Increased risk of childhood acute lymphoblastic leukemia (ALL) by prenatal and postnatal exposure to high voltage power lines: a case control study in Isfahan, Iran.

Induction of macrophage migration inhibitory factor precedes the onset of acute tonsillitis.

Infantile leukemia and exposure to 50/60 Hz magnetic fields: review of epidemiologic evidence in 2000].

Influence of 60-Hertz magnetic fields on leukemia.

Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations.

Investigation of the sources of residential power frequency magnetic field exposure in the UK Childhood Cancer Study.

Knowledge and perceptions of the health effects of environmental hazards in the general population in Italy.

Leukaemia and residence near electricity transmission equipment: a case-control study.

Leukaemia, brain tumours and exposure to extremely low frequency magnetic fields: cohort study of Swiss railway employees.

Leukemia and lymphoma incidence in rodents exposed to low-frequency magnetic fields.

Leukemia and occupational exposure to electromagnetic fields: review of epidemiologic surveys.

Leukemia following occupational exposure to 60-Hz electric and magnetic fields among Ontario electric utility workers.

Leukemia in electric utility workers: the evaluation of alternative indices of exposure to 60 Hz electric and magnetic fields.

Leukemia in telephone linemen.

Leukemia mortality and incidence of infantile leukemia near the Vatican Radio Station of Rome].

Leukemia risk and occupational electric field exposure in Los Angeles County, California.

Leukemia, brain tumors, and exposure to extremely low frequency electromagnetic fields in Swiss railway employees.

Living near overhead high voltage transmission power lines as a risk factor for childhood acute lymphoblastic leukemia: a case-control study.

Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields.

Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice.

Lymphoma induced in mice chronically exposed to very strong low-frequency electromagnetic field.

Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers.

Magnetic fields and acute leukemia in children with Down syndrome.

Magnetic fields and acute lymphoblastic leukemia in children: a systematic review of case-control studies.

Magnetic fields and childhood cancer--a pooled analysis of two Scandinavian studies.

Magnetic fields and leukaemia risks in UK electricity supply workers.

Magnetic fields and leukemia--risk for adults living close to power lines.

Magnetic fields, leukemia, and central nervous system tumors in Swedish adults residing near high-voltage power lines.

Maternal occupational exposure to electromagnetic fields before, during, and after pregnancy in relation to risks of childhood cancers: findings from the Oxford Survey of Childhood Cancers, 1953-1981 deaths.

Meta-analysis and its application in epidemiology].

Mortality among workers in the geothermal power plants at Larderello, Italy.

Mortality from brain cancer and leukaemia among electrical workers.

Mortality in workers exposed to electromagnetic fields.

Mortality indices for hemoblastoses in Rivno Province before and after the accident at the Chernobyl Atomic Electric Power Station].

Mortality of people residing near electric power supply line with voltage of 500 kV].

Mortality of persons resident in the vicinity of electricity transmission facilities.

Myelogenous leukemia and electric blanket use.

Myeloid leukemias and myelodysplastic syndromes: chemical exposure, histologic subtype and cytogenetics in a case-control study.

Nighttime exposure to electromagnetic fields and childhood leukemia: an extended pooled analysis.

Occupation and malignant lymphoma: a population based case control study in Germany.

Occupational and residential exposure to electric and magnetic field and its relationship on acute myeloid leukemia in adults - A Meta-analysis].

Occupational and residential magnetic field exposure and leukemia and central nervous system tumors.

Occupational electric and magnetic field exposure and brain cancer: a meta-analysis.

Occupational electric and magnetic field exposure and leukemia. A meta-analysis.

Occupational exposure to electromagnetic fields and acute leukaemia: analysis of a case-control study.

Occupational exposure to electromagnetic fields and its health effects in electric energy workers].

Occupational exposure to electromagnetic fields of extremely low frequency (with particular regard to power plants) and the health status of workers, based on a literature review].

Occupational magnetic field exposure and myocardial infarction incidence.

Occupational risk factors for acute leukaemia: a case-control study.

Occupational risk factors for cancer of the central nervous system: a case-control study on death certificates from 24 U.S. states.

Overhead electricity power lines and childhood leukemia: a registry-based, case-control study.

Parental occupational exposure to magnetic fields and childhood cancer (Sweden).

Pharmacological correction of the acute effects of microwave irradiation in an experiment].

Pooled analysis of recent studies on magnetic fields and childhood leukaemia.

Potential motion related bias in the worn dosimeter measurements of two childhood leukemia studies.

Power lines and the geomagnetic field.

Power-frequency electric and magnetic fields and risk of childhood leukemia in Canada.

Probing lymphoma infiltration in spleen of AKR/J mice chronically exposed to electromagnetic fields for risk assessment--toward noninvasive modeling.

Proximity to overhead power lines and childhood leukaemia: an international pooled analysis.

Radio and microwave frequency radiation and health--an analysis of the literature].

Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer.

Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems.

Reanalysis of risks of childhood leukaemia with distance from overhead power lines in the UK.

Recent data from the literature on the biological and pathologic effects of electromagnetic radiation, radio waves and stray currents].

Refinements in magnetic field exposure assignment for a case-cohort study of electrical utility workers.

Remote effects of occupational and non-occupational exposure to electromagnetic fields of power-line frequency. Epidemiological studies].

Residence close to high-tension electric power lines and its association with leukemia in children].

Residential electric consumption and childhood cancer in Canada (1971-1986)

Residential EMF exposure and childhood leukemia: meta-analysis and population attributable risk.

Residential exposure to 60-Hertz magnetic fields and adult cancers in Taiwan.

Residential exposure to electromagnetic fields and childhood leukaemia: a meta-analysis.

Residential exposure to magnetic fields and risk of canine lymphoma.

Residential magnetic fields and childhood leukemia: a meta-analysis.

Residential magnetic fields as a risk factor for childhood acute leukaemia: results from a German population-based case-control study.

Residential magnetic fields predicted from wiring configurations: I. Exposure model.

Residential magnetic fields predicted from wiring configurations: II. Relationships To childhood leukemia.

Residential magnetic fields, contact voltage and their relationship: the effects of distribution unbalance and residential proximity to a transmission line.

Residential mobility and childhood leukemia.

Residential mobility of populations near UK power lines and implications for childhood leukaemia.

Residential proximity to electricity transmission and distribution equipment and risk of childhood leukemia, childhood lymphoma, and childhood nervous system tumors: systematic review, evaluation, and meta-analysis.

Residential wire codes: reproducibility and relation with measured magnetic fields.

Review of the epidemiologic literature on EMF and Health.

Risk factors for leukemia in Thailand.

Risk for leukaemia and brain and breast cancer among Danish utility workers: a second follow-up.

Risk of childhood leukemia and environmental exposure to ELF electromagnetic fields].

Risk of childhood leukemia in areas passed by high power lines.

Risk of leukemia in children living near high-voltage transmission lines.

Risk of major lymphoma subtypes and use of mobile phones].

Risk of neoplastic diseases in conditions of exposure to power magnetic fields--epidemiologic investigations].

Risk of neoplastic diseases in conditions of exposure to radio- and microwave fields--epidemiologic investigations].

Risks of leukaemia among residents close to high voltage transmission electric lines.

Selection bias and its implications for case-control studies: a case study of magnetic field exposure and childhood leukaemia.

Setting prudent public health policy for electromagnetic field exposures.

Should the threshold limit value for power frequency (60 Hz) magnetic fields be changed? Perceptions among scientists and other risk experts.

Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves.

Study of extremely low frequency electromagnetic fields in infant incubators.

Suggestion of concomitant changes of electric power consumption and childhood leukemia in Greece.

Synthesis of the epidemiological evidence concerning childhood leukemia in relation to exposure to 50 Hz. electric and magnetic fields].

Teratogenic effect of broad-band electromagnetic field on neonatal mice (Mus musculus).

The Bernal Lecture 2004 Are low-frequency electromagnetic fields a health hazard?

The determinants of Canadian children's personal exposures to magnetic fields.

The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats.

The effects of low-energy 60-Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase.

The epidemiology of exposure to electromagnetic fields: an overview of the recent literature.

The possible role of contact current in cancer risk associated with residential magnetic fields.

The potential hazard for the development of leukemia from exposure to electromagnetic radiation (a review of the literature)].

The potential impact of bias in studies of residential exposure to magnetic fields and childhood leukemia.

The precautionary principle and electric and magnetic fields.

The sensitivity of children to electromagnetic fields.

Time trend in incidence of malignant neoplasms of the central nervous system in relation to mobile phone use among young people in Japan.

Variation in cancer risk estimates for exposure to powerline frequency electromagnetic fields: a meta-analysis comparing EMF measurement methods.

Viral contacts confound studies of childhood leukemia and high-voltage transmission lines.

Wire codes, magnetic fields, and childhood cancer.

FACTOR 14

Theme – Precancerous conditions

Key MeSH Headings - Atrophy, Precancerous Conditions, Hyperplasia, Hypersensitivity, Delayed, Thymus Gland, Capillary Permeability, Lymphoma

Titles

A histopathological study on alterations in DMBA-induced mammary carcinogenesis in rats with 50 Hz, 100 muT magnetic field exposure.

A study on skin tumour formation in mice with 50 Hz magnetic field exposure.

Aluminum, calcium ion and radiofrequency synergism in acceleration of lymphomagenesis.

Animal carcinogenicity studies on radiofrequency fields related to mobile phones and base stations.

Calreticulin protects rat microvascular endothelial cells against microwave radiation-induced injury by attenuating endoplasmic reticulum stress.

Case-control study of childhood cancer and exposure to 60-Hz magnetic fields.

Cerebrovascular permeability to 86Rb in the rat after exposure to pulsed microwaves.

Childhood cancer in relation to a modified residential wire code.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers.

Dependence of microwave effect on the secondary structure of DNA on molecular weight of polynucleotide].

Detrimental effect of electromagnetic pulse exposure on permeability of in vitro blood-brain-barrier model.

Differential response of the permeability of the rat liver canalicular membrane to sucrose and mannitol following in vivo acute single and multiple exposures to microwave radiation (2.45 GHz) and radiant-energy thermal stress.

Effect of electromagnetic pulse exposure on brain micro vascular permeability in rats.

Effect of electromagnetic radiation of millimetric wave band on genome of somatic cells].

Effect of extremely high frequency electromagnetic radiation of low intensity on parameters of humoral immunity in healthy mice].

Effect of extremely low frequency electromagnetic radiation and ultra-violet radiation on aggregation of thymocytes and erythrocytes].

Effect of global system for mobile communication (GSM) microwave exposure on blood-brain barrier permeability in rat.

Effect of global system for mobile communication (gsm)-like radiofrequency fields on vascular permeability in mouse brain.

Effect of long-term mobile communication microwave exposure on vascular permeability in mouse brain.

Effect of microwaves on the expression by thymocytes of various surface membrane markers].

Effect of millimeter waves on cyclophosphamide induced suppression of the immune system.

Effect of pulsed magnetic fields on leukemia-prone AKR mice. No-effect on mortality through five generations.

Effects of electromagnetic pulse on blood-brain barrier permeability and tight junction proteins in rats].

Effects of extremely high-frequency electromagnetic radiation on the immune system and systemic regulation of homeostasis].

Effects of GSM-modulated 900 MHz radiofrequency electromagnetic fields on the hematopoietic potential of mouse bone marrow cells.

Effects of low level microwave radiation on carcinogenesis in Swiss Albino mice.

Effects of low-intensity extremely high frequency electromagnetic radiation on chromatin structure of lymphoid cells in vivo and in vitro].

Effects of microwave radiation on thymocytes in mice at different power densities].

Electromagnetic fields from high-voltage installations and cancer in childhood].

Environmental factors and childhood acute leukemias and lymphomas.

Estimates of Environmental Exposure to Radiofrequency Electromagnetic Fields and Risk of Lymphoma Subtypes.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Geomagnetic field variation in early ontogenesis as a risk factor for oncopathology].

Hematopoietic neoplasia in C57BL/6 mice exposed to split-dose ionizing radiation and circularly polarized 60 Hz magnetic fields.

Immune function and host defense in rodents exposed to 60-Hz magnetic fields.

Immunomorphologic changes in the testes upon exposure to a microwave electromagnetic field].

Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies.

Increased sensitivity of the non-human primate eye to microwave radiation following ophthalmic drug pretreatment.

Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure.

Japanese encephalitis virus (JEV): potentiation of lethality in mice by microwave radiation.

Leukemia and lymphoma incidence in rodents exposed to low-frequency magnetic fields.

Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields.

Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice.

Lymphoma induced in mice chronically exposed to very strong low-frequency electromagnetic field.

Magnetic fields and childhood cancer--a pooled analysis of two Scandinavian studies.

Melatonin protects rat thymus against oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes.

Metabolic and ultrastructural adaptation mechanisms during the primary prophylactic action of low-intensity electromagnetic radiation under normal and radiation conditions].

Microwave alteration of the blood-brain barrier system of rats.

Microwave irradiation of rats at 2.45 GHz activates pinocytotic-like uptake of tracer by capillary endothelial cells of cerebral cortex.

Modulation of cell death in the rat thymus. Light and electron microscopic investigations.

Modulation of natural killer cell function after exposure to 60 Hz magnetic fields: confirmation of the effect in mature B6C3F1 mice.

Mortality in workers exposed to electromagnetic fields.

Mortality indices for hemoblastoses in Rivno Province before and after the accident at the Chernobyl Atomic Electric Power Station].

Nonlinear determinism in the immune system. In vivo influence of electromagnetic fields on different functions of murine lymphocyte subpopulations.

Nonlinear dynamical law governs magnetic field induced changes in lymphoid phenotype.

Occupation and malignant lymphoma: a population based case control study in Germany.

Odontologic survey of referred patients with symptoms allegedly caused by electricity or visual display units.

Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz.

Prenatal exposure to radiofrequencies: effects of WiFi signals on thymocyte development and peripheral T cell compartment in an animal model.

Probing lymphoma infiltration in spleen of AKR/J mice chronically exposed to electromagnetic fields for risk assessment--toward noninvasive modeling.

Radiofrequency exposure and mammalian cell toxicity, genotoxicity, and transformation.

Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems.

Residential electric consumption and childhood cancer in Canada (1971-1986)

Residential exposure to magnetic fields and risk of canine lymphoma.

Residential proximity to electricity transmission and distribution equipment and risk of childhood leukemia, childhood lymphoma, and childhood nervous system tumors: systematic review, evaluation, and meta-analysis.

Retinal damage experimentally induced by microwave radiation at 55 mW/cm2.

Reversible microwave effects on the blood-brain barrier.

Risk of major lymphoma subtypes and use of mobile phones].

Risk of neoplastic diseases in conditions of exposure to power magnetic fields--epidemiologic investigations].

Risk of neoplastic diseases in conditions of exposure to radio- and microwave fields--epidemiologic investigations].

Teratogenic effect of broad-band electromagnetic field on neonatal mice (Mus musculus).

The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats.

The effect of ultrahigh-frequency radiation on adaptation thresholds and the damages to blood system cells].

The effect on rat thymocytes of the simultaneous in vivo exposure to 50-Hz electric and magnetic field and to continuous light.

The effects of low-energy 60-Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase.

The efficiency and direction of thymus changes after whole-body exposure of mice to the weak electromagnetic field are determined by the initial status of the thymus].

The functional state of thymus cells following microwave exposure of endocrine glands.

The immunological and hormonal effects of combined exposure to a bitemporal ultrahigh-frequency electrical field and to decimeter waves at different sites].

The immunological mechanism of the modulation of IgE antibody formation during microwave irradiation of the thymus].

The role of fatty acids in anti-inflammatory effects of low-intensity extremely high-frequency electromagnetic radiation.

FACTOR 15

Theme - Circadian Rhythm

Key MeSH Headings - Melatonin, Circadian Rhythm, Pineal Gland

Titles

900-MHz microwave radiation promotes oxidation in rat brain.

A 0.5 G, 60 Hz magnetic field suppresses melatonin production in pinealocytes.

A 50-Hz electromagnetic field impairs sleep.

Acceleration of mammary tumorigenesis by exposure of 7,12-dimethylbenz[a]anthracene-treated female rats in a 50-Hz, 100-microT magnetic field: replication study.

Acute exposure to 50 Hz magnetic fields with harmonics and transient components: lack of effects on nighttime hormonal secretion in men.

Age-dependent association of exposure to television screen with children's urinary melatonin excretion?

Anatomical localization of human detection of weak electromagnetic radiation: experiments with dowsers.

Anxiogenic effect of chronic exposure to extremely low frequency magnetic field in adult rats.

Biological effects of continuous exposure of embryos and young chickens to electromagnetic fields emitted by video display units.

Biological effects of extremely low-frequency electromagnetic fields: in vivo studies.

Biological effects of non-ionizing electromagnetic radiation].

Biological effects produced by the influence of low frequency electromagnetic fields on hormone secretion].

Biological influences of electromagnetic fields].

Biologically based epidemiological studies of electric power and cancer.

Breast cancer and electric power.

Can disturbances in the atmospheric electric field created by powerline corona ions disrupt melatonin production in the pineal gland?

Cardiac autonomic control mechanisms in power-frequency magnetic fields: a multistudy analysis.

Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on nonchemical factors.

Chronic exposure to 2.9 mT, 40 Hz magnetic field reduces melatonin concentrations in humans.

Chronic exposure to ELF fields may induce depression.

Chronic exposure to ELF magnetic fields during night sleep with electric sheet: effects on diurnal melatonin rhythms in men.

Chronotoxicity of 1800 MHz microwave radiation on sex hormones and spermatogenesis in male mice].

Circadian locomotor activity of Musca flies: recording method and effects of 10 Hz square-wave electric fields.

Circadian rhythmicity of antioxidant markers in rats exposed to 1.8 GHz radiofrequency fields.

Designing EMF experiments: what is required to characterize "exposure"?

Direct suppressive effects of weak magnetic fields (50 Hz and 16 2/3 Hz) on melatonin synthesis in the pineal gland of Djungarian hamsters (Phodopus sungorus).

Do magnetic fields cause increased risk of childhood leukemia via melatonin disruption?

Does evening exposure to mobile phone radiation affect subsequent melatonin production?

Earthing: health implications of reconnecting the human body to the Earth's surface electrons.

Effect of occupational EMF exposure from radar at two different frequency bands on plasma melatonin and serotonin levels.

Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats.

Effects of 60-Hz magnetic field exposure on nocturnal 6-sulfatoxymelatonin, estrogens, luteinizing hormone, and follicle-stimulating hormone in healthy reproductive-age women: results of a crossover trial.

Effects of electric and magnetic fields from high-power lines on female urinary excretion of 6-sulfatoxymelatonin.

Effects of electric and magnetic fields on nocturnal melatonin concentrations in dairy cows.

Effects of electromagnetic fields exposure on plasma hormonal and inflammatory pathway biomarkers in male workers of a power plant.

Effects of electromagnetic fields on photophasic circulating melatonin levels in American kestrels.

Effects of electromagnetic radiation from 3G mobile phone on heart rate, blood pressure and ECG parameters in rats.

Effects of exposure to 16.7 Hz magnetic fields on urinary 6-hydroxymelatonin sulfate excretion of Swiss railway workers.

Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats.

Effects of mobile phone electromagnetic fields at nonthermal SAR values on melatonin and body weight of Djungarian hamsters (Phodopus sungorus).

Effects of mobile phone radiation on UV-induced skin tumourigenesis in ornithine decarboxylase transgenic and non-transgenic mice.

Effects of static electromagnetic fields on chick embryo pineal gland development.

Effects of weak alternating magnetic fields on nocturnal melatonin production and mammary carcinogenesis in rats.

Electric blanket or mattress cover use and breast cancer incidence in women 50-79 years of age.

Electric power, pineal function, and the risk of breast cancer.

Endocrine functions in young men exposed for one night to a 50-Hz magnetic field. A circadian study of pituitary, thyroid and adrenocortical hormones.

Evaluation in humans of the effects of radiocellular telephones on the circadian patterns of melatonin secretion, a chronobiological rhythm marker.

Evaluation of the nocturnal levels of urinary biogenic amines in men exposed overnight to 50-Hz magnetic field.

Evidence of oxidative stress in American kestrels exposed to electromagnetic fields.

Exacerbation of hypertension and disturbances of the geomagnetic field].

Examination of the melatonin hypothesis in women exposed at night to EMF or bright light.

Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons.

Exposure to electromagnetic fields and suicide among electric utility workers: a nested case-control study.

Extremely low frequency electromagnetic fields (EMF) and brain cancer in adults and children: review and comment.

Geomagnetic activity and human melatonin metabolite excretion.

Geomagnetic disturbances are associated with reduced nocturnal excretion of a melatonin metabolite in humans.

Human melatonin during continuous magnetic field exposure.

Immune markers and ornithine decarboxylase activity among electric utility workers.

Impact of microwave at X-band in the aetiology of male infertility.

Incidence of micronuclei in human peripheral blood lymphocytes exposed to modulated and unmodulated 2450 MHz radiofrequency fields.

Increases in geomagnetic activity are associated with increases in thyroxine levels in a single patient: implications for melatonin levels.

Influence of electromagnetic fields emitted by GSM-900 cellular telephones on the circadian patterns of gonadal, adrenal and pituitary hormones in men.

Influence of extremely-low-frequency magnetic field on antioxidative melatonin properties in AT478 murine squamous cell carcinoma culture.

Influence of light and electromagnetic radiation of Sun on circadian rhythms of the total antioxidant capacity of human saliva in the North].

Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure.

Interaction of static and extremely low frequency electric and magnetic fields with living systems: health effects and research needs.

Is melatonin the hormonal missing link between magnetic field effects and human diseases?

Is newborn melatonin production influenced by magnetic fields produced by incubators?

Is problematic mobile phone use explained by chronotype and personality?

Magnetic fields and pineal function in humans: evaluation of nocturnal acute exposure to extremely low frequency magnetic fields on serum melatonin and urinary 6-sulfatoxymelatonin circadian rhythms.

Magnetic storm effect on the circulation of rabbits.

Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells.

Melatonin and magnetic fields.

Melatonin attenuates radiofrequency radiation (900 MHz)-induced oxidative stress, DNA damage and cell cycle arrest in germ cells of male Swiss albino mice.

Melatonin metabolite levels in workers exposed to 60-Hz magnetic fields: work in substations and with 3-phase conductors.

Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain.

Melatonin protects rat cerebellar granule cells against electromagnetic field-induced increases in Na(+) currents through intracellular Ca(2+) release.

Melatonin protects rat thymus against oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes.

Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain.

Melatonin suppression by static and extremely low frequency electromagnetic fields: relationship to the reported increased incidence of cancer.

Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin.

Mobile phones and health: a literature overview.

Modifying effect of light and electromagnetic field on development of mammary tumors induced by N-nitrosomethyl urea in female rats].

Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin.

Morphometric and structural study of the pineal gland of the Wistar rat subjected to the pulse action of a 52 Gauss, (50 Hz) magnetic field. Evolutive analysis over 21 days.

Multi-night exposure to 60 Hz magnetic fields: effects on melatonin and its enzymatic metabolite.

Neuroprotective effects of melatonin and omega-3 on hippocampal cells prenatally exposed to 900 MHz electromagnetic fields.

Nighttime exposure to electromagnetic fields and childhood leukemia: an extended pooled analysis.

Nocturnal 6-hydroxymelatonin sulfate excretion in female workers exposed to magnetic fields.

Nocturnal excretion of a urinary melatonin metabolite among electric utility workers.

Nocturnal exposure to intermittent 60 Hz magnetic fields alters human cardiac rhythm.

Non-thermal biomarkers of exposure to radiofrequency/microwave radiation.

Nonionizing electromagnetic fields and cancer: a review.

Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin.

Pathophysiology of microwave radiation: effect on rat brain.

Prevention of mobile phone induced skin tissue changes by melatonin in rat: an experimental study.

Protective effect of melatonin and vitamin E against prooxidative action of iron ions and static magnetic field].

Rapid-onset/offset, variably scheduled 60 Hz electric and magnetic field exposure reduces nocturnal serum melatonin concentration in nonhuman primates.

Rate of occurrence of transient magnetic field events in U.S. residences.

Reduced excretion of a melatonin metabolite in workers exposed to 60 Hz magnetic fields.

Relationship between amyloid beta protein and melatonin metabolite in a study of electric utility workers.

Residential magnetic fields and the risk of breast cancer.

Risk factors, health risks, and risk management for aircraft personnel and frequent flyers.

Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca2+ signaling molecular pathways in breast cancer.

Serum-thyroxine levels in microwave-exposed rats.

Shift work, light at night, and breast cancer on Long Island, New York.

Temporal trends and misclassification in residential 60 Hz magnetic field measurements.

The effect of melatonin on body mass and behaviour of rats during an exposure to microwave radiation from mobile phone.

The effect of melatonin on the liver of rats exposed to microwave radiation.

The Effects of Electromagnetic Field on the Endocrine System in Children and Adolescents.

The effects of electromagnetic radiation (2450 MHz wireless devices) on the heart and blood tissue: role of melatonin.

The effects of extremely low-frequency magnetic fields on melatonin and cortisol, two marker rhythms of the circadian system.

The Effects of Melatonin on Oxidative Stress Parameters and DNA Fragmentation in Testicular Tissue of Rats Exposed to Microwave Radiation.

The excretion of 6-hydroxymelatonin sulfate in healthy young men exposed to electromagnetic fields emitted by cellular phone -- an experimental study.

The impact of electromagnetic field at a frequency of 50 Hz and a magnetic induction of 2.5 mT on viability of pineal cells in vitro.

The influence of long-term exposure of mice to randomly varied power frequency magnetic fields on their nocturnal melatonin secretion patterns.

The melatonin hypothesis: electric power and breast cancer.

The relationship between electromagnetic field and light exposures to melatonin and breast cancer risk: a review of the relevant literature.

The therapeutic effect of a pulsed electromagnetic field on the reproductive patterns of male Wistar rats exposed to a 2.45-GHz microwave field.

Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats.

Understanding the effects of electromagnetic field emissions from Marine Renewable Energy Devices (MREDs) on the commercially important edible crab, Cancer pagurus (L.).

Urinary 6-sulphatoxymelatonin excretion is increased in rats after 24 hours of exposure to vertical 50 Hz, 100 microT magnetic field.

Variations of melatonin and stress hormones under extended shifts and radiofrequency electromagnetic radiation.

FACTOR 16

Theme - Eye diseases

Key MeSH Headings - Eye Diseases, Cataract, Vision Disorders, Sensation Disorders, Neurotic Disorders, Lens, Crystalline, Corneal Diseases, Edema, Hematologic Diseases

Titles

A quantitative study on early changes in rabbit lens capsule epithelium induced by low power density microwave radiation].

Acute microwave irradiation and cataract formation in rabbits and monkeys.

Acute ocular lesions after exposure to electromagnetic radiation of ultrahigh frequency (an experimental study)].

Age-Related Modulations of AQP4 and Caveolin-1 in the Hippocampus Predispose the Toxic Effect of Phoneutria nigriventer Spider Venom.

Ascorbic acid changes in cultured rabbit lenses after microwave irradiation.

Biologic effects and hygienic regulation of electromagnetic fields caused by mobile communication devices].

Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields].

Cataracts induced by microwave and ionizing radiation.

Changes in gap junctional intercellular communication in rabbits lens epithelial cells induced by low power density microwave radiation.

Combined microwave energy and fixative agent for cataract induction in pig eyes.

Comments on Frey's "Data analysis reveals significant microwave-induced eye damage in humans".

Data analysis reveals significant microwave-induced eye damage in humans.

Dependence of anti-inflammatory effects of high peak-power pulsed electromagnetic radiation of extremely high frequency on exposure parameters].

DNA damage and repair induced by acute exposure of microwave from mobile phone on cultured human lens epithelial cells].

Dosimetric study of microwave cataractogenesis.

Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro].

Effect of high-power density microwave irradiation on the soluble proteins of the rabbit lens.

Effect of long-term power frequency electromagnetic field exposure on proliferation and apoptosis of SRA01/04 cells].

Effect of low-intensity microwave radiation on proliferation of cultured epithelial cells of rabbit lens].

Effect of superposed electromagnetic noise on DNA damage of lens epithelial cells induced by microwave radiation.

Effects of different dose microwave radiation on protein components of cultured rabbit lens].

Effects of exposure to microwaves: problems and perspectives.

Effects of Long-Term Exposure to 60 GHz Millimeter-Wavelength Radiation on the Genotoxicity and Heat Shock Protein (Hsp) Expression of Cells Derived from Human Eye.

Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats.

Effects of microwave radiation on the eye: the occupational health perspective.

Effects of microwave radiation on the lens epithelium in the rabbit eye.

Effects of mobile phones and radar radiofrequencies on the eye].

Effects of mobile phones on oxidant/antioxidant balance in cornea and lens of rats.

Effects of repeated microwave irradiations to the albino rabbit eye.

Electrical properties of lens material at microwave frequencies.

Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells.

Epidemiologic studies of the effect of microwaves (neurophysiologic, hematologic and ophthalmologic aspects)].

Epidemiological studies of human exposures to radiofrequency radiation. A critical review.

Evaluation of lens transparency in persons exposed to electromagnetic radiation of 27--30 MHz frequency].

Evaluation of possible microwave-induced lens changes in the United States Air Force.

Experimental studies on the influence of millimeter radiation on light transmission through the lens].

Features of anti-inflammatory effects of modulated extremely high-frequency electromagnetic radiation.

Glutathione concentration and peptidase activity in the lens after exposure to microwaves.

Hazards of radio frequency magnetic field and their prevention and control].

Health problems among workers of iron welding machines: an effect of electromagnetic fields.

In vitro studies of microwave-induced cataract. II. Comparison of damage observed for continuous wave and pulsed microwaves.

In vitro studies of microwave-induced cataract: reciprocity between exposure duration and dose rate for pulsed microwaves.

Increased occurrence of nuclear cataract in the calf after erection of a mobile phone base station].

Inducing cataract in postmortem pig eyes for cataract surgery training purposes.

Localized effects of microwave radiation on the intact eye lens in culture conditions.

Low power density microwave radiation induced early changes in rabbit lens epithelial cells.

Low power microwave radiation inhibits the proliferation of rabbit lens epithelial cells by upregulating P27Kip1 expression.

Low-intensity microwave blockes cell cycle and regulate cell cycle related gene expression in rabbit lens epithelial cells].

Microwave cataract and litigation: a case study.

Microwave cyclodestruction: evaluation on human eyes.

Microwave lens effects in humans. II. Results of five-year survey.

Microwave radiation-induced chromosomal aberrations in corneal epithelium of Chinese hamsters.

Microwaves and the visual analyzer].

Millimeter wave absorption in the nonhuman primate eye at 35 GHz and 94 GHz.

Mobile Phone Radiation: Physiological & Pathophysiologcal Considerations.

Neurotic disturbances, depression and anxiety disorders in the population living in the vicinity of overhead high-voltage transmission line 400 kV. Epidemiological pilot study].

Non-thermal cellular effects of lowpower microwave radiation on the lens and lens epithelial cells.

Observation of microwave-induced eye lens surface motion in vitro.

Ocular effects of radiofrequency energy.

Odontologic survey of referred patients with symptoms allegedly caused by electricity or visual display units.

On the microwave exposure.

Phantom vibration and phantom ringing among mobile phone users: A systematic review of literature.

Post-mortem histologic evaluation of microwave lesions after epicardial pulmonary vein isolation for atrial fibrillation.

Prevalence of nuclear cataract in Swiss veal calves and its possible association with mobile telephone antenna base stations.

Proteomic analysis of human lens epithelial cells exposed to microwaves.

Radiofrequency and microwave radiation in the microelectronics industry.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

Some ocular symptoms experienced by users of mobile phones.

State of peripheral blood of technical personnel exposed to constant magnetic fields].

The effect of extremely low frequency magnetic field on the conjunctiva and goblet cells.

The effects of cell phone use on peripheral vision.

The effects of ionizing radiation, microwaves, and ultrasound on the developing embryo: clinical interpretations and applications of the data.

The ocular effects of microwaves on hypothermic rabbits: a study of microwave cataractogenic mechanisms.

Thermal cataract formation in rabbits.

Thresholds for lenticular damage in the rabbit eye due to single exposure to CW microwave radiation: an analysis of the experimental information at a frequency of 2.45 GHz.

Ultrastructural change of rabbit lens epithelial cells induced by low power level microwave radiation].

Ultrastructural changes in the rabbit lens induced by microwave radiation.

Video display terminals: risk of electromagnetic radiation.

FACTOR 17

Theme - Electromagnetic interference in implanted electronic devices

Key MeSH Headings - Tachycardia, Ventricular, Ventricular Fibrillation, Death, Sudden, Cardiac, Arrhythmias, Cardiac

Titles

AANA Journal Course: update for nurse anesthetists. Arrhythmia management devices and electromagnetic interference.

Accidental deaths caused by electricity in Sweden, 1975-2000.

Are patients with cardiac implants protected against electromagnetic interference in daily life and occupational environment?

Avoidance behaviors in patients with implantable cardioverter defibrillators.

Cardiac autonomic control mechanisms in power-frequency magnetic fields: a multistudy analysis.

Deaths associated with implantable cardioverter defibrillator failure and deactivation reported in the United States Food and Drug Administration Manufacturer and User Facility Device Experience Database.

Detection of refrigerator-associated 60 Hz alternating current as ventricular fibrillation by an implantable defibrillator.

Disturbances in the function of cardiac pacemaker caused by short wave and microwave diathermies and pulsed high frequency current.

Do airport metal detectors interfere with implantable pacemakers or cardioverter-defibrillators?

Do media players cause interference with pacemakers?

Do mobile telephones have adverse effects on the functions of implantable cardioverter defibrillators?].

ECG changes caused by the effect of static magnetic fields of nuclear magnetic resonance tomography using magnets with a field power of 0.5 to 4.0 Telsa].

Effects of 900 MHz electromagnetic field emitted by cellular phones on electrocardiograms of guinea pigs.

Electromagnetic fields and health effects--epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease.

Electromagnetic Interference (EMI) and arrhythmic events in ICD patients undergoing gastrointestinal procedures.

Electromagnetic interference in cardiac rhythm management devices.

Electromagnetic interference in implantable cardioverter defibrillators: present but rare.

Electromagnetic interference of cardiac rhythmic monitoring devices to radio frequency identification: analytical analysis and mitigation methodology.

Electromagnetic interference with cardiac pacemakers and implantable cardioverter-defibrillators from low-frequency electromagnetic fields in vivo.

Electromagnetic interference with implantable cardioverter-defibrillators at power frequency: an in vivo study.

Fine structural alterations in radiofrequency energy-induced lesions in dog hearts: possible basis for reduced arrhythmic complications.

Implantable cardioverter defibrillators and cellular telephones: is there any interference?

Implanted devices and electromagnetic interference: case presentations and review.

Induction ovens and electromagnetic interference: what is the risk for patients with implantable cardioverter defibrillators?

Induction ovens and electromagnetic interference: what is the risk for patients with implanted pacemakers?

Influence of 50 Hz electric and magnetic fields on the human heart.

Influence of digital and analogue cellular telephones on implanted pacemakers.

Interference of electrical dental equipment with implantable cardioverter-defibrillators.

Interference with cardiac pacemakers by cellular telephones.

Interference with cardiac pacing.

Is there any risk interaction between electromagnetic field generated by mobile phones and artificial pacemakers].

Magnetic field exposure and arrythmic risk: evaluation in railway drivers.

Magnetism and cardiac arrhythmias.

Microwave effects on isolated chick embryo hearts.

Modifications in ventricular fibrillation and capture capacity induced by a linear radiofrequency lesion.

Risk of severe cardiac arrhythmia in male utility workers: a nationwide danish cohort study.

Selective interference with pacemaker activity by electrical dental devices.

Studies on microwaves in medicine and biology: from snails to humans.

The effect of power frequency high intensity electric fields on implanted cardiac pacemakers.

Ventricular fibrillation induced by radiofrequency energy delivery for premature ventricular contractions arising from the right ventricular outflow tract: is implantablecardioverterdefibrillator indicated?

FACTOR 18

Theme – Liver Neoplasms

Key MeSH Headings - Liver Neoplasms, Carcinoma, Hepatocellular, Neoplasm Recurrence, Local, Lymphatic Metastasis

Titles

40 GHz RF biosensor based on microwave coplanar waveguide transmission line for cancer cells (HepG2) dielectric characterization.

A case of hepatocellular carcinoma rupturing after angiography.

A case of recurring hepatocellular carcinoma with a solitary Virchow's lymph node metastasis.

A case report of primary hepatic carcinoid with lymph node metastasis--treatment of hepatic arterial infusion to post-reoperative liver and radiation to metastasis of para-aortic lymph nodes].

Construction and clinical significance of a predictive system for prognosis of hepatocellular carcinoma.

Effects of extremely low-frequency electromagnetic fields (ELF-EMF) exposure on B6C3F1 mice.

Geomagnetic field variation in early ontogenesis as a risk factor for oncopathology].

Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure.

Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice.

Mobile phone radiation alters proliferation of hepatocarcinoma cells.

MoS2 nanosheets encapsulated in sodium alginate microcapsules as microwave embolization agents for large orthotopic transplantation tumor therapy.

Multimodal treatment of hepatocellular carcinoma.

Non-resection approaches for colorectal liver metastases.

Rat liver foci study on coexposure with 50 Hz magnetic fields and known carcinogens.

FACTOR 19

Theme – Symptoms of discomfort

Key MeSH Headings - Headache, Dizziness, Fatigue, Depression, Anxiety, Tremor, Sleep Wake Disorders, Neurotic Disorders, Stress, Psychological, Anxiety Disorders, Nervous System Diseases

Titles

A 50-Hz electromagnetic field impairs sleep.

A literature review of medical side effects from radio-frequency energy in the human environment: involving cancer, tumors, and problems of the central nervous system.

A study on the biological effects of exposure mobile-phone frequency EMF].

A survey study on some neurological symptoms and sensations experienced by long term users of mobile phones.

Altered cortical excitability in subjectively electrosensitive patients: results of a pilot study.

An analysis of the impact of cell phone use on depressive symptoms among Japanese elders.

Anxiety-like behavioural effects of extremely low-frequency electromagnetic field in rats.

Anxiogenic effect of chronic exposure to extremely low frequency magnetic field in adult rats.

Are media reports able to cause somatic symptoms attributed to WiFi radiation? An experimental test of the negative expectation hypothesis.

Association between Excessive Use of Mobile Phone and Insomnia and Depression among Japanese Adolescents.

Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study.

Association between mobile phone use and depressed mood in Japanese adolescents: a cross-sectional study.

Association between overuse of mobile phones on quality of sleep and general health among occupational health and safety students.

Association between problematic cellular phone use and suicide: the moderating effect of family function and depression.

Association of low job control with a decrease in memory (CD4+ CD45RO+) T lymphocytes in Japanese middle-aged male workers in an electric power plant.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Association of tinnitus and electromagnetic hypersensitivity: hints for a shared pathophysiology?

Associations between problematic mobile phone use and psychological parameters in young adults.

Avoidance behaviors in patients with implantable cardioverter defibrillators.

Bedtime mobile phone use and sleep in adults.

Behavior and memory evaluation of Wistar rats exposed to 1.8 GHz radiofrequency electromagnetic radiation.

Can exposure to a terrestrial trunked radio (TETRA)-like signal cause symptoms? A randomised double-blind provocation study.

Cancer incidence and magnetic field exposure in industries using resistance welding in Sweden.

Cell phones: modern man's nemesis?

Cellular phones for reducing battlefield stress: rationale and a preliminary research.

Cerebral radiofrequency exposures during adolescence: Impact on astrocytes and brain functions in healthy and pathologic rat models.

Chronic exposure to an extremely low-frequency magnetic field induces depression-like behavior and corticosterone secretion without enhancement of the hypothalamic-pituitary-adrenal axis in mice.

Chronic exposure to ELF fields may induce depression.

Clinical features of headache associated with mobile phone use: a cross-sectional study in university students.

Cohort study on the effects of everyday life radio frequency electromagnetic field exposure on non-specific symptoms and tinnitus.

Contribution of physical factors to the complex anthropogenic load in an industrial town].

Coping and self-image in patients with visual display terminal-related skin symptoms and perceived hypersensitivity to electricity.

Correction of microcirculatory disturbances with terahertz electromagnetic radiation at nitric oxide frequencies in albino rats under conditions of acute stress.

Delayed biological effect of electromagnetic fields action].

Depression in high voltage power line workers.

Determinants and stability over time of perception of health risks related to mobile phone base stations.

Development of a problematic mobile phone use scale for Turkish adolescents.

Do mobile phone base stations affect sleep of residents? Results from an experimental double-blind sham-controlled field study.

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FACTOR 20

Theme - Neoplasms

Key MeSH Headings - Lung Neoplasms, Ovarian Neoplasms, Pituitary Neoplasms, Lymphoma, Prostatic Neoplasms, Colonic Neoplasms, Carcinoma, Breast Neoplasms, Hematologic Neoplasms, Neoplasms, Liver Neoplasms, Cell Transformation, Neoplastic, Nervous System Neoplasms

Titles

2-GHz band CW and W-CDMA modulated radiofrequency fields have no significant effect on cell proliferation and gene expression profile in human cells.

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Risks of carcinogenesis from electromagnetic radiation of mobile telephony devices.

Rodent cell transformation and immediate early gene expression following 60-Hz magnetic field exposure.

Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca2+ signaling molecular pathways in breast cancer.

Role of radical pairs and feedback in weak radio frequency field effects on biological systems.

Scaling Relationship of In Vivo Muscle Contraction Strength of Rabbits Exposed to High-Frequency Nanosecond Pulse Bursts.

Searching for the perfect wave: the effect of radiofrequency electromagnetic fields on cells.

Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells.

Shift work, light at night, and breast cancer on Long Island, New York.

Socioeconomic status, social mobility and cancer occurrence during working life: a case-control study among French electricity and gas workers.

Some characteristics of the glutathione cycle revealed by ionising and non-ionising electromagnetic radiation.

Speculations on the influence of electromagnetism on genomic and associated structures.

Studies on microwaves in medicine and biology: from snails to humans.

Studying the protein expression in human B lymphoblastoid cells exposed to 1.8-GHz (GSM) radiofrequency radiation (RFR) with protein microarray.

Teratogenic effect of broad-band electromagnetic field on neonatal mice (Mus musculus).

Testing electromagnetic fields for potential carcinogenic activity: a critical review of animal models.

The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumors and other neoplasms in rats.

The effects of ionizing radiation, microwaves, and ultrasound on the developing embryo: clinical interpretations and applications of the data.

The effects of low-energy 60-Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase.

The effects of recall errors and of selection bias in epidemiologic studies of mobile phone use and cancer risk.

The electromagnetic spectrum: current and future applications in oncology.

The enhanced lethality of cells in suspension during simultaneous exposure to pulsed electrical and shock-wave acoustic fields].

The epidemiology of exposure to electromagnetic fields: an overview of the recent literature.

The INTERPHONE study: design, epidemiological methods, and description of the study population.

The measured electrical properties of normal and malignant human tissues from 50 to 900 MHz.

The melatonin hypothesis: electric power and breast cancer.

The relationship between electromagnetic field and light exposures to melatonin and breast cancer risk: a review of the relevant literature.

The relative merits of contemporary measurements and historical calculated fields in the Swedish childhood cancer study.

The residential case-specular method to study wire codes, magnetic fields, and disease.

The role of chemical and physical factors in cancer development].

The role of household electromagnetic fields in the development of mammary tumors in women: clinical case-record observations.

The use of cell phone and insight into its potential human health impacts.

Towards 5G communication systems: Are there health implications?

Transmission electron microscopy study of the effects produced by wide-band low-power millimeter waves on MCF-7 human breast cancer cells in culture.

Use of cellular telephones and risk of cancer. A Danish cohort study].

Use of electric bedding devices and risk of breast cancer in African-American women.

Use of electric blankets and risk of postmenopausal breast cancer.

Validation of self-reported cellular phone use.

Variable E-cadherin expression in a MNU-induced colon tumor model in rats which exposed with 50 Hz frequency sinusoidal magnetic field.

Appendix 4 – Hierarchical Text Clustering Taxonomy of Adverse EMF Effects Database

A4-A. Cluster Themes

A query to retrieve Medline records showing adverse health effects of wireless radiation was generated. The query was entered into the Medline search engine, and ~15,000 records were retrieved. Filtering was applied to the retrieval to remove records not associated with adverse health effects of wireless radiation, and 5311 records remained. Further filtering was not done, and more records showing no adverse effects, examining ELF frequencies, and exceeding the FCC exposure limits, were included compared to the filtered database in Appendix 2. The partially filtered records were imported into the CLUTO software, and a 48-cluster hierarchical text clustering of titles/abstracts was performed.

The following tables (A4-1, A4-2) show the categories in the taxonomy. The first table shows hierarchical Levels 2 and 4, and the second table shows Level 4 and its associated leaf (lowest level) clusters. For each cluster in both tables, the number of associated records is shown in parentheses, followed by the cluster theme. Following the tables, each leaf cluster is shown, including numbers of records, theme, and associated record titles. The Level 4 clusters in the second table are hyperlinked to their positions in the list of titles. Because of the filtering process limitations, most, but not all, records are associated with adverse effects of wireless radiation. To access the full record, insert the titles of interest into Pubmed or other Medline search engine.

Main adverse effects identified at the cluster theme level include cancer, brain tumors, mammary cancer, childhood cancer, childhood leukemia, breast cancer, acoustic neuromas, neurodegenerative diseases, cognitive function, neural function, oxidative stress, genotoxic, DNA damage, chromosome damage, gene expression alterations, implanted electronic device malfunction, sleep, melatonin secretion, embryos, cataracts, hearing, electrohypersensitivity.

Table A4-1 - CLUTO-Based Text Clustering Taxonomy – Top Levels

SECOND LEVEL	FOURTH LEVEL
Cluster 92 (2561) – Adverse effects of wireless radiation at cellular level, including radiation absorption at different frequencies	Cluster 78 (912) – Adverse impacts of wireless radiation, especially on cataracts, cells, and cognitive functions
	Cluster 79 (428) – Microwave radiation absorption at different frequencies
	Cluster 82 (529) – Adverse effects of mobile phone radiation, especially oxidative stress
	Cluster 84 (692) – Genotoxic effects of radiofrequency radiation
Cluster 93 (2750) – Adverse health effects of EMF on humans, especially cancer and neurodegenerative diseases, and on implanted electronic devices	Cluster 81 (673) – Adverse impacts of power-line EMF
	Cluster 85 (540) – Adverse impacts of low-frequency EMF, emphasizing cancer and neurodegenerative diseases
	Cluster 83 (668) – Adverse effects of mobile phone use, especially brain tumors, and brain and neural function
	Cluster 89 (869) – Human health risks from electromagnetic radiation, including adverse effects on implanted electronic devices, and possible protections

Table A4-2. CLUTO-Based Text Clustering Taxonomy - Bottom Levels

FOURTH LEVEL	LEAF (LOWEST) LEVEL
Cluster 78 (912) – Adverse impacts of wireless radiation, especially on cataracts, cells, and cognitive functions	Cluster 46 (331) – Adverse effects of microwave radiation, mainly on rats
	Cluster 3 (39) – Adverse impact of wireless radiation on eye lens
	Cluster 35 (107) – Adverse impacts of microwave radiation on cells and cognitive functions
	Cluster 39 (211) – Adverse effects from microwave radiation
	Cluster 29 (94) – Adverse effects of microwave radiation, especially pulsed microwave
	Cluster 31 (130) – Adverse effects of microwave exposures on rats, especially at WiFi frequencies
Cluster 79 (428) – Microwave radiation absorption at different frequencies	Cluster 10 (75) – Dielectric properties of tissue at different microwave frequencies
	Cluster 23 (88) – Specific absorption rate in human body models
	Cluster 21 (63) – Adverse effects of millimeter-wave exposures on biological systems
	Cluster 44 (95) – Adverse effects of microwave resonances in biological systems
	Cluster 47 (107) – Adverse biological effects of decimeter waves
Cluster 82 (529) – Adverse effects of mobile phone radiation, especially oxidative stress	Cluster 22 (127) – Effects of radiofrequency radiation, especially from mobile phones, on rats
	Cluster 26 (129) - Oxidative stress effects from mobile phone radiofrequency radiation
	Cluster 37 (140) – Effect of radiofrequency exposure, especially prenatal exposure, on rats
	Cluster 38 (133) – Effect of radiofrequency radiation on rat brain
Cluster 84 (692) – Genotoxic effects of radiofrequency radiation	Cluster 20 (126) – DNA damage after microwave radiation
	Cluster 28 (100) – Chromosome damage in lymphocytes exposed to radiofrequency radiation
	Cluster 45 (179) – Adverse effects of low-frequency EMF on cells
	Cluster 24 (111) – Gene expression alterations following radiofrequency exposure
	Cluster 11 (51) – Adverse impacts of radiofrequency fields on sleep
	Cluster 41 (125) – Adverse effects of radiofrequency fields on cells
Cluster 81 (673) – Adverse impacts of power-line EMF	Cluster 9 (43) – Adverse effects of ELF magnetic field exposures
	Cluster 17 (55) – Adverse impacts of EMF on mammary cancer development
	Cluster 6 (67) – Adverse health effects of magnetic fields associated with magnetic resonance imaging
	Cluster 32 (139) – Health risks of power-line electromagnetic fields on humans
	Cluster 34 (188) – Adverse effects of low-frequency electromagnetic fields on humans

	Cluster 40 (116) – Adverse effects of low-frequency magnetic fields on rodents
	Cluster 2 (27) – Effects of electromagnetic fields on chicken embryos
	Cluster 12 (38) – Impact of static and low-frequency magnetic fields on melatonin secretion
Cluster 85 (540) – Adverse impacts of low-frequency EMF, emphasizing cancer and neurodegenerative diseases	Cluster 4 (97) – Exposure to power lines and risk of childhood cancer
	Cluster 15 (131) – Residential magnetic fields and childhood leukemia
	Cluster 13 (113) – Electromagnetic fields and cancer, especially breast cancer
	Cluster 18 (62) – Mortality studies of electrical utility workers, focusing on electromagnetic field exposures
	Cluster 27 (137) – Occupational exposure to electromagnetic fields, emphasizing neurodegenerative disease and cancer
Cluster 83 (668) – Adverse effects of mobile phone use, especially brain tumors, and brain and neural function	Cluster 30 (321) – Adverse health symptoms from mobile phone use
	Cluster 1 (36) – Effects of mobile phones on brain and neural function
	Cluster 25 (68) – Effects of cell phone radiation on cognitive function and hearing
	Cluster 14 (93) – Myriad adverse health effects from cellphones
	Cluster 7 (44) – Risks from cell phone use, especially brain tumors
	Cluster 8 (106) – Risk of brain tumors/acoustic neuromas from mobile phone use
Cluster 89 (869) – Human health risks from electromagnetic radiation, including adverse effects on implanted electronic devices, and possible protections	Cluster 0 (63) – Electromagnetic interference with cardiac pacemakers
	Cluster 16 (103) – Electromagnetic interference on implanted cardiac devices
	Cluster 5 (120) – Health risks from mobile phone base stations
	Cluster 19 (84) – Electromagnetic hypersensitivity
	Cluster 43 (202) – Health risks from low-frequency electromagnetic fields
	Cluster 33 (91) – Health risks to workers in different occupations
	Cluster 36 (84) – Precautionary measures to reduce potential EMF health risks
	Cluster 42 (122) - Regulatory protections against electromagnetic fields

A4-B. Cluster Record Titles

Fourth Level Cluster 78 (912)
Theme - Adverse impacts of wireless radiation, especially on cataracts, cells, and cognitive functions

--Leaf Cluster 46 (331)

Theme - Adverse effects of microwave radiation, mainly on rats

Titles

Recent advances in the effects of microwave radiation on brains.

Microwave radiation absorption: behavioral effects.

Behavioral thermoregulation with microwave radiation of albino rats.

[Effect of microwave irradiation on biological systems].

[Microwave radiation sources requiring periodic or sporadic hygienic control].

Microwave radiation (2.45 GHz)-induced oxidative stress: Whole-body exposure effect on histopathology of Wistar rats.

Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain.

Mechanism of low-level microwave radiation effect on nervous system.

Apoptosis of Lewis Lung Carcinoma Cells Induced by Microwave via p53 and Proapoptotic Proteins In vivo.

Studies on the interaction of microwave radiation with cholinesterase.

A system for studying effects of microwaves on cells in culture.

Enzymatic alterations in developing rat brain cells exposed to a low-intensity 16.5 GHz microwave radiation.

Bioeffects of microwave--a brief review.

Behavioral effects of chlorpromazine and diazepam combined with low-level microwaves.

Microwave radiation induced oxidative stress, cognitive impairment and inflammation in brain of Fischer rats.

Interaction of microwave radiation with turkey sperm.

Effect of Low Level Subchronic Microwave Radiation on Rat Brain.

Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation.

Effect of 2.45 GHz microwave radiation on the fertility pattern in male mice.

Alterations in activity at auditory nuclei of the rat induced by exposure to microwave radiation: autoradiographic evidence using [14C]2-deoxy-D-glucose.

[Effect of microwave radiation on the rat hematopoietic system].

The effect of exposure of acetylcholinesterase to 2,450-MHz microwave radiation.

[The impact of electromagnetic radiation at microwave frequency (9.8 HhZ)on the embryonic and postembryonic development of the tick Hyalomma asiaticum (Acarina, Ixodidae)].

[Structural and metabolic analysis of the reaction of the central nervous system to the combined action of microwave and ionizing radiations].

[Mechanism of the effect of nonionizing radiation on animals at the level of sensory systems].

Effects of microwaves on membranes of hematopoietic cells in their structural and functional organization.

Microwave radiation and chlordiazepoxide: synergistic effects on fixed-interval behavior.

Behavioral effects of microwaves.

[Long-term exposure to low intensity microwave radiation affects male reproductivity].

[Effect of microwave radiation on cellular immunity indices in conditions of chronic exposure].

[Long-term microwave radiation affects male reproduction in rats].

Results of our 15-year study into the biological effects of microwave exposure.

Effect of whole-body 1800MHz GSM-like microwave exposure on testicular steroidogenesis and histology in mice.

Genotoxic Effects in Human Fibroblasts Exposed to Microwave Radiation.

[The effect of microwave radiation on the levels of MDA and the activity of SOD of nasopharyngeal carcinoma cells].

The effects of low-level radiofrequency and microwave radiation on brain tissue and animal behaviour.

Physiological changes in rats after exposure to low levels of microwaves.

The influence of prenatal 10 GHz microwave radiation exposure on a developing mice brain.

[The phenomenon of adaptive immunity in exposure to nonionizing microwave radiation].

Non-thermal effects of 500MHz - 900MHz microwave radiation on enzyme kinetics.

Activation of TLR signalling regulates microwave radiation-mediated impairment of spermatogenesis in rat testis.

Effect of 2.45 GHz microwave radiation on permeability of unilamellar liposomes to 5(6)-carboxyfluorescein. Evidence of non-thermal leakage.

Spatial memory and learning performance and its relationship to protein synthesis of Swiss albino mice exposed to 10 GHz microwaves.

[Effects of the microwave radiation from the cellular phones on humans and animals].

Cognitive impairment and neurogenotoxic effects in rats exposed to low-intensity microwave radiation.

Microwave hearing: evidence for thermoacoustic auditory stimulation by pulsed microwaves.

Effects of 2.45 GHz microwave radiation and heat on mouse spermatogenic epithelium.

Studies of the induction of dominant lethals and translocations in male mice after chronic exposure to microwave radiation.

Effect of Low-Intensity Microwave Radiation on Monoamine Neurotransmitters and Their Key Regulating Enzymes in Rat Brain.

Fluorescence depolarization studies of red cell membrane fluidity. The effect of exposure to 1.0-GHz microwave radiation.

[Effects of microwave radiation on the content of five elements in mice bone tissue].

Research on the neurological effects of nonionizing radiation at the University of Washington.

Cellular neoplastic transformation induced by 916 MHz microwave radiation.

Radiofrequency and microwave radiation in the microelectronics industry.

Reduced exposure to microwave radiation by rats: frequency specific effects.

[Reaction of the brain receptor system to the effect of low intensity microwaves].

Ten gigahertz microwave radiation impairs spatial memory, enzymes activity, and histopathology of developing mice brain.

Cytogenetic effects of 18.0 and 16.5 GHz microwave radiation on human lymphocytes in vitro.

Influence of microwave exposure on fertility of male rats.

Health aspects of radio and microwave radiation.

Japanese encephalitis virus (JEV): potentiation of lethality in mice by microwave radiation.

Microwave exposure induces Hsp70 and confers protection against hypoxia in chick embryos.

Potentially hazardous microwave radiation source--a review.

Behavioral effects of microwave reinforcement schedules and variations in microwave intensity on albino rats.

[Metabolic changes in cells under electromagnetic radiation of mobile communication systems].

Effect of microwave radiation on inactivation of Clostridium sporogenes (PA 3679) spores.

A circular dichroism study of human erythrocyte ghost proteins during exposure to 2450 MHz microwave radiation.

[Evaluation of bone density in rats after hydrocortisone and microwave radiation].

[Chronotoxicity of 1800 MHz microwave radiation on sex hormones and spermatogenesis in male mice].

Radiation hazard assessment of pulsed microwave radars.

Results of a United States and Soviet Union joint project on nervous system effects of microwave radiation.

Non-thermal microwave effects on protein dynamics? An X-ray diffraction study on tetragonal lysozyme crystals.

Effect of microwave radiation on permeability of liposomes. Evidence against non-thermal leakage.

Assessment of cytogenetic damage and oxidative stress in personnel occupationally exposed to the pulsed microwave radiation of marine radar equipment.

[Modification of the effects of microwave irradiation on biochemical processes by using foreign protein].

Some behavioral effects of short-term exposure of rats to 2.45 GHz microwave radiation.

[5-HT contents change in peripheral blood of workers exposed to microwave and high frequency radiation].

Microwave radiation effects on the thermally driven oxidase of erythrocytes.

Effects of microwave radiation (340 and 900 MHz) on different structural levels of erythrocyte membranes.

Environmental radiation hazards.

[Radiation protection and possible mechanisms for low intensity microwave].

Effects of low level microwave radiation on carcinogenesis in Swiss Albino mice.

Effects of pulsed 2.856 GHz microwave exposure on BM-MSCs isolated from C57BL/6 mice.

[Effect of pulse electromagnetic radiation on erythrocyte ghosts].

[Experimental modeling of autoimmune reactions as affected by nonionizing microwave radiation].

Biologic effects of microwave exposure. II. Studies on the mechanisms controlling susceptibility to microwave-induced increases in complement receptor-positive spleen cells.

Hearing of microwave pulses by humans and animals: effects, mechanism, and thresholds.

Individual responsiveness to induction of micronuclei in human lymphocytes after exposure in vitro to 1800-MHz microwave radiation.

Immunologic and hematopoietic alterations by 2,450-MHz electromagnetic radiation.

Exposure of cultured astroglial and microglial brain cells to 900 MHz microwave radiation.

Influence of microwave exposure on chlordiazepoxide effects in the mouse staircase test.

Activation of endoplasmic reticulum stress in rat brain following low-intensity microwave exposure.

Non-thermal effects of microwaves on proteins: thermophilic enzymes as model system.

[Morinda officialis how extract improves microwave-induced reproductive impairment in male rats].

Reception of microwaves by the brain.

Interaction of radiofrequency and microwave radiation with living systems. A review of mechanisms.

Effect on the immune system of mice exposed chronically to 50 Hz amplitude-modulated 2.45 GHz microwaves.

Selective changes in locomotor activity in mice due to low-intensity microwaves amplitude modulated in the EEG spectral domain.

Physical basis of adverse and therapeutic effects of low intensity microwave radiation.

Resonance effect of microwaves on the genome conformational state of E. coli cells.

Induction of micronuclei in human lymphocytes exposed in vitro to microwave radiation.

Biological effects of electromagnetic fields--mechanisms for the effects of pulsed microwave radiation on protein conformation.

The influence of microwave radiation on transdermal delivery systems.

Parametric mechanism of excitation of the electroencephalographic rhythms by modulated microwave radiation.

[Cytogenetic changes induced by low-intensity microwaves in the species Triticum aestivum].

High-frequency electromagnetic radiation injury to the upper extremity: local and systemic effects.

Effect of chronic microwave radiation on T cell-mediated immunity in the rabbit.

Prenatal microwave exposure and behavior.

[The state of receptor-dependent signal pathways in the agranulocytes from the peripheral blood of the reconvalescent patients following community-acquired pneumonia under the influence of microwave radiation].

Effects of 2.45 GHz microwaves on meiotic chromosomes of male CBA/CAY mice.

Inhibitory Effects of Microwave Radiation on LPS-Induced NFkappaB Expression in THP-1 Monocytes.

The relation of dose rate of microwave radiation to the time of death and total absorbed dose in the mouse.

Differential damage in bacterial cells by microwave radiation on the basis of cell wall structure.

Effect of 7, 14 and 21 Hz modulated 450 MHz microwave radiation on human electroencephalographic rhythms.

Immunotropic influence of 900 MHz microwave GSM signal on human blood immune cells activated in vitro.

[Are microwaves a co-teratogen? Experimental model concept and its verification].

Studies on microwaves in medicine and biology: from snails to humans.

Changes in human EEG caused by low level modulated microwave stimulation.

Electromagnetic radiations and cancer. Cause and prevention.

Microwave elution of red cell antibodies.

Influence of low intensity 2,450 MHz microwave radiation upon the growth of various micro-organisms and their sensitivity towards chemical inactivation.

Low power microwave interaction with phospholipase C and D signal transduction pathways in myogenic cells.

[Antagonistic effect of microwave on hematopoietic damage of mice induced by gamma-ray irradiation].

Acid resistance and verocytotoxin productivity of enterohemorrhagic Escherichia coli O157:H7 exposed to microwave.

The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation.

Effects of differently polarized microwave radiation on the microscopic structure of the nuclei in human fibroblasts.

The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro.

A search for nonthermal effects of 434 MHz microwave radiation on whole human blood.

Review of the specific effects of microwave radiation on bacterial cells.

Microwave dissociation of antigen-antibody complexes: a new elution technique to permit phenotyping of antibody-coated red cells.

[Nature of the changes in the morphofunctional and cytochemical indices of blood leukocytes as affected by low-intensity microwaves].

Effects of 2.45-GHz microwave radiation and phorbol ester 12-O-tetradecanoylphorbol-13-acetate on dimethylhydrazine-induced colon cancer in mice.

Effects of X-band microwave exposure on rabbit erythrocytes.

Transgenic nematodes as biomonitors of microwave-induced stress.

The effects of microwave radiation on avian dominance behavior.

Dominant lethal studies in male mice after exposure to 2.45 GHz microwave radiation.

[Action of UHF microwaves on the germ and somatic cells of mammals].

Sperm count and sperm abnormality in male mice after exposure to 2.45 GHz microwave radiation.

Effect of microwaves (2450-MHz) on the immune system in mice: studies of nucleic acid and protein synthesis.

The influence of differently polarised microwave radiation on chromatin in human cells.

Effects of 10-GHz microwaves on hematological parameters in Swiss albino mice and their modulation by Prunus avium.

Effects of fetal microwave radiation exposure on offspring behavior in mice.

Ibuprofen effects on behavioral thermoregulation with microwave radiation in albino rats.

Microwave and man: the direct and indirect hazards, and the precautions.

Biomarkers in volunteers exposed to mobile phone radiation.

Effects of 900-MHz microwave radiation on gamma-ray-induced damage to mouse hematopoietic system.

Detection of probable effects of microwave exposure of blood parameters of RBC, PCV and Hb in rat.

Effects of microwaves on the colony-forming capacity of haemopoietic stem cells in mice.

2.45-GHz microwave irradiation adversely affects reproductive function in male mouse, Mus musculus by inducing oxidative and nitrosative stress.

Adaptation of human brain bioelectrical activity to low-level microwave.

Biochemical changes in rat brain exposed to low intensity 9.9 GHz microwave radiation.

Effect of microwave radiation on the permeability of carbonic anhydrase loaded unilamellar liposomes.

Effects on the nervous system by exposure to electromagnetic fields: experimental and clinical studies.

Microwave effect on diffusion: a possible mechanism for non-thermal effect.

Teratology, survival, and reversal learning after fetal irradiation of mice by 2450-MHz microwave energy.

[Changes in drug pharmacokinetics and pharmacodynamics under the influence of microwaves of different ranges].

Increase in the frequency of Fc receptor (FcR) bearing cells in the mouse spleen following a single exposure of mice to 2450 MHz microwaves.

Modification of membrane fluidity in melanin-containing cells by low-level microwave radiation.

Effects of low level microwave radiation on the digestive transit of the rat.

Microwave-stimulated drug release from liposomes.

[Changes in immunobiological reactivity under the combined action of microwave, infrasonic and gamma irradiation].

Behavioral and cognitive effects of microwave exposure.

The effect of microwave radiation on the cell genome.

Neurological effects of microwave exposure related to mobile communication.

Microwave effects on plasmid DNA.

Differential response of the permeability of the rat liver canalicular membrane to sucrose and mannitol following in vivo acute single and multiple exposures to microwave radiation (2.45 GHz) and radiant-energy thermal stress.

Effects of microwave radiation and strychnine on cerebral biopotentials in narcotized rats.

[Some biochemical indexes in white rabbit's blood affected by acute high intensity microwave].

Long-term exposure to microwave radiation provokes cancer growth: evidences from radars and mobile communication systems.

Effect of low power microwave on the mouse genome: a direct DNA analysis.

Fluorescence depolarization studies of the phase transition in multilamellar phospholipid vesicles exposed to 1.0-GHz microwave radiation.

Effect of low frequency modulated microwave exposure on human EEG: individual sensitivity.

A negative test for mutagenic action of microwave radiation in Drosophila melanogaster.

[Non-thermal microwave effect on nerve fiber function].

Influence of microwaves on different types of receptors and the role of peroxidation of lipids on receptor-protein shedding.

[Effect of nonionizing microwave radiation on autoimmune reactions and antigenic structure of serum proteins].

Effects of 9.4 GHz microwave exposure on meiosis in mice.

[Two-step exposure of biological objects to infrared laser and microwave radiation].

Influence of in vitro microwave radiation on the fertilizing capacity of turkey sperm.

Measure of enzymatic activity coincident with 2450 MHz microwave exposure.

Effect of microwave radiation on redissolving precipitated matter in fluorouracil injection.

[Germ reduction by microwaves--microwave specific effects].

Effects of microwave (2.45 GHz) irradiation on some biological characters of Salmonella typhimurium.

The relation of sex, age, and weight of mice to microwave radiation sensitivity.

The origins of U.S. safety standards for microwave radiation.

Pathophysiology of microwave radiation: effect on rat brain.

Influence of CW microwave radiation on in vitro release of enzymes from retinol- treated hepatic lysosomes.

Cytological effects of microwave radiation in Chinese hamster cells in vitro.

Ouabain inhibition of kidney ATPase is altered by 9.14 GHz radiation.

Cytogenetic investigations on microwaves emitted by a 455.7 MHz car phone.

[The reaction of the tick Hyalomma asiaticum (Acarina, Ixodidae) to 1- to 4-GHz microwaves].

Microwave effects on the central nervous system--a study of radar mechanics.

[The characteristics of the reactions of excitable tissue to combined exposure to microwaves and low-intensity ultrasound].

[Cumulated biological effects of microwaves and their reflection in behavior, work capacity, growth of body mass and state of brain neurons].

[Quantitative patterns in the cytogenetic action of microwaves].

[The action of microwave radiation on potassium ion transport and oxygen consumption in the perfused rat liver].

The effects of low level microwaves on the fluidity of photoreceptor cell membrane.

Insensitivity of cardiovascular function to low power cm-/mm-microwaves.

[Ultracytochemical changes in the brain and liver in exposure to low-intensity nonionizing microwave radiation].

Microwave effect on camphor binding to rat olfactory epithelium.

In vitro effects of microwave radiation on rat liver mitochondria.

Induction of neoplastic transformation in C3H/10T1/2 cells by 2.45-GHz microwaves and phorbol ester.

Evidence for microwave carcinogenesis in vitro.

Microwave radiation injury.

Effect of microwave radiation on human EEG at two different levels of exposure.

Effects of noinionizing radiation on the central nervous system, behavior, and blood: a progress report.

Brain enzyme histochemistry following stabilization by microwave irradiation.

Poly ADP ribosylation as a possible mechanism of microwave--biointeraction.

Investigation of an acute microwave-oven hand injury.

Rat lymphocytes in cell culture exposed to 2450 MHz (CW) microwave radiation.

Effect of electromagnetic microwave radiation on the growth of Ehrlich ascites carcinoma.

Effects of 36.6 GHz and static magnetic field on degree of endoreduplication in Drosophila melanogaster polytene chromosomes.

Extremely low-level microwaves attenuate immune imbalance induced by inhalation exposure to low-level toluene in mice.

[The effect of microwaves on the neuronal activity of the hyperstriatum in chick embryos at the critical developmental period].

Psychological symptoms and intermittent hypertension following acute microwave exposure.

Very new waves in very old meridians: quantum medical physics of the living.

[The effect of various occupational exposures to microwave radiation on the concentrations of immunoglobulins and T lymphocyte subsets].

Microwave effect upon chlorpromazine-inhibited kidney ATPase.

Evidence for genetic control of microwave-induced augmentation of complement receptor-bearing B lymphocytes.

[The combined action of microwave radiation and hydrogen peroxide on the viability and ultrastructure of Pseudomonas aeruginosa cells].

A demonstration of athermal effects of continuous microwave irradiation on the growth and antibiotic sensitivity of Pseudomonas aeruginosa PAO1.

Influence of low power cm-/mm-microwaves on cardiovascular function.

Local cerebral blood flow after microwave exposure.

The properties of bird feathers as converse piezoelectric transducers and as receptors of microwave radiation. II. Bird feathers as dielectric receptors of microwave radiation.

Different methods for evaluating the effects of microwave radiation exposure on the nervous system.

Microwave cell death: Immunohistochemical and enzyme histochemical evaluation.

Study of nonionizing microwave radiation effects upon the central nervous system and behavior reactions.

[Cellular effects of microwaves of thermal intensity].

Microwave induced stimulation of 32Pi incorporation into phosphoinositides of rat brain synaptosomes.

Laser doppler flowmetry as a method for evaluating the microwave radiation effect on cutaneous microcirculation.

Microwave effects on acetylcholine-induced channels in cultured chick myotubes.

[The participation of thyroid hormones in modifying the mutagenic effect of microwaves].

Investigation of the effects of continuous-wave, pulse- and amplitude-modulated microwaves on single excitable cells of Chara corallina.

Microwave frequency electromagnetic fields (EMFs) produce widespread neuropsychiatric effects including depression.

[Effect of centimeter microwaves on the antibody production in mice].

[Effect of electromagnetic SHF-radiation on the morphofunctional status of early mouse embryos].

The effect of acute far field exposure at 2.45 GHz on the mouse testis.

[Pharmacological correction of the acute effects of microwave irradiation in an experiment].

Effects of exposure to microwaves: problems and perspectives.

Semen analysis of military personnel associated with military duty assignments.

Setting exposure limits for radiofrequency radiation and microwaves in China.

Effect of exposure to operant-controlled microwaves on certain blood and immunological parameters in the young chick.

In vitro cytogenetic effects of 2450 MHz waves on human peripheral blood lymphocytes.

[Microwaves and blood-brain barrier].

Microwave radiation: an epidemiologic assessment.

[The dynamics of the immunobiological effects in transcerebral microwave exposures].

Microwave absorption by normal and tumor cells.

Microwaves induce an increase in the frequency of complement receptor-bearing lymphoid spleen cells in mice.

Microwave radiation-induced calcium ion efflux from human neuroblastoma cells in culture.

Studies on possible genetic effects of microwaves in procaryotic and eucaryotic cells.

Middle-ear structures contribute little to auditory perception of microwaves.

[Effect of long wave pre-illumination on the kinetic characteristics of microwave photoconductivity signals in Chlorella cells and the Emerson effect].

Non-thermal effects of 2.45 GHz microwaves on spindle assembly, mitotic cells and viability of Chinese hamster V-79 cells.

Microwave diathermy: the invisible healer.

[Biological effects of microwave radiation of low nonthermal intensity (regarding the maximal admissible values)].

[Effect of electromagnetic radiation of radio frequency (340 and 800 MHz) on liposomes from dimyristoyl lecithin].

[A comparative analysis of the biological action of microwaves and laser radiation].

[Studies on the microwave leakage of the interphone].

Effect of microwave radiation on the stability of frozen cefoxitin sodium solution in plastic bags.

[The role of TLR4 receptor in the stress response of lymphocytes].

Febrile convulsions induced by microwaves and the alteration in behavior of albino mouse OF1.

[Immunobiological effect of bitemporal exposure of rabbits to microwaves].

Thermal effects of 2450 MHz microwave exposure near a titanium alloy plate implanted in rabbit limbs.

[The role of protein kinase SAPK/JNK in cell responses to low-intensity nonionizing radiation].

[Activity of cytochromes P-450p and P-450h in liver microsomes and blood corticosteroid levels in experimental animals under the action of physical factors].

The effect of high intensity microwave exposure on enucleation of murine erythroid cells in vitro.

Microwave-evoked brainstem potentials in cats.

Tight junctional changes upon microwave and x-ray irradiation.

The analysis of animal bioelectric brain activity influenced by microwaves or by the introduction of strychnine.

[The characteristics of the effect of centimeter-range microwaves on drug pharmacokinetics in the body of experimental animals].

[Effect of centimeter microwaves and the combined magnetic field on the tumor necrosis factor production in cells of mice with experimental tumors].

[Enzymatic activity of some tissues and blood serum from animals and humans exposed to microwaves and hypothesis on the possible role of free radical processes in the nonlinear effects and modification of emotional behavior of animals].

Non-thermal effects in the microwave induced unfolding of proteins observed by chaperone binding.

[Study of bioeffects of ship-borne microwave navigation radar in chronic experiments].

[Combined effect of microwaves and gamma-rays on the imprinting of chickens, irradiated in early embryogenesis].

Effects of nonionizing radiation on birds.

[The effect of electromagnetic radiation on the membranes of the sarcoplasmic reticulum].

[The combined action of microwave irradiation and hypoxia on the biogenic amine content of the blood in guinea pigs in anaphylactic shock].

Possible humoral mechanism of 2450-MHz microwave-induced increase in complement receptor positive cells.

[Synaptic transmission in the frog spinal cord exposed to intensive microwave radiation].

Microwave-enhanced folding and denaturation of globular proteins.

[Hematologic changes in workers exposed to radio wave radiation].

[The reaction of glia in visual centers during the whole body effect of combined microwaves and x-rays].

Elimination of microwave effects on the vitality of nerves after blockage of active transport.

Low frequency amplitude modulated microwave fields change calcium efflux rates from synaptosomes.

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Association of microwaves and ionizing radiation: potentiation of teratogenic effects in the rat.

[The use of microwave for immunohistochemical technology in forensic pathology].

[The effect of microwaves on the bioelectric brain activity].

MoS2 nanosheets encapsulated in sodium alginate microcapsules as microwave embolization agents for large orthotopic transplantation tumor therapy.

Microwave drying of microorganisms: I. Influence of the microwave energy and of the sample thickness on the drying of yeast.

Effect of non-ionising radiation on body weight and growth of the gastro-intestinal tract in broilers.

[The role of the thyroid hormones in regulating chromosomal resistance to microwave exposure].

[Analysis of ECG on the staffs exposed to microwave in the radio calling signal station].

Cochlear microphonics generated by microwave pulses.

Holographic assessment of a hypothesized microwave hearing mechanism.

Enhancement of allergic skin wheal responses by microwave radiation from mobile phones in patients with atopic eczema/dermatitis syndrome.

Microwave antigen retrieval blocks endogenous peroxidase activity in immunohistochemistry.

[Increase in the immunogenicity of cancer cells exposed to microwaves].

Aspirin (acetylsalicylic acid) effects on behavioral thermoregulation with microwave radiation.

[The effect of microwaves on lipid peroxidation and on lipid and mineral metabolism in warm-blooded animals (experimental research)].

Effects of microwave radiation on house dust mites, Dermatophagoides pteronyssinus and Dermatophagoides farinae (Astigmata: Pyroglyphidae).

The effect of non ionising electromagnetic radiation on RAAF personnel during World War II.

[Action of millimeter-range electromagnetic radiation on the Ca pump of sarcoplasmic reticulum].

After-effect induced by microwave radiation in human electroencephalographic signal: a feasibility study.

Superconductivity--a possible mechanism for non-thermal biological effects of microwaves.

Microwaving for double indirect immunofluorescence with primary antibodies from the same species and for staining of mouse tissues with mouse monoclonal antibodies.

[Accelerated decalcification using microwaves].

Influence of chopper and mixer speeds and microwave power level during the high-shear granulation process on the final granule characteristics.

[Effects of prolonged low-intensity radiofrequency radiation in cm-range on the development of subcutaneously grafted Ehrlich's adenocarcinoma].

The effects of irradiation intensity on the microwave-enhanced advanced oxidation process.

The role of coherence time in the effect of microwaves on ornithine decarboxylase activity.

[Effect of microwaves over Staphylococcus aureus and Salmonella spp. inoculated into frozen minced meat].

Microwave decalcification of human temporal bones.

An EM radiation safety controller.

Electrosmog and autoimmune disease.

Capability of Thai Mission grass (Pennisetum polystachyon) as a new weedy lignocellulosic feedstock for production of monomeric sugar.

A novel autonomic activation measurement method for stress monitoring: non-contact measurement of heart rate variability using a compact microwave radar.

Late heat damage in normal swine rectum: a comparison of thermosensitivity of rectum and oesophagus.

[Effect of UHF and ionizing radiation on the Na-K-ATPase activity of Ehrlich ascitic carcinoma cells].

Acute multiple mononeuropathy after accidental exposure to oven microwaves.

Microwave enhanced ion exchange of cationic and anionic clays.

[New mechanisms of biological effects of electromagnetic fields].

Non-contact determination of parasympathetic activation induced by a full stomach using microwave radar.

Immunohistochemistry and microwave decalcification of human temporal bones.

Monitoring of lung edema by microwave reflectometry during lung ischemia-reperfusion injury in vivo.

Joint effects of microwave and chromium trioxide on root tip cells of Vicia faba.

An alternative approach to the treatment of mammary duct fistulas: a combination of microwave and ultrasound.

[Body's reaction to weakened geomagnetic field (the effect of magnetic deprivation)].

Influence of radar radiation on breeding biology of tits (Parus sp.).

[Effect of short-term exposure to ash from electric power plants on histochemical reactions of succinate dehydrogenase and lactate dehydrogenase in the lungs of experimental animals].

--Leaf Cluster 3 (39)

Theme - Adverse impact of wireless radiation on eye lens

Titles

[Effect of low-intensity microwave radiation on proliferation of cultured epithelial cells of rabbit lens].

Localized effects of microwave radiation on the intact eye lens in culture conditions.

[Effects of different dose microwave radiation on protein components of cultured rabbit lens].

Non-thermal electromagnetic radiation damage to lens epithelium.

Non-thermal cellular effects of lowpower microwave radiation on the lens and lens epithelial cells.

[A quantitative study on early changes in rabbit lens capsule epithelium induced by low power density microwave radiation].

[Ultrastructural change of rabbit lens epithelial cells induced by low power level microwave radiation].

Cataracts induced by microwave and ionizing radiation.

Ultrastructural changes in the rabbit lens induced by microwave radiation.

Low power density microwave radiation induced early changes in rabbit lens epithelial cells.

Effects of microwave radiation on the eye: the occupational health perspective.

[Experimental studies on the influence of millimeter radiation on light transmission through the lens].

Glutathione concentration and peptidase activity in the lens after exposure to microwaves.

Low power microwave radiation inhibits the proliferation of rabbit lens epithelial cells by upregulating P27Kip1 expression.

Microwave lens effects in humans. II. Results of five-year survey.

Changes in gap junctional intercellular communication in rabbits lens epithelial cells induced by low power density microwave radiation.

Combined microwave energy and fixative agent for cataract induction in pig eyes.

[Evaluation of lens transparency in persons exposed to electromagnetic radiation of 27--30 MHz frequency].

[Low-intensity microwave blockes cell cycle and regulate cell cycle related gene expression in rabbit lens epithelial cells].

Thermal cataract formation in rabbits.

Effects of microwave radiation on the lens epithelium in the rabbit eye.

On the microwave exposure.

Observation of microwave-induced eye lens surface motion in vitro.

Data analysis reveals significant microwave-induced eye damage in humans.

Microwave irradiation and soft contact lens parameters.

Inducing cataract in postmortem pig eyes for cataract surgery training purposes.

Dosimetric study of microwave cataractogenesis.

Evaluation of possible microwave-induced lens changes in the United States Air Force.

[Acute ocular lesions after exposure to electromagnetic radiation of ultrahigh frequency (an experimental study)].

Microwave radiation-induced chromosomal aberrations in corneal epithelium of Chinese hamsters.

The ocular effects of microwaves on hypothermic rabbits: a study of microwave cataractogenic mechanisms.

Microwave cataract and litigation: a case study.

[Biologic effects and hygienic regulation of electromagnetic fields caused by mobile communication devices].

Comments on Frey's "Data analysis reveals significant microwave-induced eye damage in humans".

Microwave-induced retinal destruction with sparing of sclera and choriocapillaris.

[The effect of chronic irradiation with intermittent unmodulated microwaves on the functional status of the rabbit].

[Hazardous health effects of microwaves and radio waves].

Microwave cyclodestruction: evaluation on human eyes.

Effects of radiofrequency radiation on rabbit kidney: a morphological and immunological study.

--Leaf Cluster 35 (107)

Theme - Adverse impacts of microwave radiation on cells and cognitive functions

Titles

[A aquaporin 4 expression and effects in rat hippocampus after microwave radiation].

Impairment of long-term potentiation induction is essential for the disruption of spatial memory after microwave exposure.

[Changes of apoptosis, mitochondrion membrane potential and Ca2+ of hypothalamic neurons induced by high power microwave].

Upregulation of HIF-1alpha via activation of ERK and PI3K pathway mediated protective response to microwave-induced mitochondrial injury in neuron-like cells.

[Microwave radiation induces injury to GC-2spd cells].

The relationship between NMDA receptors and microwave-induced learning and memory impairment: a long-term observation on Wistar rats.

Apoptosis induced by microwave radiation in pancreatic cancer JF305 cells.

[The cardiac injury effect of microwave radiation on rabbit and its mechanism].

[The injury effects of microwave exposure on visual performance and retinal ganglion cells (RGCs) in rats].

[Inhibitory effect of microwave radiation on proliferation of human pancreatic cancer JF305 cells and its mechanism].

Study on dose-dependent, frequency-dependent, and accumulative effects of 1.5 GHz and 2.856 GHz microwave on cognitive functions in Wistar rats.

Microwave induces apoptosis in A549 human lung carcinoma cell line.

Acute effects of pulsed microwaves and 3-nitropropionic acid on neuronal ultrastructure in the rat caudate-putamen.

[Influence of microwave radiation on synaptic structure and function of hippocampus in Wistar rats].

Microwave-induced Apoptosis and Cytotoxicity of NK Cells through ERK1/2 Signaling.

Identification of a Novel Rat NR2B Subunit Gene Promoter Region Variant and Its Association with Microwave-Induced Neuron Impairment.

Microwave exposure impairs synaptic plasticity in the rat hippocampus and PC12 cells through over-activation of the NMDA receptor signaling pathway.

iTRAQ quantitatively proteomic analysis of the hippocampus in a rat model of accumulative microwave-induced cognitive impairment.

The apoptotic effect and the plausible mechanism of microwave radiation on rat myocardial cells.

Neural cell apoptosis induced by microwave exposure through mitochondria-dependent caspase-3 pathway.

The effect of 2450 MHz microwave radiation on the ultrastructure of snail neurons.

Relationship between cognition function and hippocampus structure after long-term microwave exposure.

2.45 GHz Microwave Radiation Impairs Learning and Spatial Memory via Oxidative/Nitrosative Stress Induced p53-Dependent/Independent Hippocampal Apoptosis: Molecular Basis and Underlying Mechanism.

Real-time Microwave Exposure Induces Calcium Efflux in Primary Hippocampal Neurons and Primary Cardiomyocytes.

AduoLa Fuzhenglin down-regulates microwave-induced expression of beta1-adrenergic receptor and muscarinic type 2 acetylcholine receptor in myocardial cells of rats.

Alterations of cognitive function and 5-HT system in rats after long term microwave exposure.

Extracellular calcium and microwave enhancement of membrane conductance in snail neurons.

[Effect of handportable mobiletelephone microwave radiation on rat central neuron apoptosis].

[Effect of electromagnetic radiation in a decimeter wave-length range on the calcium current of molluscan neurons].

[Effects of microwave radiation on thymocytes in mice at different power densities].

Long term impairment of cognitive functions and alterations of NMDAR subunits after continuous microwave exposure.

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Retinal damage experimentally induced by microwave radiation at 55 mW/cm2.

[Microwave radiation decreases the expressions of occludin and JAM-1 in rats].

[Changes of the expression of beta1-adrenergic receptor and M2-muscarinic acetylcholine receptor in rat hearts after high power microwave radiation].

[Effect of qindan fuzheng capsule on ultrastructure of microwave radiation injured cardiomyocytes and hepatocytes in rats].

From the Cover: 2.45-GHz Microwave Radiation Impairs Hippocampal Learning and Spatial Memory: Involvement of Local Stress Mechanism-Induced Suppression of iGluR/ERK/CREB Signaling.

[Influence of microwave radiation on synapsin I expression in PC12 cells and its mechanism].

[Effect of vitamin E on morphological variation of retinal ganglion cells after microwave radiation].

[Effect of microwave radiation on primary cultured Sertoli cells].

Chronic exposure to GSM 1800-MHz microwaves reduces excitatory synaptic activity in cultured hippocampal neurons.

Microwave radiation leading to shrinkage of dendritic spines in hippocampal neurons mediated by SNK-SPAR pathway.

Activation of VEGF/Flk-1-ERK Pathway Induced Blood-Brain Barrier Injury After Microwave Exposure.

The study of retinal ganglion cell apoptosis induced by different intensities of microwave irradiation.

The effects of high-power microwaves on the ultrastructure of Bacillus subtilis.

The effect of microwave radiation on passive membrane properties of snail neurons.

[Effect of 900MHz electromagnetic fields on energy metabolism of cerebral cortical neurons in postnatal rat].

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Low intensity microwave radiation effects on the ultrastructure of Chang liver cells.

[Effect of 900Mhz electromagnetic fields on energy metabolism in postnatal rat cerebral cortical neurons].

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Protective Role of NMDAR for Microwave-Induced Synaptic Plasticity Injuries in Primary Hippocampal Neurons.

Abnormality of synaptic vesicular associated proteins in cerebral cortex and hippocampus after microwave exposure.

RKIP Regulates Neural Cell Apoptosis Induced by Exposure to Microwave Radiation Partly Through the MEK/ERK/CREB Pathway.

[The protective effects of Aduola Fuzhenglin on the heart injury induced by microwave exposure in rats].

Differentiation of murine erythroleukemic cells during exposure to microwave radiation.

Cytokines produced by microwave-radiated Sertoli cells interfere with spermatogenesis in rat testis.

[Effect of 900 MHz electromagnetic fields on the expression of GABA receptor of cerebral cortical neurons in postnatal rats].

[Effects of high power microwave exposure on cholinergic neurotrophic factors protein in rabbit retina].

[Neuroeffects of prolonged exposure to microwaves: systemic, neuronal and electron microscope study].

Microwave enhancement of membrane conductance: calmodulin hypothesis.

[Influence of electromagnetic radiation on raf kinase inhibitor protein and its related proteins of hippocampus].

2.45 GHz microwave radiation induced oxidative and nitrosative stress mediated testicular apoptosis: Involvement of a p53 dependent bax-caspase-3 mediated pathway.

Microwave effects on input resistance and action potential firing of snail neurons.

MicroRNAs: Novel Mechanism Involved in the Pathogenesis of Microwave Exposure on Rats' Hippocampus.

The Screening of Genes Sensitive to Long-Term, Low-Level Microwave Exposure and Bioinformatic Analysis of Potential Correlations to Learning and Memory.

[Pathological study of testicular injury induced by high power microwave radiation in rats].

Noise-modulated-microwave-induced response in snail neurons.

[Early ultrastructural reactions in various parts of the visual analyzer in guinea pigs after thermogenic microwave irradiation].

[Changes of rat testicular germ cell apoptosis after high power microwave radiation].

The transmission of reflexes in the spinal cord of cats during direct irradiation with microwaves.

[Lipid peroxide damage in retinal ganglion cells induced by microwave].

Calreticulin attenuated microwave radiation-induced human microvascular endothelial cell injury through promoting actin acetylation and polymerization.

[Reaction of the ultrastructure of the rat spinal ganglion to exposure to a pulsed electromagnetic field].

Specific electromagnetic effects of microwave radiation on Escherichia coli.

Microwave enhancement of membrane conductance: effects of EDTA, caffeine and tetracaine.

[Effect of Qidan Granule on PMC Derived Peptide Content and Structure of Hippocampal CA1 Region in Microwave Radiated Rats].

[The microarray study on the stress gene transcription profile in human retina pigment epithelial cells exposed to microwave radiation].

[The electroporation effects of high power pulse microwave and electromagnetic pulse irradiation on the membranes of cardiomyocyte cells and the mechanism therein involved].

Non-thermal effects of continuous 2.45 GHz microwaves on Fas-induced apoptosis in human Jurkat T-cell line.

[Zinc protective effects on pig retinal pigment epithelial cell damage of lipid peroxide induced by 2450 MHz microwave].

Pathological changes in the sinoatrial node tissues of rats caused by pulsed microwave exposure.

[High power microwave radiation damages blood-testis barrier in rats].

[Experimental analysis of biological effects of microwaves: their systemic, ultrastructural and neuronal mechanisms].

[Relationship between activation of microglia and Jaks phosphorylation induced by microwave irradiation].

Ultrastructural changes following treatment with a microwave pulse in the oocyst of Eimeria magna Perard, 1925.

The functional state of thymus cells following microwave exposure of endocrine glands.

Non-thermal effects of electromagnetic fields at mobile phone frequency on the refolding of an intracellular protein: myoglobin.

[Effect of microwaves on the expression by thymocytes of various surface membrane markers].

[Dynamics of morphological changes in the spinal cord following exposure to non-ionizing microwave radiation].

Immunoreactivity of normal rabbit serum with epinephrine (E) cells of the rat adrenal medulla after microwave antigen retrieval.

[Changes in response of neurons in visual area of cerebral cortex of rabbits to flashes of light under the influence of low-intensity physical factors of non-ionizing nature].

Ultrastructural studies of alterations induced by microwaves in Toxocara canis eggs: prophylactic interest.

Morphological changes in the liver after microwave destruction.

Studies of childhood brain tumors using immunohistochemistry and microwave technology: methodological considerations.

Cell attachment and viability on micro-arc-oxidation (MAO) microwave/hydrothermal treated titanium surface.

The role of the NF-kappaB, SAPK/JNK, and TLR4 signalling pathways in the responses of RAW 264.7 cells to extremely low-intensity microwaves.

Calreticulin stabilizes F-actin by acetylating actin and protects microvascular endothelial cells against microwave radiation.

Dual effects of microwaves on single Ca(2+)-activated K+ channels in cultured kidney cells Vero.

Microwave antigen retrieval of beta-amyloid precursor protein immunoreactivity.

Nerve agent exposure elicits site-specific changes in protein phosphorylation in mouse brain.

Cyclic AMP-dependent signaling system is a primary metabolic target for non-thermal effect of microwaves on heart muscle hydration.

[Microwaves and the visual analyzer].

Evaluation of immunohistochemical staining of human duodenal endocrine cells after microwave antigen retrieval.

[Quantitative histologic changes of the glioneuronal complex in the central and intermediate parts of the visual analyzer exposed to microwaves of thermogenic intensity].

Study of interlaboratory reliability and reproducibility of estrogen and progesterone receptor assays in Europe. Documentation of poor reliability and identification of insufficient microwave antigen retrieval time as a major contributory element of unreliable assays.

Structural changes in abdominal aorta and vena cava inferior after experimental microwave destruction.

--Leaf Cluster 39 (211)

Theme - Adverse effects from microwave radiation

Titles

[Effect of quinacrine on inflammatory reaction of blood system induced by microwave irradiation].

Cumulative effect in microwave irradiation.

[Protective effects of Genistein on human renal tubular epithelial cells damage of microwave radiation].

[Effects of occupational microwave irradiation on heat shock protein 70 expressions in rat hippocampus].

Effects of radiation on frozen lactate dehydrogenase.

Effect of microwave energy on the metabolism of Enterobacteriaceae.

[Analysis of pulsed bioelectric activity of rabbit cerebral cortex in response to low-intensity microwave radiation].

[Pro- and antioxidant effect of electromagnetic fields of extremely high frequency (460 MHz) on brain tissues in experiment].

[Pulse flows of neuronal populations of the cerebral cortex exposed to low intensity microwaves].

[Recovery responses in the bodies of rats following irradiation with microwaves (2400 MHz)].

Microwave irradiation induces neurite outgrowth in PC12m3 cells via the p38 mitogen-activated protein kinase pathway.

Application of high-powered microwave irradiation for acetylcholine analysis in mouse brain.

Effect of microwave irradiation on brain tissue structure and catecholamine distribution.

[Effect of microwave irradiation on neurocyte mitochondrial ultrastructure and mtTFA mRNA expression in rats cerebral cortex and hippocampus].

[Development of the Chlamydomonas actinochloris culture after microwave irradiation].

Reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves.

[Changes in body weight of rats during irradiation with microwaves of nonthermal intensity].

[Dependence of changes in summary bioelectric activity of the brain on low-intensity microwave irradiation from density of flow energy].

Microwave facilitation of domperidone antagonism of apomorphine-induced stereotypic climbing in mice.

[Experimental data on reaction of neurons of the brain to low-intensity package-pulsing microwave irradiation].

Growth and development of mice offspring after irradiation in utero with 2,450-MHz microwaves.

[Traumatic ulcer following microwave irradiation and local anesthesia].

Behavioral evaluation of microwave irradiation.

[Survival and physical development of progeny of Swiss mice after 2450 Mhz microwave irradiation during pregnancy].

[Effects of injurying and restoring the body of mice with microwave (2400 MHz) irradiation].

Effect of microwave irradiation on monoamine metabolism in dissected rat brain.

Effects of microwave irradiation on rat hepatic tissue evaluated by enzyme histochemistry for acid phosphatase.

The effects of microwave radiation from mobile telephones on humans and animals.

Increase of brain ammonia after microwave irradiation and its mechanism.

[Behavioral effects of the combined chronic action of 9375 and 1765 MHz microwaves].

[Effect of the agents of general anesthesia on mice after microwave irradiation].

[Effects of microwave irradiation and electrostatic field on the survival, growth and reproduction of Moina mongolica Daday].

Incidence of low-level microwave irradiation on intestinal myoelectrical activity in the rat.

Multinucleated giant cell appearance after whole body microwave irradiation of rats.

[The pathogenesis of central nervous system functional disordersafter exposure to microwave radiation].

Comparison of native and microwave irradiated DNA.

Effect of high-power density microwave irradiation on the soluble proteins of the rabbit lens.

Microwave radiation (2450-MHz) potentiates the lethal effect of endotoxin in mice.

Pulse activity of populations of cortical neurons under microwave exposures of different intensity.

Changes in the blood count of growing rats irradiated with a microwave pulse field.

[Effect of continuous low-intensity microwave irradiation on the behavior of albino rats].

Microwave irradiation and cross-linking of collagen.

[Combined effect of microwave and ionizing radiation].

Reproduction of Japanese quail after microwave irradiation (2.45 GHz CW) during embryogeny.

Psychoactive-drug response is affected by acute low-level microwave irradiation.

Microwaves and cellular immunity. I. Effect of whole body microwave irradiation on tumor necrosis factor production in mouse cells.

Radio and microwave radiation and experimental atherosclerosis.

Biosynthesis of acetylcholine in different brain regions in vivo following alternative methods of sacrifice by microwave irradiation.

Microwaves and cellular immunity. II. Immunostimulating effects of microwaves and naturally occurring antioxidant nutrients.

Effects of microwave irradiation on blood flow in the dog hindlimb.

Tissue structure of rat brain after microwave irradiation using maximum magnetic field component.

[Experimental study of the effects of acute uneven microwave irradiation].

Microwave accelerated transglycosylation of rutin by cyclodextrin glucanotransferase from Bacillus sp. SK13.002.

The effect of 2.45 GHz microwave irradiation on human peripheral lymphocytes.

[Motor activity of rabbits in conditions of chronic low-intensity pulse microwave irradiation].

Autoradiographic analysis of protein synthesis and measurements of nuclear volume in WISH cell cultures irradiated with 3 GHz electromagnetic radiation.

Leukocyte numbers during the humoral and cell-mediated immune response of Japanese quail after microwave irradiation in ovo.

The respiratory response to microwaves.

Aversion/attraction of blue jays to microwave irradiation.

Low-level microwave irradiation attenuates naloxone-induced withdrawal syndrome in morphine-dependent rats.

Response of Aspergillus nidulans and Physarum polycephalum to microwave irradiation.

[Combined action of gamma and UHF radiation on conditioned reflex behavior of rats].

Ethanol-induced hypothermia and ethanol consumption in the rat are affected by low-level microwave irradiation.

Inhibitory action of microwave radiation on gamma-glutamyl transpeptidase activity in liver of rats treated with hydrocortisone.

Effect of low-level microwave irradiation on the duodenal electrical activity of the unanesthetized rat.

Behavioral sensitivity to microwave irradiation.

In vitro microwave effects on human neutrophil precursor cells (CFU-C).

The effect of microwave irradiation on vasopressin in plasma and hypothalamo-neurohypophyseal system.

Karyometric observations of WISH cell cultures irradiated with 3 GHz microwaves.

[Effects of injurying and restoring the body of rats with microwave (2400 MHz) irradiation].

[Spontaneous electrical activity of the rat cerebral cortex during microwave irradiation].

[The immune and hormonal effects of the local action of microwaves of different intensities].

[Effects of microwave irradiation on ATPase activity and voltage dependent ion channel of rat hippocampus cell membrane].

Assessment of immune function development in mice irradiated in utero with 2450-MHz microwaves.

[Response of neurons of the sensomotor region of the cerebral cortex to low-intensity pulsed ultra-high frequency irradiation].

Effects of modulated microwave and X-ray irradiation on the activity and distribution of Ca(2+)-ATPase in small intestine epithelial cells.

[Cross-correlation analysis of the interconnection in neuronal pulses in living sections of the neocortex under the effect of microwave irradiation].

[Total bioelectric activity of various structures of the brain in low-intensity microwave irradiation].

Effects of microwave irradiation on some membrane-related processes in bacteria.

Effects of modulated and continuous microwave irradiation on the morphology and cell surface negative charge of 3T3 fibroblasts.

Microwave facilitation of methylatropine antagonism of central cholinomimetic drug effects.

[Effects of microwave acute irradiation on biomechanic properties of rabbit tissues].

Search for millimeter microwave effects on enzyme or protein functions.

[The effect of microwave irradiation on the status of the thyroid gland].

Study of the use of the microwave magnetic field for the rapid inactivation of brain enzymes.

Serum enzymes in hemorrhaged Japanese quail after microwave irradiation during embryogeny.

[Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro].

[An effect of delayed behavioral activation during a single exposure to microwaves].

[The status of the higher nervous activity in animals exposed to microwaves in conditions simulating the intermittent work of radiolocators].

[Effect of microwave irradiation on expression of heat shock proteins family in primary cultured rat hippocampal neurons].

Cytogenetic consequences of microwave irradiation on mammalian cells incubated in vitro.

[Pulse flows of populations of cortical neurons under microwave radiation: the number of burst activity].

Photic cuing of escape by rats from an intense microwave field.

Effect of microwaves on the activity of murine macrophages in vitro.

Determination of a thermal equivalent of millimeter microwaves in living cells.

Ascorbic acid changes in cultured rabbit lenses after microwave irradiation.

The response of the 22A strain of scrapie agent to microwave irradiation compared with boiling.

[Tactical behavior of rats when choosing among negative stimuli: pain or exposure to an electromagnetic field].

[Pulse flows of populations of cortical neurons under low-intensity pulsed microwave: interspike intervals].

[Pulse flows of cortical neuron populations exposed to microwaves: interspike intervals].

[Changes in the activity and conditioned-reflex behavior of white rats during and after chronic microwave irradiation].

Analysis of the effects of microwave energy on enzymatic activity of lactate dehydrogenase (LDH).

Changes of amino acid gradients in brain tissues induced by microwave irradiation and other means.

Plasma and red cell volumes of microwave irradiated mice tissues.

A comparison between microwave irradiation and decapitation: basal levels of dynorphin and enkephalin and the effect of chronic morphine treatment on dynorphin peptides.

Microwaves (2,450 MHz) suppress murine natural killer cell activity.

[Effect of impulse-intermittent ultrahigh frequency irradiation on synthesis of nucleic acids in tumor cells].

The effect of microwave irradiation on the vitality of various dermatophytes.

Focused microwave irradiation of the brain preserves in vivo protein phosphorylation: comparison with other methods of sacrifice and analysis of multiple phosphoproteins.

Brain regional levels of adenosine and adenosine nucleotides in rats killed by high-energy focused microwave irradiation.

The effect of 2450 MHz microwave radiation on histamine secretion by rat peritoneal mast cells.

Microwave-induced hearing: some preliminary theoretical observations.

[Effect of microwave radiation on regional blood flow and tissue oxygenation in the brain].

The effect of electromagnetic radiation on the hematopoietic stem cells of mice.

Reversible irritative effect of acute 2.45GHz microwave exposure on rabbit eyes--a preliminary evaluation.

Acute microwave irradiation and cataract formation in rabbits and monkeys.

Chronic non-thermal exposure of modulated 2450 MHz microwave radiation alters thyroid hormones and behavior of male rats.

Microwave-mediated enzymatic modifications of DNA.

Excellent acceleration of the Diels-Alder reaction by microwave irradiation for the synthesis of new fluorine-substituted ligands of NMDA receptor.

[Effect of 2450 MHz microwaves on the fertility of Swiss female mice].

[Myelokaryocyte mitotic activity during microwave irradiation (2375 MHz)].

Anesthesia as an effective agent against the production of congenital anomalies in mouse fetuses exposed to electromagnetic radiation.

[Stimulation of production of tumor necrosis factor by murine macrophages when exposed in vio and in vitro to weak electromagnetic waves in the centimeter range].

[Evaluation of changes in electrophysiological and hormonal parameters in rabbits resulting from short-term low-intensity ultra-high-frequency irradiation].

[The effect of millimeter-range electromagnetic and of ionizing radiation on the body and thymocytes of mice and rats].

Effect of microwave irradiation on the blow fly Chrysomya megacephala (F.) (Diptera: Calliphoridae).

Leukocyte numbers in hemorrhaged Japanese quail after microwave irradiation in ovo.

The effect of repeated microwave irradiation on the frequency of sex-linked recessive lethal mutations in Drosophila melanogaster.

[The epididymal adipose tissue of mice after nanosecond pulse-periodic microwave irradiation].

A method for dissection of discrete regions of rat brain following microwave irradiation.

Effect of microwave irradiation (2450 MHz) on murine cytotoxic lymphocyte and natural killer (NK) cells.

Dynamics of Metabolic Parameters in Rats during Repeated Exposure to Modulated Low-Intensity UHF Radiation.

[The effect of superhigh-frequency electromagnetic radiation on the course of Helicobacter pylori-associated peptic ulcer].

Effect of microwave electromagnetic field on skeletal muscle fibre activity.

[Effects of microwaves on the cellular immune response of Swiss mice].

[The effect of ultrahigh-frequency electromagnetic radiation on learning and memory processes].

Does microwave irradiation have other than thermal effects on glutaraldehyde crosslinking of collagen?

The tissue content of cyclic AMP in rats after microwave irradiation in vivo.

[Effects of microwave irradiation on NMDA receptor subunits mRNA expressions in rat hippocampus].

Plasma corticosterone in hemorrhaged Japanese quail after microwave irradiation in ovo.

[Electron microscopic analysis of the effect of modulated microwave radiation on isolated rat olfactory mucosa].

Animal study on electromagnetic field biological potency.

[The effect of low-intensity prolonged impulse electromagnetic irradiation in the UHF range on the testes and the appendages of the testis in rats].

Germ cell degeneration in normal and microwave-irradiated rats: potential sperm production rates at different developmental steps in spermatogenesis.

[Experimental research on the biological action of the pulse-modulated microwave radiation created by shipboard radar stations].

Cell-density dependent effects of low-dose ionizing radiation on E. coli cells.

[Effects of electromagnetic irradiation on glucocorticoid in serum and its receptor expression in rat hippocampus].

[The effect of microwave irradiation on the peroxide modification of low density lipoproteins in human blood serum].

[Stimulation of murine natural killer cells by weak electromagnetic waves in the centimeter range].

Use of 300-msec microwave irradiation for enzyme inactivation: a study of effects of sodium pentobarbital on acetylcholine concentration in mouse brain regions.

[Studies on the screening high yield acid protease producing strain L336 by combining microwave irradiation with chemical inducing].

[Effect of super-high electromagnetic radiation and hormones on the osmotic resistance of mouse erythrocytes].

Effects of 2.45 GHz microwave exposures on the peroxidation status in Wistar rats.

[Effect of electromagnetic waves in the centimeter range on the production of tumor necrosis factor and interleukin-3 in immunized mice].

Radiation-induced lung toxicity in mice irradiated in a strong magnetic field.

Nonthermal effect of microwave irradiation in nonaqueous enzymatic esterification.

[Role of the thyroid gland in developing the genetic effects of microwaves of nonthermal intensity].

Regional levels of cyclic AMP in rat brain: pitfalls of microwave inactivation.

[Effect of electromagnetic radiation on discharge activity of neurons in the hippocampus CA1 in rats].

Effects of repeated microwave irradiations to the albino rabbit eye.

Inactivation of kallikrein and kininases and stabilization of whole rat brain kinin levels following focused microwave irradiation.

Fragmentation of genomic DNA using microwave irradiation.

[Modifying effect of low-intensive electromagnetic radiation on the irradiated cells].

Intraoperative peritoneal washing cytology with the rapid immunoperoxidase method using microwave irradiation.

[Effect of SHF-radiation on spontaneous impulse activity of cerebral cortex slices in vitro].

[The effect of millimeter-band radiation of nonthermal intensity on sensitivity of Staphylococcus to various antibiotics].

[Infrared spectra of erythrocyte shadows in the region of the amide I and amide II bands following microwave irradiation].

[The immunological mechanism of the modulation of IgE antibody formation during microwave irradiation of the thymus].

Microwave irradiation influences on the state of human cell nuclei.

Acetylcholine: oscillation of levels in mouse brain following electroshock.

[The inhibiting action of superhigh-frequency millimeter waves on adenovirus (author's transl)].

Slow potentials and spike unit activity of the cerebral cortex of rabbits exposed to microwaves.

[The immunostimulating properties of erythrocytes subjected to the action of ultraviolet irradiation and electromagnetic radiation during vibration exposure].

GSM 900 MHz microwave radiation affects embryo development of Japanese quails.

[Immunomodulating effect of electromagnetic waves on production of tumor necrosis factor in mice with various rates of neoplasm growth].

[Clinical significance of tonsillar provocation test in diagnosis of tonsillar focal infection--by indirect irradiation of ultra-micro waves].

[The immunomodulating action of microwaves in the induction of an immune response to Vi antigen].

[A comparison of conditioned avoidance reflex in rabbits formed under the influence of permanent magnetic fields, ultra-high-frequency irradiation, light and sound].

Physiological measurements during radio-frequency irradiation.

Microwave-induced formation of oligomeric amyloid aggregates.

[Biological oxidation in cells exposed to microwaves in the millimeter range].

Brain amino acid concentrations in rats killed by decapitation and microwave irradiation.

[The effect of electromagnetic waves of very high frequency of molecular spectra of radiation and absorption of nitric oxide on the functional activity of platelets].

Exposure to low-intensive superhigh frequency electromagnetic field as a factor of carcinogenesis in experimental animals.

[Effects of centimeter waves on the immune system of mice in endotoxic shock].

Effect of continuous irradiation with terahertz electromagnetic waves of the NO frequency range on behavioral reactions of male albino rats under stress conditions.

[The effect of electromagnetic radiation with extremely high frequency and low intensity on cytotoxic activity of human natural killer cells].

Exocytosis sensitivity to growth hormone-releasing hormone in subsets of GH cells in rats under different corticosterone conditions. Ultrastructural study using microwave irradiation for fixation and immunocytochemistry.

[Effect of extremely high frequency electromagnetic radiation of low intensity on parameters of humoral immunity in healthy mice].

[The electrical activity of symmetrical areas of the rat cerebral cortex during the use of a low-intensity UHF field].

[Effect of local SHF-irradiation of the rat foot on impulse activity in the tibial nerve].

[Effect of radiofrequency of electromagnetic radiation on yeast sensitivity to fungicide antibiotics].

Effects of acute and chronic ethanol administration on thromboxane and prostacyclin levels and release in rat brain cortex.

[Effect of microwave on the dentin of root canal wall].

[Constant direct action of the magnetic field on the brain fabric].

[Functional activity and metabolism of blood neutrophils exposed to low-intensity microwaves].

[Effect of weak electromagnetic radiation on regeneration of the pharynx in Dugesia tigrina planaria].

[Effects of hypogeomagnetic fields on the structural-functional activity of rat cerebral cortex].

[Effect of microwaves of nonthermal intensity on the number of aberrant hepatocytes in rats].

[Stimulation of the defenses of trypanosomic mice by a combination of magnetic field and electromagnetic wave radiation].

Visual abnormalities associated with high-energy microwave exposure.

[Changes in the proteinase-inhibitor system of rats with hyperlipoproteinemia during transcerebral exposures to a 100-Hz-frequency pulse current and to an ultrahigh-frequency field].

Hypothalamic cholinergic and noradrenergic neurons in hyperglycemia induced by 2-deoxyglucose.

[A mathematical modelling study of the respiratory system during exposure to a low-intensity UHF field].

[Heterogeneity of neurocytes of different brain regions to repeated superhigh-frequency irradiation].

Effects of electro-acupuncture and physical exercise on regional concentrations of neuropeptides in rat brain.

Continuous microwave enhances the healing process of septic and aseptic wounds in rabbits.

[Effect of weak electromagnetic radiation on larva development and metamorphosis of grain beetle Tenebrio molitor].

[Effects of low-intensity EHF-radiation on peripheral sections of the nervous system].

[The influence of electromagnetic field on active avoidance reaction, biogenic amines and amino acids in brain of rats in spite of backround of food-stuff addition seratonus].

Visualization of in vivo metabolic flows reveals accelerated utilization of glucose and lactate in penumbra of ischemic heart.

--Leaf Cluster 29 (94)

Theme - Adverse effects of microwave radiation, especially pulsed microwave

Titles

Physiological effects of 2.8 GHz radio-frequency radiation: a comparison of pulsed and continuous-wave radiation.

Abnormal cardiovascular responses induced by localized high power microwave exposure.

Thermoregulatory responses of rats exposed to 9.3-GHz radiofrequency radiation.

Microwave alteration of the blood-brain barrier system of rats.

Blood-brain barrier permeation in the rat during exposure to low-power 1.7-GHz microwave radiation.

Low-level microwave irradiations affect central cholinergic activity in the rat.

Cerebrovascular permeability to 86Rb in the rat after exposure to pulsed microwaves.

Effects of pulsed microwave radiation on the contractile rate of isolated frog hearts.

High-peak-power microwave pulses: effects on heart rate and blood pressure in unanesthetized rats.

Microwave-induced lethal heat stress: effects of phentolamine, prazosin and metoprolol.

Low-level microwave irradiation and central cholinergic activity: a dose-response study.

Cardiorespiratory changes during microwave-induced lethal heat stress and beta-adrenergic blockade.

Tolazoline decreases survival time during microwave-induced lethal heat stress in anesthetized rats.

Studies on blood-brain barrier permeability after microwave-radiation.

Low-level microwave irradiation and central cholinergic systems.

Heart rate changes due to 5.6-GHz radiofrequency radiation: relation to average power density.

Effects of 2.8-GHz microwaves on restrained and ketamine-anesthetized rats.

Effect of 2450 MHz microwave energy on the blood-brain barrier to hydrophilic molecules. C. Effect on the permeability to [14C]sucrose.

Increased sensitivity of the non-human primate eye to microwave radiation following ophthalmic drug pretreatment.

Circulating antibody response of mice exposed to 9-GHz pulsed microwave radiation.

Immediate post-exposure effects of high-peak-power microwave pulses on operant behavior of Wistar rats.

Permeability of the blood-brain barrier to mannitol in the rat following 2450 MHz microwave irradiation.

Effects of esmolol on 35 GHz microwave-induced lethal heat stress.

Studies on microwave and blood-brain barrier interaction.

Effects of 2.45-GHz microwaves on primate corneal endothelium.

Corticotropin-releasing factor antagonist blocks microwave-induced decreases in high-affinity choline uptake in the rat brain.

Cardiovascular and thermal effects of microwave irradiation at 1 and/or 10 GHz in anesthetized rats.

Effect of 2450 MHz microwave energy on the blood-brain barrier to hydrophilic molecules. B. Effect on the permeability to HRP.

Microwave influence on the isolated heart function: II. Combined effect of radiation and some drugs.

Acute low-level microwave exposure and central cholinergic activity: studies on irradiation parameters.

Microwave irradiation of rats at 2.45 GHz activates pinocytotic-like uptake of tracer by capillary endothelial cells of cerebral cortex.

Microwave influence on the isolated heart function: I. Effect of modulation.

Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz.

Rhesus monkey behavior during exposure to high-peak-power 5.62-GHz microwave pulses.

The insensitivity of frog heart rate to pulse modulated microwave energy.

Effects of low-level microwave irradiation on hippocampal and frontal cortical choline uptake are classically conditionable.

[Comparative estimation of the effects of continuous and intermittent cyclical microwave radiation on the behavior of rats in the extraordinary situation].

In vitro studies of microwave-induced cataract: reciprocity between exposure duration and dose rate for pulsed microwaves.

Effect of 2450 MHz microwave energy on the blood-brain barrier to hydrophilic molecules. A. Effect on the permeability to sodium fluorescein.

Absorption of microwave radiation by the anesthetized rat: electromagnetic and thermal hotspots in body and tail.

Opioid receptor subtypes that mediate a microwave-induced decrease in central cholinergic activity in the rat.

Cardiovascular changes in unanesthetized and ketamine-anesthetized Sprague-Dawley rats exposed to 2.8-GHz radiofrequency radiation.

Auditory unit responses to single-pulse and twin-pulse microwave stimuli.

Naltrexone pretreatment blocks microwave-induced changes in central cholinergic receptors.

Microwave effects on isolated chick embryo hearts.

Comparative effects of extremely high power microwave pulses and a brief CW irradiation on pacemaker function in isolated frog heart slices.

Temporal bisection in rats: the effects of high-peak-power pulsed microwave irradiation.

[Effects of 2375 MHz pulse-modulated microwave radiation on ATPase activity of the rat muscle actomyosin].

Influence of microwaves on the beating rate of isolated rat hearts.

Microwave radiation and heart-beat rate of rabbits.

Effects of continuous-wave, pulsed, and sinusoidal-amplitude-modulated microwaves on brain energy metabolism.

In vitro studies of microwave-induced cataract. II. Comparison of damage observed for continuous wave and pulsed microwaves.

Antibody responses of mice exposed to low-power microwaves under combined, pulse-and-amplitude modulation.

[Proposed exposure levels of pulse-modulated electromagnetic fields].

Characteristics of microwave evoked body movements in mice.

The effect of pulsed microwaves on passive electrical properties and interspike intervals of snail neurons.

[Effect of low-intensity pulse-modulated microwave on human blood aspartate aminotransferase activity].

Effect of global system for mobile communication (GSM) microwave exposure on blood-brain barrier permeability in rat.

Slow and rapid responses to CW and pulsed microwave radiation by individual Aplysia pacemakers.

Effects of continuous and pulsed 2450-MHz radiation on spontaneous lymphoblastoid transformation of human lymphocytes in vitro.

Effects of high power microwave pulses on synaptic transmission and long term potentiation in hippocampus.

Bursting responses of Lymnea neurons to microwave radiation.

Influence of acute microwave radiation on cardiac function in normal and myocardial ischemic cats.

NF-kappaB DNA-binding activity after high peak power pulsed microwave (8.2 GHz) exposure of normal human monocytes.

Measurement of blood-brain barrier permeation in rats during exposure to 2450-MHz microwaves.

Single vs. repeated microwave exposure: effects on benzodiazepine receptors in the brain of the rat.

In vitro study of microwave effects on calcium efflux in rat brain tissue.

Thermoregulatory responses of rats exposed to 9.3-GHz microwaves: a comparison of E and H orientation.

Modification of acoustic startle by microwave pulses in the rat: a preliminary report.

Effects of high peak power microwaves on the retina of the rhesus monkey.

Alteration of circulating antibody response of mice exposed to 9-GHz pulsed microwaves.

Effect of 9.6-GHz pulsed microwaves on the orb web spinning ability of the cross spider (Araneus diadematus).

Environmental-health aspects of pulse-modulated microwaves.

[The effect of pulsed cyclical microware radiation on the conditioned behavior of rats].

[Effects of electromagnetic radiation of various modes on heart activity (in experiments)].

Modification of acoustic and tactile startle by single microwave pulses.

Effect of short electromagnetic pulses on brain acetylcholine content and spontaneous motor activity of mice.

Microwave auditory effect- a comparison of some possible transduction mechanisms.

Increased susceptibility to radiofrequency radiation due to pharmacological agents.

Microwave irradiation affects radial-arm maze performance in the rat.

[Changes in serum alkaline phosphatase activity during in vitro exposure to amplitude-modulated electromagnetic field of ultrahigh frequency (2375 MHz) in guinea pigs].

Reversible microwave effects on the blood-brain barrier.

Effect of microwave radiation on the beating rate of isolated frog hearts.

Amino acid concentrations in hypothalamic and caudate nuclei during microwave-induced thermal stress: analysis by microdialysis.

Character of the effect of microwave on conduction velocity of frog ventricular muscle.

Inter-beat intervals of cardiac-cell aggregates during exposure to 2.45 GHz CW, pulsed, and square-wave-modulated microwaves.

[The efficiency and direction of thymus changes after whole-body exposure of mice to the weak electromagnetic field are determined by the initial status of the thymus].

Alterations in alpha-adrenergic and muscarinic cholinergic receptor binding in rat brain following nonionizing radiation.

[Dependence of microwave effect on the secondary structure of DNA on molecular weight of polynucleotide].

Effects of weak amplitude-modulated microwave fields on calcium efflux from awake cat cerebral cortex.

In vivo exposure of rats to GSM-modulated microwaves: flow cytometry analysis of lymphocyte subpopulations and of mitogen stimulation.

[Microwave method of determining cerebral blood flow].

[Effects of unmodulated electromagnetic radiation of decimetric diapason on the morphogenesis of Drosophila].

Pulsed magnetic field induced "analgesia" in the land snail, Cepaea nemoralis, and the effects of mu, delta, and kappa opioid receptor agonists/antagonists.

--Leaf Cluster 31 (130)

Theme - Adverse effects of microwave exposures on rats, especially at WiFi frequencies

Titles

Behavioral effects of chronic exposure to 0.5 mW/cm2 of 2,450-MHz microwaves.

Effects of 2.45 GHz CW microwave radiation on embryofetal development in mice.

Decreased body weight in fetal rats after irradiation with 2450-MHz (CW) microwaves.

Observations of rat fetuses after irradiation with 2450-MHz (CW) microwaves.

Teratogenic, biochemical, and histological studies with mice prenatally exposed to 2.45-GHz microwave radiation.

Intermittent exposure of rats to 2450 MHz microwaves at 2.5 mW cm2: behavioral and physiological effects.

Behavioral and physiological effects of chronic 2,450-MHz microwave irradiation of the rat at 0.5 mW/cm2.

Effect of nonionizing radiation on the Purkinje cells of the rat cerebellum.

Physiological and behavioral effects of prolonged exposure to 915 MHz microwaves.

Physiological and behavioral effects of chronic exposure to 2450-MHz microwaves.

Behavioral thermoregulation in the squirrel monkey: adaptation processes during prolonged microwave exposure.

Microwave radiation (2450 MHz) alters the endotoxin-induced hypothermic response of rats.

Cardiovascular, hematologic, and biochemical effects of acute ventral exposure of conscious rats to 2450-MHz (CW) microwave radiation.

Tests of mutagenesis and reproduction in male rats exposed to 2,450-MHz (CW) microwaves.

Observations of Syrian hamster fetuses after exposure to 2450-MHz microwaves.

Nonthermal effects of mobile-phone frequency microwaves on uteroplacental functions in pregnant rats.

Serum-thyroxine levels in microwave-exposed rats.

Blood-forming system in rats after whole-body microwave exposure; reference to the lymphocytes.

The in vivo effects of 2.45 GHz microwave radiation of rabbit serum components and sleeping times.

Hematologic and immunologic effects of pulsed microwaves in mice.

Microwave-induced increase of water and conductivity in submaxillary salivary gland of rats.

Effects of whole body microwave exposure on the rat brain contents of biogenic amines.

Effects of microwave exposure in utero on embryonal, fetal and postnatal development of mice.

Effect of continuous-wave and amplitude-modulated 2.45 GHz microwave radiation on the liver and brain aminoacyl-transfer RNA synthetases of in utero exposed mice.

Influence of 2.45-GHz CW microwave radiation on spontaneously beating rat atria.

Alteration of life span of mice chronically exposed to 2.45 GHz CW microwaves.

Studies on the hematologic effects of long-term, low-dose microwave exposure.

[Development of murine embryos and fetuses after irradiation with 2450 MHz microwaves].

Thermoregulatory adjustments in squirrel monkeys exposed to microwaves at high power densities.

[Effects of microwave radiation on lipid peroxidation and the content of neurotransmitters in mice].

An evaluation of the teratogenic potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation: I. Morphologic analysis at term.

[Effects of microwave radiation on conditioned behavior of rats].

Increased serum enzyme activity in microwave-exposed rats.

Long-term, low-level microwave irradiation of rats.

Microwaves modify thermoregulatory behavior in squirrel monkey.

Testicular function of rats following exposure to microwave radiation.

Uteroplacental circulatory disturbance mediated by prostaglandin f2alpha in rats exposed to microwaves. hiro-n@po.incl.ne.jp.

Effects of microwaves on three different strains of rats.

The effect of melatonin on body mass and behaviour of rats during an exposure to microwave radiation from mobile phone.

Thermoregulatory, metabolic, and cardiovascular response of rats to microwaves.

Modification of the repeated acquisition of response sequences in rats by low-level microwave exposure.

Preliminary investigations of the effects of low-level microwave radiation on spontaneous motor activity in rats.

Delineating acute neuroendocrine responses in microwave-exposed rats.

Acute exposure to pulsed 2450-MHz microwaves affects water-maze performance of rats.

Quantitative changes in potassium, sodium, and calcium in the submaxillary salivary gland and blood serum of rats exposed to 2880-MHz microwave radiation.

Effects of hypophysectomy and dexamethasone on rat adrenal response to microwaves.

Simultaneous response of brain electrical activity (EEG) and cerebral circulation (REG) to microwave exposure in rats.

[Effects of whole-body microwave exposure on the plasma adrenocorticotropic hormone, thyroid-stimulating hormone and thyroid hormones in rats].

An evaluation of the teratogenic potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation. II. Postnatal psychophysiologic analysis.

Effects of 2.45-GHz microwave radiation on embryonic quail hearts.

Chronic exposure of rabbits to 0.5 and 5 mW/cm2 2450-MHz CW microwave radiation.

Effect of 2,450 MHz microwave radiation on the development of the rat brain.

In utero exposure to microwave radiation and rat brain development.

Thermoregulatory responses of the immature rat following repeated postnatal exposures to 2,450-MHz microwaves.

Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. I. Morphologic analysis at term.

Effects of acute low-level microwaves on pentobarbital-induced hypothermia depend on exposure orientation.

Adjustments in metabolic heat production by squirrel monkeys exposed to microwaves.

Effect of 2450 MHz microwave radiation on hematopoiesis of pregnant mice.

Natural killer cell activity reduced by microwave exposure during pregnancy is mediated by opioid systems.

Interaction of microwaves and a temporally incoherent magnetic field on spatial learning in the rat.

Effects of exposure to microwaves on cellular immunity and placental steroids in pregnant rats.

Acute, whole-body microwave exposure and testicular function of rats.

Reproduction in male Japanese quail exposed to microwave radiation during embryogeny.

Repeated exposure to low-level extremely low frequency-modulated microwaves affects baseline and scopolamine-modified electroencephalograms in freely moving rats.

Microwaves: effect on thermoregulatory behavior in rats.

Effects of microwaves on the adrenal cortex.

[Effects of whole-body microwave exposure on the plasma corticosterone, glucose, uric acid and allantoin levels in rats].

Effects of microwave exposure on the hamster immune system. IV. Spleen cell IgM hemolytic plaque formation.

Microwave irradiation and instrumental behavior in rats: unitized irradiation and behavioral evaluation facility.

Cytogenetic effects of microwave irradiation on male germ cells of the mouse.

Effect of microwave irradiation (2.45 GHz, CW) on egg weight loss, egg hatchability, and hatchling growth of the Coturnix quail.

Microwave effects on energy metabolism of rat brain.

[Experimental estimation of thermogenic levels of acute microwave exposure for different animal species].

Lethality in mice and rats exposed to 2450 MHz circularly polarized microwaves as a function of exposure duration and environmental factors.

Heat-dissipation rate of mice after microwave irradiation.

Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. II. Postnatal psychophysiologic evaluations.

Effects of 2450 MHz microwave radiation during the gestational period on the postnatal hematology of rats.

Effects of 2.45 GHz microwave radiation on the development of Japanese quail cerebellum.

Repeated exposure to low-level extremely low frequency-modulated microwaves affects cortex-hypothalamus interplay in freely moving rats: EEG study.

Comparative effects of pulsed and continuous-wave 2.8-GHz microwaves on temporally defined behavior.

[Endocrine mechanism of placental circulatory disturbances induced by microwave in pregnant rats].

Response of Japanese quail to hemorrhagic stress after exposure to microwave radiation during embryogeny.

Exposure of fertile chicken eggs to microwave radiation (2.45 GHz, CW) during incubation: technique and evaluation.

B16 melanoma development in black mice exposed to low-level microwave radiation.

Complement receptor positive spleen cells in microwave (2450-MHz)-irradiated mice.

Prolonged microwave irradiation of rats: effects on concurrent operant behavior.

Effects of microwave exposure on the hamster immune system. II. Peritoneal macrophage function.

Space efficient system for small animal, whole body microwave exposure at 1.6 GHz.

[Animal death after exposure to ultra-high frequency waves in the dependence of power flux density and specific absorption rate].

Effects of microwave exposure on the hamster immune system. I. Natural killer cell activity.

Influence of pre- and postnatal exposure of rats to 2.45-GHz microwave radiation on neurobehavioral function.

Morphological changes in cerebellum of neonatal rats exposed to 2.45 GHz microwaves.

Effect of nonionizing radiation on the Purkinje cells of the uvula in squirrel monkey cerebellum.

Pulse modulated and continuous wave microwave radiation yield equivalent changes in operant behavior of rodents.

Effects of postnatal microwave exposure on thyrotropin level in the adult male rat.

The effects of single and repeated exposure to 2.45 GHz radiofrequency fields on c-Fos protein expression in the paraventricular nucleus of rat hypothalamus.

Miniature anechoic chamber for chronic exposure of small animals to plane-wave microwave fields.

Microwaves induce peripheral vasodilation squirrel monkeys.

Some effects of exposure of the Japanese quail embryo to 2.45-GHz microwave radiation.

Protein kinase C activity in developing rat brain cells exposed to 2.45 GHz radiation.

Longevity and food consumption of microwave-treated (2.45 GHz CW) honeybees in the laboratory.

Age-dependent effect of long-term microwave radiation on postnatal neurogenesis in rats: morphological and behavioral study.

Radial arm maze performance of rats following repeated low level microwave radiation exposure.

Microwave radiation enhances teratogenic effect of cytosine arabinoside in mice.

Effects of microwaves (900 MHz) on the cochlear receptor: exposure systems and preliminary results.

Effects of GSM-900 microwaves on the experimental allergic encephalomyelitis (EAE) rat model of multiple sclerosis.

Transbilayer movement of 24Na in sonicated phosphatidylcholine vesicles exposed to frequency-modulated microwave radiation.

Antipruritic effect of millimeter waves in mice: evidence for opioid involvement.

Effects on energy absorption of orientation and size of animals exposed to 2.45-GHz microwave radiation.

Biological studies with continuous-wave radiofrequency (28 MHz) radiation.

Flight, orientation, and homing abilities of honeybees following exposure to 2.45-GHz CW microwaves.

Age-related changes in the noradrenergic pattern and receptor responses of the rat cardiovascular system after repeated microwave exposure.

Failure of rats to escape from a potentially lethal microwave field.

Humoral and cell-mediated immune function in adult Japanese Quail following exposure to 2.45-GHz microwave radiation during embryogeny.

[Which neurophysiologic effects at low level 2.45 GHz RF exposure?].

[Effects of 2450 MHz microwave on long-term potentiation of hippocampus and lipofuscin contents in rat brain].

The effect of microwave radiation on the primary IgM response to sheep red blood cells in mice.

[Effects of electromagnetic field of thermal intensity on the hypophysis-thyroid unit of the neuroendocrine system].

[Action of a UHF field on GABA-ergic and acetylcholinergic systems in synaptic transmission].

Noradrenergic innervation and receptor responses of cardiovascular tissues from young and aged rats after acute microwave exposure.

Effects of weak microwave fields amplitude modulated at ELF on EEG of symmetric brain areas in rats.

Erythropoietic dynamic equilibrium in rats maintained after microwave irradiation.

Glucose administration attenuates spatial memory deficits induced by chronic low-power-density microwave exposure.

Behavioral Abnormality along with NMDAR-related CREB Suppression in Rat Hippocampus after Shortwave Exposure.

Effects of microwave exposure on the hamster immune system. III. Macrophage resistance to vesicular stomatitis virus infection.

Retrograde amnesia: effects of handling and microwave radiation.

[Effect of high frequency electromagnetic fields on the processes of transamination in the liver and small intestine tissues of rats].

Influence of postnatal exposition to microwaves on brain and hypothalamo-pituitary monoamines in the adult male rat.

Electric power induction through an isolated intestinal pouch.

[Effect of microwaves on the spike activity of cerebellar Purkinje cells in the cat].

Fourth Level Cluster 79 (428)
Theme - Microwave radiation absorption at different frequencies

--Leaf Cluster 10 (75)

Theme - Dielectric properties of tissue at different microwave frequencies

Titles

The UHF and microwave dielectric properties of normal and tumour tissues: variation in dielectric properties with tissue water content.

Changes in the dielectric properties of rat tissue as a function of age at microwave frequencies.

Dielectric properties of muscle and liver from 500 MHz-40 GHz.

Dielectric properties of tissues; variation with age and their relevance in exposure of children to electromagnetic fields; state of knowledge.

A large-scale study of the ultrawideband microwave dielectric properties of normal, benign and malignant breast tissues obtained from cancer surgeries.

A quick accurate method for measuring the microwave dielectric properties of small tissue samples.

A method for in vivo detection of abnormal subepidermal tissues based on dielectric properties.

Microwave method for determing dielectric parameters of living biological objects I.

Microwave dielectric studies on proteins, tissues, and heterogeneous suspensions.

Dielectric properties of porcine brain tissue in the transition from life to death at frequencies from 800 to 1900 MHz.

A large-scale study of the ultrawideband microwave dielectric properties of normal breast tissue obtained from reduction surgeries.

Dielectric property measurement of ocular tissues up to 110 GHz using 1 mm coaxial sensor.

Radio-frequency and microwave dielectric properties of insects.

Dielectric properties of Co-gamma-irradiated and microwave-heated rat tumour and skin measured in vivo between 0.2 and 2.4 GHz.

Microwave dielectric relaxation in muscle. A second look.

Dielectric properties of animal tissues in vivo at radio and microwave frequencies: comparison between species.

Dielectric properties of rat embryo and foetus as a function of gestation.

Dielectric properties of porcine cerebrospinal tissues at microwave frequencies: in vivo, in vitro and systematic variation with age.

Dielectric properties of insect tissues.

Development of anatomically realistic numerical breast phantoms with accurate dielectric properties for modeling microwave interactions with the human breast.

Dielectric properties at microwave frequencies studied in partially filled cylindrical TE011 cavities.

Dielectric properties of human brain tissue measured less than 10 h postmortem at frequencies from 800 to 2450 MHz.

[Dielectric properties of human sweat fluid in the microwave range].

Dielectric behavior of DNA solution at radio and microwave frequencies (at 20 degrees C).

A heterogeneous breast phantom for microwave breast imaging.

Dielectric properties of supersaturated alpha-D-glucose aqueous solutions at 2450 MHz.

Variation of the dielectric properties of tissues with age: the effect on the values of SAR in children when exposed to walkie-talkie devices.

Microwave dielectric properties of tissue. Some comments on the rotational mobility of tissue water.

Monitoring water content of rat lung tissue in vivo using microwave reflectometry.

Microwave dielectric properties and thermochemical characteristics of the mixtures of walnut shell and manganese ore.

Microwave-induced thermal imaging of tissue dielectric properties.

Dielectrical model of cellular structures in radio frequency and microwave spectrum. Electrically interacting versus noninteracting cells.

Average dielectric property analysis of complex breast tissue with microwave transmission measurements.

Modeling of the dielectric properties of trabecular bone samples at microwave frequency.

Dielectric properties for non-invasive detection of normal, benign, and malignant breast tissues using microwave theories.

Microwave dielectric measurements (0.8-70 GHz) on Artemia cysts at variable water content.

An evaluation of the mutagenic, carcinogenic and teratogenic potential of microwaves.

Microwave dielectric analysis of human stratum corneum in vivo.

Dielectric properties of human ovary follicular fluid at 9.2 GHz.

A macroscopic model of lungs and a material simulating their properties at radio and microwave frequencies.

Microwave dielectric measurements and tissue characteristics of the human brain: potential in localizing intracranial tissues.

Theoretical evaluation of dielectric absorption of microwave energy at the scale of nucleic acids.

40 GHz RF biosensor based on microwave coplanar waveguide transmission line for cancer cells (HepG2) dielectric characterization.

Modeling of noninvasive microwave characterization of breast tumors.

Cole-Cole parameters for the dielectric properties of porcine tissues as a function of age at microwave frequencies.

The dielectric properties of normal and tumour mouse tissue between 50 MHz and 10 GHz.

Dielectric Properties for Differentiating Normal and Malignant Thyroid Tissues.

The dielectric properties of the cerebellum, cerebrum and brain stem of mouse brain at radiowave and microwave frequencies.

Effect of ultraviolet light on the dielectric behavior of bone at microwave frequencies.

Microwave dielectric measurements of erythrocyte suspensions.

Electrical properties of lens material at microwave frequencies.

A semi-automatic method for developing an anthropomorphic numerical model of dielectric anatomy by MRI.

Microwave absorption in aqueous solutions of DNA.

The measured electrical properties of normal and malignant human tissues from 50 to 900 MHz.

Numerical assessment of the reduction of specific absorption rate by adding high dielectric materials for fetus MRI at 3 T.

Non-invasive and continuous monitoring of the sol-gel phase transition of supramolecular gels using a fast (open-ended coaxial) microwave sensor.

Carbon-coated CoFe-CoFe2O4 composite particles with high and dual-band electromagnetic wave absorbing properties.

A microwave radiometric method for the study of the semiconductor properties of living tissue: its potential application to tumour location.

Theoretical evaluation of the distributed power dissipation in biological cells exposed to electric fields.

[Mechanism of microwave radiation absorption by biological membranes].

A generalized model for the interaction of microwave radiation with bound water in biological material.

Understanding physical mechanism of low-level microwave radiation effect.

Analytical approximations in multiple scattering of electromagnetic waves by aligned dielectric spheroids.

A novel discrete particle swarm optimization algorithm for estimating dielectric constants of tissue.

Multi-physics modeling to study the influence of tissue compression and cold stress on enhancing breast tumor detection using microwave radiometry.

Microwave facilities for welding thermoplastic composites and preliminary results.

Biological effects of low-level environmental agents.

Multifunctional composites: optimizing microstructures for simultaneous transport of heat and electricity.

The properties of bird feathers as converse piezoelectric transducers and as receptors of microwave radiation. I. Bird feathers as converse piezoelectric transducers.

Brain banks and non nervous tissues.

Characterization of three iron ferredoxins by microwave power saturation.

[Possible mechanisms of aftereffects of GSM electromagnetic radiation on air-dry seeds].

Microwave grafted, composite and coprocessed materials: drug delivery applications.

Production of a Novel Mineral-based Sun Lotion for Protecting the Skin from Biohazards of Electromagnetic Radiation in the UV Region.

Microwave drying remediation of petroleum-contaminated drill cuttings.

--Leaf Cluster 23 (88)

Theme - Specific absorption rate in human body models

Titles

Body effects on SAR distributions for microwave exposures in a realistic model of the human head.

Analysis of SAR distribution in human head of antenna used in wireless power transform based on magnetic resonance.

FDTD chiral brain tissue model for specific absorption rate determination under radiation from mobile phones at 900 and 1800 MHz.

FDTD calculations of specific energy absorption rate in a seated voxel model of the human body from 10 MHz to 3 GHz.

Development of a rat head exposure system for simulating human exposure to RF fields from handheld wireless telephones.

Radio frequency electromagnetic exposure: tutorial review on experimental dosimetry.

SAR calculations in an anatomically realistic model of the head for mobile communication transceivers at 900 MHz and 1.8 GHz.

Specific absorption rate (SAR) in models of the human head exposed to hand-held UHF portable radios.

Initial analysis of SAR from a cell phone inside a vehicle by numerical computation.

Dosimetry associated with exposure to non-ionizing radiation: very low frequency to microwaves.

SAR versus S(inc): What is the appropriate RF exposure metric in the range 1-10 GHz? Part I: Using planar body models.

Whole-body and local dosimetry in rats exposed to 2.45-GHz microwave radiation.

Specific absorption rate in rats exposed to 2,450-MHz microwaves under seven exposure conditions.

Electromagnetic fields: human safety issues.

Microwave radiation absorption in the rat: frequency-dependent SAR distribution in body and tail.

Observing-responses of rats exposed to 1.28- and 5.62-GHz microwaves.

Multibody effects on microwave power absorption by multilayered cylindrical models of man.

Numerical compliance testing of human exposure to electromagnetic radiation from smart-watches.

A simulation for effects of RF electromagnetic radiation from a mobile handset on eyes model using the finite-difference time-domain method.

Outdoor measurement of SAR in a full-sized human model exposed to 29.9 MHz in the near field.

Effects of frequency, irradiation geometry and polarisation on computation of SAR in human brain.

SAR in a child voxel phantom from exposure to wireless computer networks (Wi-Fi).

Preliminary studies: far-field microwave dosimetric measurements of a full-scale model of man.

Numerical simulation of pressure waves in the cochlea induced by a microwave pulse.

The effects of RF absorbers on exposure levels at 100 MHz.

Comparison of numerical and experimental methods for determination of SAR and radiation patterns of handheld wireless telephones.

Dominant factors influencing whole-body average SAR due to far-field exposure in whole-body resonance frequency and GHz regions.

Numerical evaluation of human exposure to WiMax patch antenna in tablet or laptop.

Far-field microwave dosimetry in a rhesus monkey model.

A method for safety testing of radiofrequency/microwave-emitting devices using MRI.

Local exposure system for rats head using a figure-8 loop antenna in 1500-MHz band.

Analytic SAR computation in a multilayer elliptic cylinder for bioelectromagnetic applications.

Computation of high-resolution SAR distributions in a head due to a radiating dipole antenna representing a hand-held mobile phone.

Thermal mapping on male genital and skin tissues of laptop thermal sources and electromagnetic interaction.

Acute dosimetry and estimation of threshold-inducing behavioral signs of thermal stress in rabbits at 2.45-GHz microwave exposure.

Comparison of Thermal Response for RF Exposure in Human and Rat Models.

Systems for exposing mice to 2,450-MHz electromagnetic fields.

A suggested limit for population exposure to radiofrequency radiation.

SAR distribution in a bio-medium in close proximity with dual segment cylindrical dielectric resonator antenna.

A comparative study of the PIFA and printed monopole antenna EM absorption.

Compact shielded exposure system for the simultaneous long-term UHF irradiation of forty small mammals. II. Dosimetry.

Thermal effects of radiation from cellular telephones.

SAR in rats exposed in 2,450-MHz circularly polarized waveguides.

A formula for human average whole-body SARwb under diffuse fields exposure in the GHz region.

Scaling the physiological effects of exposure to radiofrequency electromagnetic radiation: consequences of body size.

Radiofrequency dosimetry in subjects implanted with metallic straight wires: a numerical study.

Further studies of human whole-body radiofrequency absorption rates.

Estimation of whole-body SAR from electromagnetic fields using personal exposure meters.

Numerical modelling of thermal effects in rats due to high-field magnetic resonance imaging (0.5-1 GHZ).

Dosimetry for a study of effects of 2.45-GHz microwaves on mouse testis.

SAR exposure from UHF RFID reader in adult, child, pregnant woman, and fetus anatomical models.

Head and neck resonance in a rhesus monkey--a comparison with results from a human model.

On the averaging area for incident power density for human exposure limits at frequencies over 6 GHz.

Dosimetric study on eye's exposure to wide band radio frequency electromagnetic fields: variability by the ocular axial length.

Absorption of microwave energy by muscle models and by birds of differing mass and geometry.

Influence of electromagnetic polarization on the whole-body averaged SAR in children for plane-wave exposures.

Thermal effects of MR imaging: worst-case studies on sheep.

Comparison of dose dependences for bioeffects of continuous-wave and high-peak power microwave emissions using gel-suspended cell cultures.

An attempt at quantitative specification of SAR distribution homogeneity.

A new method of SAR determination in animals exposed to microwave/radiofrequency radiation (MW/RFR).

Metabolic and vasomotor responses of rhesus monkeys exposed to 225-MHz radiofrequency energy.

Exposure assessment of one-year-old child to 3G tablet in uplink mode and to 3G femtocell in downlink mode using polynomial chaos decomposition.

Harmful effects of 41 and 202 MHz radiations on some body parts and tissues.

Whole-body new-born and young rats' exposure assessment in a reverberating chamber operating at 2.4 GHz.

A 3-D hp finite/infinite element method to calculate power deposition in the human head.

Ocular effects of radiofrequency energy.

The development of biomedical approaches and concepts in radiofrequency radiation protection.

Absorbed energy distribution from radiofrequency electromagnetic radiation in a mammalian cell model: effect of membrane-bound water.

Statistical analysis of whole-body absorption depending on anatomical human characteristics at a frequency of 2.1 GHz.

Computational human model VHP-FEMALE derived from datasets of the national library of medicine.

Modeling the detectability of vesicoureteral reflux using microwave radiometry.

Radio-wave exposure of the human head: analytical study based on a versatile eccentric spheres model including a brain core and a pair of eyeballs.

Millimeter-wave absorption by cutaneous blood vessels: a computational study.

Simple method to measure power density entering a plane biological sample at millimeter wavelengths.

Exposure of Insects to Radio-Frequency Electromagnetic Fields from 2 to 120 GHz.

[Use of dose parameters of UHF irradiation in the interpretation of lethal effects in laboratory animals].

Noninvasive measurement of current in the human body for electromagnetic dosimetry.

Exposure to non-ionizing radiation provokes changes in rat thyroid morphology and expression of HSP-90.

Induced EM fields inside human bodies irradiated by EM waves of up to 500 MHz.

A dual vial waveguide exposure facility for examining microwave effects in vitro.

[Estimation of the restricted area related to the limitation of exposure of the general public to electromagnetic fields in the vicinity of microwave relay antenna systems].

FDTD simulation of electromagnetic wave scattering from retina cells.

Effect of metal-framed spectacles on microwave radiation hazards to the eye of humans.

Effect of insertion depth on helical antenna performance in a muscle-equivalent phantom.

A survey of the urban radiofrequency (RF) environment.

[The evaluation of the consequences of electromagnetic irradiation of hands in operators of high-frequency welding devices].

Dosimetry considerations in far field microwave exposure of mammalian cells.

[Changes of neurocytes in CNS under general exposure to UHF field with local protection applied].

--Leaf Cluster 21 (63)

Theme - Adverse effects of millimeter-wave exposures on biological systems

Titles

[Effects of millimeter wave irradiation with different frequency and power density on their offsprings in mice].

[Relationship between millimeter wave irradiation in pregnant mice and c-Fos protein expression in hippocampus and learning and memory functions in their offsprings].

Gene expression changes in the skin of rats induced by prolonged 35 GHz millimeter-wave exposure.

[Effect of low intensity of electromagnetic radiation in the centimeter and millimeter range on proliferative and cytotoxic activity of murine spleen lymphocytes].

[Effects of millimeter wave on gene expression in human keratinocytes].

Current state and implications of research on biological effects of millimeter waves: a review of the literature.

Acute ocular injuries caused by 60-Ghz millimeter-wave exposure.

A non-thermal effect of millimeter wave radiation on the puffing of giant chromosomes.

Effects of millimeter waves on ionic currents of Lymnaea neurons.

Evaluation of the potential in vitro antiproliferative effects of millimeter waves at some therapeutic frequencies on RPMI 7932 human skin malignant melanoma cells.

[Acoustic detection of absorption of millimeter-band electromagnetic waves in biological objects].

Reception of low-intensity millimeter-wave electromagnetic radiation by the electroreceptors in skates.

Effect of cyclophosphamide and 61.22 GHz millimeter waves on T-cell, B-cell, and macrophage functions.

Frequency and irradiation time-dependant antiproliferative effect of low-power millimeter waves on RPMI 7932 human melanoma cell line.

Hypothalamic effects of millimeter wave irradiation depend on location of exposed acupuncture zones in unanesthetized rabbits.

Some basic properties of biological tissues for potential biomedical applications of millimeter waves.

[The electrical activity of the hypothalamus in exposure to millimeter-wave radiation at biologically active points].

Electromagnetic millimeter wave induced hypoalgesia: frequency dependence and involvement of endogenous opioids.

Comparison of blood pressure and thermal responses in rats exposed to millimeter wave energy or environmental heat.

Modulation of neuronal activity and plasma membrane properties with low-power millimeter waves in organotypic cortical slices.

Thermal modeling of millimeter wave damage to the primate cornea at 35 GHz and 94 GHz.

Thermal response of tissues to millimeter waves: implications for setting exposure guidelines.

[Activity of natural killer cells of the spleen of mice exposed to low-intensity of extremely high frequency electromagnetic radiation].

Study of narrow band millimeter-wave potential interactions with endoplasmic reticulum stress sensor genes.

Effect of millimeter waves on natural killer cell activation.

Transmission electron microscopy study of the effects produced by wide-band low-power millimeter waves on MCF-7 human breast cancer cells in culture.

Morphological changes in skin nerves caused by electromagnetic radiation of the millimeter range.

The use of millimeter wavelength electromagnetic waves in cardiology.

Millimeter wave exposure reverses TPA suppression of gap junction intercellular communication in HaCaT human keratinocytes.

Millimeter wave induced reversible externalization of phosphatidylserine molecules in cells exposed in vitro.

[The simulation of the cooperative effect of development in a culture of early mouse embryos after irradiation with electromagnetic waves in the millimeter range].

Effects of Millimeter-Wave Electromagnetic Radiation on the Experimental Model of Migraine.

Effect of 99 GHz continuous millimeter wave electro-magnetic radiation on E. coli viability and metabolic activity.

Effect of low-intensity millimeter wave electromagnetic radiation on regeneration of the sciatic nerve in rats.

Millimeter waves thermally alter the firing rate of the Lymnaea pacemaker neuron.

Numerical model of heat transfer in the rabbit eye exposed to 60-GHz millimeter wave radiation.

[Power density analysis on millimeter waves irradiated into cell monolayers in culture dishes].

Effect of millimeter waves on cyclophosphamide induced suppression of the immune system.

Immunomodulating action of low intensity millimeter waves on primed neutrophils.

Suppression of pain sensation caused by millimeter waves: a double-blinded, cross-over, prospective human volunteer study.

[The effect of continuous millimeter low-intensity radiation on the Na+ ion transport in the frog skin].

[Effects of millimeter wave on gap junctional intercellular communication in human keratinocytes].

[The effect of electromagnetic radiation in the millimeter-wave range on the immune status of peptic ulcer patients].

Effect of millimeter waves on cyclophosphamide induced suppression of T cell functions.

Millimeter-wave effects on electric activity of crayfish stretch receptors.

Reflection and penetration depth of millimeter waves in murine skin.

Search for frequency-specific effects of millimeter-wave radiation on isolated nerve function.

Millimeter wave-induced modulation of calcium dynamics in an engineered skin co-culture model: role of secreted ATP on calcium spiking.

Millimeter wave absorption in the nonhuman primate eye at 35 GHz and 94 GHz.

[Effect of extremely high-frequency electromagnetic radiation on the function of skin sensory endings].

Induced movements of giant vesicles by millimeter wave radiation.

Sustained 35-GHz radiofrequency irradiation induces circulatory failure.

[The effects of electromagnetic radiation of extremely high frequency and low intensity on the growth rate of bacteria Escherichia coli and the role of medium pH].

[Effect of coherent extremely high-frequency and low-intensity electromagnetic radiation on the activity of membrane systems in Escherichia coli].

Effect of millimeter waves on cyclophosphamide induced NF-kappaB.

[Resonance effect of coherent millimeter-band electromagnetic waves on living organisms].

Large Metasurface Aperture for Millimeter Wave Computational Imaging at the Human-Scale.

[The effect of millimeter-range electromagnetic radiation on the evoked potentials from the vestibular cortical area of the cerebral hemispheres (an experimental study)].

Long-lasting (fatiguing) activity of isolated muscle fibres influenced by microwave electromagnetic field.

The mechanisms of athermal microwave biological effects.

[Experimental study on possibility of corneal injury by electromagnetic wave].

Multi-center feasibility study of microwave radiometry thermometry for non-invasive differential diagnosis of arterial disease in diabetic patients with suspected critical limb ischemia.

Tagging frogs with passive integrated transponders causes disruption of the cutaneous bacterial community and proliferation of opportunistic fungi.

--Leaf Cluster 44 (95)

Theme – Adverse effects of microwave resonances in biological systems

Titles

Thermal Response of Human Skin to Microwave Energy: A Critical Review.

Tissue models for RF exposure evaluation at frequencies above 6 GHz.

Thermal models for microwave hazards and their role in standards development.

A thermal model for human thresholds of microwave-evoked warmth sensations.

Modeling thermal responses in human subjects following extended exposure to radiofrequency energy.

Physiological interaction processes and radio-frequency energy absorption.

Human exposure at two radio frequencies (450 and 2450 MHz): similarities and differences in physiological response.

Vibrational resonances in biological systems at microwave frequencies.

Thermoregulatory physiologic responses in the human body exposed to microwave radiation.

[Dosimetric aspects in studying the biological action of nonionizing electromagnetic radiation].

High-resolution simulations of the thermophysiological effects of human exposure to 100 MHz RF energy.

Thermal Modeling for the Next Generation of Radiofrequency Exposure Limits: Commentary.

Microwave-induced pressure waves in a model of muscle tissue.

Radiofrequency energy on cortical bone and soft tissue: a pilot study.

[Role of polarization and resonance in assessing the biological effects of electromagnetic radiation].

Microwave challenges to the thermoregulatory system.

Physiologic regulation in electromagnetic fields.

Thermal Response of In Vivo Human Skin to Fractional Radiofrequency Microneedle Device.

Impact of monopolar radiofrequency energy on subchondral bone viability.

A comparative study of human sensory thresholds: 2450-MHz microwaves vs far-infrared radiation.

Energy deposition processes in biological tissue: nonthermal biohazards seem unlikely in the ultra-high frequency range.

Generalized model of the microwave auditory effect.

Thermophysiological responses of human volunteers during controlled whole-body radio frequency exposure at 450 MHz.

Mechanical and biochemical effect of monopolar radiofrequency energy on human articular cartilage: an in vitro study.

The influence of radiofrequency/microwave energy absorption on physiological regulation.

Considerations for human exposure standards for fast-rise-time high-peak-power electromagnetic pulses.

[Mechanisms of biophysical effects of microwaves].

Effects of electromagnetic radiation on the Q of quartz resonators.

Energy issues in microwave food processing: A review of developments and the enabling potentials of solid-state power delivery.

Biophysical limits on athermal effects of RF and microwave radiation.

Auditory response to pulsed radiofrequency energy.

Thresholds for lenticular damage in the rabbit eye due to single exposure to CW microwave radiation: an analysis of the experimental information at a frequency of 2.45 GHz.

A Closer Look at the Thresholds of Thermal Damage: Workshop Report by an ICNIRP Task Group.

Theory of the anomalous resonant absorption of DNA at microwave frequencies.

Intrinsic and roughness-induced absorption of electromagnetic radiation incident on optical surfaces.

Ultrawide-band electromagnetic pulses induced hypotension in rats.

Ultrawide-band electromagnetic pulses induced hypotension in rats.

2D plasmon excitation and nonthermal effects of microwaves on biological membranes.

Thresholds of microwave-evoked warmth sensations in human skin.

The effect of radiofrequency energy on the ultrastructure of joint capsular collagen.

[Resonance interactions of surface charged lipid vesicles with the microwave electromagnetic field].

Synchronization in a mechanical resonator array coupled quadratically to a common electromagnetic field mode.

Ovicidal levels of 2.45 GHz electromagnetic energy for the southern corn rootworm.

On the possibility of nonthermal biological effects of pulsed electromagnetic radiation.

Fine structural alterations in radiofrequency energy-induced lesions in dog hearts: possible basis for reduced arrhythmic complications.

Microwave and RF hazard standard considerations.

A model of the electric field of the brain at EEG and microwave frequencies.

[The peculiarities of the microwave in the frequency range of 51-52 GHz spectrum effects on E. coli cells].

[Electromagnetic radiation in the radiofrequency range: radiation safety].

Ultrashort microwave signals: a didactic discussion.

A cooperative model for Ca(++) efflux windowing from cell membranes exposed to electromagnetic radiation.

Electromagnetic-field exposure and cancer.

Monte Carlo simulations of electromagnetic wave scattering from a random rough surface with three-dimensional penetrable buried object: mine detection application using the steepest-descent fast multipole method.

Microwave absorption by magnetite: a possible mechanism for coupling nonthermal levels of radiation to biological systems.

Nonlinear changes in brain electrical activity due to cell phone radiation.

Thermoregulatory responses of febrile monkeys during microwave exposure.

[Experiment with the local effect of superhigh-frequency electromagnetic energy on biologically active points].

Monte Carlo simulations for scattering of electromagnetic waves from perfectly conductive random rough surfaces.

[Electromagnetic radiofrequency radiation (microwaves): principles and criteria of standardization, threshold dose levels].

Development of a hybrid microwave-optical tissue oxygenation probe to measure thermal response in the deep tissue.

[Dependence of anti-inflammatory effects of high peak-power pulsed electromagnetic radiation of extremely high frequency on exposure parameters].

Electrical discontinuity of tissue substitute models at 27.12 MHz.

Analysis of strain-induced EPR-line shapes and anisotropic spin-lattice relaxation in a [2Fe-2S] ferredoxin.

The human skin as a sub-THz receiver - Does 5G pose a danger to it or not?

Effects of microwave radiation on living tissues.

Monitoring variations of biological impedances using microwave Doppler radar.

Scaling Relationship of In Vivo Muscle Contraction Strength of Rabbits Exposed to High-Frequency Nanosecond Pulse Bursts.

Biophysical injury mechanisms in electrical shock trauma.

Dynamic nuclear polarisation of biological matter.

Microwave imaging using the finite-element method and a sensitivity analysis approach.

[An evaluation of absorbed doses of high energy electromagnetic radiation in radiotherapy of laryngeal cancer].

[The effects of pulsed low-level EM fields on memory processes].

Ultrawideband radiation and pentylenetetrazol-induced convulsions in rats.

Multi-Center Pilot Study to Evaluate the Safety Profile of High Energy Fractionated Radiofrequency With Insulated Microneedles to Multiple Levels of the Dermis.

Propagation of an electromagnetic wave in an absorbing anisotropic medium and infrared transmission of liquid crystals: comparison with experiments.

Microwave medical imaging based on sparsity and an iterative method with adaptive thresholding.

Microwave-field-driven acoustic modes in DNA.

Long-term study of 435 MHz radio-frequency radiation on blood-borne end points in cannulated rats. Part I: Engineering considerations.

Comment I on "Generation of focused, nonspherically decaying pulses of electromagnetic radiation"

Moisture Monitoring in Fluid-Bed Granulation by Multi-Resonance Microwave Sensor: Applicability on Crystal-Water Containing Donepezil Granules.

[Immunotropic effects of electromagnetic fields in the range of radio- and microwave frequencies].

Multiple scattering of electromagnetic waves by an array of parallel gyrotropic rods.

Human leukocyte functions and the U.S. safety standard for exposure to radio-frequency radiation.

[Biological and ecological aspects of the effects combined electromagnetic rays on farm animals].

A model of cell electromagnetic susceptibility associated with the membrane electric field.

An algorithm to derive the fraction of photosynthetically active radiation absorbed by photosynthetic elements of the canopy (FAPAR(ps)) from eddy covariance flux tower data.

Transfer of light-induced electron-spin polarization from the intermediary acceptor to the prereduced primary acceptor in the reaction center of photosynthetic bacteria.

A signal-to-noise standard for pulsed EPR.

[Effect of decimeter polarized electromagnetic radiation on germinating capacity of seeds].

Multifrequency electron paramagnetic resonance study on deproteinized human bone.

Measurement of heart rate variability and stress evaluation by using microwave reflectometric vital signal sensing.

Frequency selective solutions for an efficient non-ionising radiation protection in the radiofrequency and microwave ranges.

How might spatial nonuniformity of dose in a homogeneous biological system affect its total response?

EM-field effect upon properties of NADPH-cytochrome P-450 reductase with model substrates.

Comment II on "Generation of focused, nonspherically decaying pulses of electromagnetic radiation"

--Leaf Cluster 47 (107)

Theme - Adverse biological effects of decimeter waves

Titles

[Energy and plastic metabolism of the heart muscle in rabbits undergoing thyroid irradiation with decimeter waves].

[The action of decimeter waves and merkazolil on myocardial metabolism in the rabbit and its hormonal regulation].

[The immunological and hormonal effects of combined exposure to a bitemporal ultrahigh-frequency electrical field and to decimeter waves at different sites].

[Effect of electromagnetic radiation of the decimetric wave range on myocardium cell membranes].

[Role of ultrasonic dopplerography in monitoring the effectiveness of treatment of patients who have sustained a stroke with decimeter-range electromagnetic waves].

[Dynamic ultrastructural shifts in the cardiomyocytes during the irradiation of the cardiac area with decimeter electromagnetic waves].

[Myocardial energy metabolism in decimeter-wave exposures].

Modulation of a compressional electromagnetic wave in a magnetized electron-positron quantum plasma.

Interference of electromagnetic waves in dynamic metabolism.

Electromagnetic wave scattering from a rough interface above a chiral medium: generalized field transforms.

[Ultrastructure of the cerebral cortex in the rat after the effect of electromagnetic impulse].

Information transfer by electromagnetic waves in cortex layers.

Levy noise improves the electrical activity in a neuron under electromagnetic radiation.

Response of Electrical Activity in an Improved Neuron Model under Electromagnetic Radiation and Noise.

Possible microwave mechanisms of the mammalian nervous system.

[The effect of decimeter waves on the metabolism of the myocardium and its hormonal regulation in rabbits with experimental ischemia].

[Ultrastructure of cells of the lateral field of the hypothalamus of the cat after exposure to electromagnetic radiation].

[Morphological changes in the thyroid and adrenals under the bitemporal action of a UHF electrical field and decimeter waves (experimental research)].

Some neurotropic effects of low-intensity electromagnetic waves in rats with different typological characteristics of higher nervous activity.

[Systematic analysis of the state of man exposed to radio wave irradiation for a long time].

Poly(dimethylsilylene)diacetylene-Guided ZIF-Based Heterostructures for Full Ku-Band Electromagnetic Wave Absorption.

[The brain function of animals exposed to the action of centimeter electromagnetic waves].

Modulation of coherence of vectorial electromagnetic waves in the Young interferometer.

[Effect of electromagnetic waves with 59-63 GHz frequency on myocardial infarct patients in the subacute stage].

Reflection and transmission of electromagnetic waves at a temporal boundary.

Effect of low-intensity millimeter-range electromagnetic irradiation on the recovery of function in lesioned sciatic nerves in rats.

[Changes in physico-chemical parameters of homeopathic remedies ferrum metallicum CH6 and ferrum metallicum CH30 after exposure to high frequency electromagnetic radiation of low intensity].

Multi-functional coding metasurface for dual-band independent electromagnetic wave control.

[Changes in intracellular regeneration and the indices of endocrine function and cardiac microcirculation in exposure to decimeter waves].

Multiple scattering of electromagnetic waves by an aggregate of uniaxial anisotropic spheres.

Low power radio-frequency and microwave effects on human electroencephalogram and behavior.

Electrophysiological effects of non-invasive Radio Electric Asymmetric Conveyor (REAC) on thalamocortical neural activities and perturbed experimental conditions.

[Electromagnetic radiation of non-thermal intensity and short exposition as a sub-threshold irritant for the central nervous system].

[The effects of influence of electromagnetic irradiation of millimeter wavelength on background impulse activity of supraoptic nucleus' neurons of rats' hypothalamus].

Current problems of nonionizing radiation.

MOF-Derived Porous Co/C Nanocomposites with Excellent Electromagnetic Wave Absorption Properties.

Hierarchical neuronal modeling of cognitive functions: from synaptic transmission to the Tower of London.

Electromagnetic wave absorbing properties of amorphous carbon nanotubes.

[Electromagnetic radiation damage to the retina (author's transl)].

Prediction and measurement of the electromagnetic environment of high-power medium-wave and short-wave broadcast antennas in far field.

Hierarchical neuronal modeling of cognitive functions: from synaptic transmission to the Tower of London.

Out of time: a possible link between mirror neurons, autism and electromagnetic radiation.

About the biological effects of high and extremely high frequency electromagnetic fields.

Exact description of free electromagnetic wave fields in terms of rays.

[Health disorders caused by radiation].

Nanometer-scale surface modification of epoxy with carbon black and electromagnetic waves.

The histologic effects of pulsed and continuous radiofrequency lesions at 42 degrees C to rat dorsal root ganglion and sciatic nerve.

[Effect of low intensity and ultra high frequency electromagnetic irradiation on memory functions].

[The enhanced lethality of cells in suspension during simultaneous exposure to pulsed electrical and shock-wave acoustic fields].

Food collection and response to pheromones in an ant species exposed to electromagnetic radiation.

Morphology-Control Synthesis of a Core-Shell Structured NiCu Alloy with Tunable Electromagnetic-Wave Absorption Capabilities.

[Degranulation of skin mast cells caused by high frequency electromagnetic irradiation of low intensity].

[The general patterns in the development of the ultrastructural reactions under the action of electromagnetic radiations].

Controlling Energy Radiations of Electromagnetic Waves via Frequency Coding Metamaterials.

Intensity statistics and the finesse of electromagnetic radiation in random structures.

Effects of the action of microwave-frequency electromagnetic radiation on the spike activity of neurons in the supraoptic nucleus of the hypothalamus in rats.

[Immunosuppressive effect of the decimeter-band electromagnetic field].

A Route to Chaotic Behavior of Single Neuron Exposed to External Electromagnetic Radiation.

[Radiosensitivity of morphoenzymological structural elements of the jejunum mucous membrane in chronodynamics of the impact of electromagnetic fields impulses].

Radiofrequency neurolysis in a clinical model. Neuropathological correlation.

[Experimental research on the electromagnetic radiation immunity of a kind of portable monitor].

Possible physical substrates for the interaction of electromagnetic fields with biologic membranes.

Modifications in ventricular fibrillation and capture capacity induced by a linear radiofrequency lesion.

Evaluation of the maximum permissible level of low-intensity electromagnetic radiation at mobile connection frequency (1 GHz) by changes in motor activity of Spirostomum Ambiguum.

Post-mortem histologic evaluation of microwave lesions after epicardial pulmonary vein isolation for atrial fibrillation.

Modulation of surface electromagnetic waves.

[Adaptive changes in the body upon exposure to electromagnetic radiation].

[Effect of impulse extrabroad-band electromagnetic radiation on electroencephalogram and sleep in laboratory animals].

[Influence of ultra-high electromagnetic irradiation on the electrophoretic mobility of erythrocytes].

Nanocomposite synthesis by absorption of nanoparticles into macroporous hydrogels. Building a chemomechanical actuator driven by electromagnetic radiation.

Spontaneous bodily rotations and direction of locomotion at different times after radio frequency lesions at sites in and near the substantia nigra.

Effect of lesion morphology on microwave signature in 2-D ultra-wideband breast imaging.

[The evaluation of the body response of experimental animals to exposure to the magnetic component of electromagnetic radiation for setting a hygiene standard].

Quantitative analysis of lesion parameters in radiofrequency trigeminal rhizotomy.

Features of electromagnetic radiation time-and-frequency fluctuation intensity distributions from human brain structures.

[The effects of space flight factors on the central nervous system. Structural-functional aspects of radio-modifying action].

[Dissipative functions of processes of electromagnetic radiation interaction with biological objects].

[Specific and non-specific electromagnetic irradiation effects on biological objects].

[Changes in gastric electric activity and serum catecholamine level under the influence of electromagnetic microwaves (experimental studies)].

[Features of control of electromagnetic radiation emitted by personal computers].

[Phenomenology and genesis of changes in the total bioelectrical activity of the brain in response to electromagnetic radiation].

Nano sulfur particles decorated bi-lamella composites for superior electromagnetic wave absorption.

Meridian is a three-dimensional network from bio-electromagnetic radiation interference: an interference hypothesis of meridian.

Modulational instability of electromagnetic waves in birefringent fibers with periodic and random dispersion.

[Effect of extremely low frequency electromagnetic radiation and ultra-violet radiation on aggregation of thymocytes and erythrocytes].

Numerical study of electromagnetic waves interacting with negative index materials.

[The structural dynamics of the afferent flow in the action on the receptor field of a low-intensity stimulant].

General description of electromagnetic radiation processes based on instantaneous charge acceleration in "endpoints".

[Behavior of the human skin under the influence of electromagnetic radiation in the visible and near infrared region (author's transl)].

Physical modalities other than stretch in spastic hypertonia.

[Ultrastructure of skeletal muscle tissue of microwave damaged chick embryos].

Response to pulsed and continuous radiofrequency lesioning of the dorsal root ganglion and segmental nerves in patients with chronic lumbar radicular pain.

The biological effectiveness of solar electromagnetic radiation in space.

[Myocardial damage after high tension electricity injury in rabbits].

Bacterial transformation using micro-shock waves.

[Effect of electromagnetic irradiation of the millimetric range on hemodynamics in patients with arterial hypertension].

The biological effects of solar activity.

Mode of action of Phoneutria nigriventer spider venom at the isolated phrenic nerve-diaphragm of the rat.

GSM 900 MHz radiation inhibits ants' association between food sites and encountered cues.

Extending human perception of electromagnetic radiation to the UV region through biologically inspired photochromic fuzzy logic (BIPFUL) systems.

[Ecological and hygienic studies of electromagnetic irradiation of navigation safety system in Eastern area of the Finnish Gulf].

Failure of chronic exposure to nonthermal FM radio waves to mutate Drosophila.

[Normal doses of visible light can cause mutations in skin].

Effect of cyclooxygenase blockade on blood flow through well-developed coronary collateral vessels.

Long-wavelength red light emission from TV and photosensitive siezures.

The aversive effect of electromagnetic radiation on foraging bats: a possible means of discouraging bats from approaching wind turbines.

Bilateral symmetry of local inflammatory activation in human carotid atherosclerotic plaques.

Fourth Level Cluster 82 (529)
Theme - Adverse effects of mobile phone radiation, especially oxidative stress

--Leaf Cluster 22 (127)

Theme - Effects of radiofrequency radiation, especially from mobile phones, on rats

Titles

Effects of electromagnetic field produced by mobile phones on the oxidant and antioxidant status of rats.

Nanometer-scale elongation rate fluctuations in the Myriophyllum aquaticum (Parrot feather) stem were altered by radio-frequency electromagnetic radiation.

Nanometer-scale elongation rate fluctuations in the Myriophyllum aquaticum (Parrot feather) stem were altered by radio-frequency electromagnetic radiation.

The effect of electromagnetic radiation in the mobile phone range on the behaviour of the rat.

Radio frequency electromagnetic radiation (RF-EMR) from GSM (0.9/1.8GHz) mobile phones induces oxidative stress and reduces sperm motility in rats.

The effects of radiofrequency electromagnetic radiation on sperm function.

Effects of folic acid on rat kidney exposed to 900 MHz electromagnetic radiation.

[Experimental justification of possible mechanisms of action of low intensity electromagnetic radiation (EMR) on animals' behavior].

Modulation of mammalian immunity by electromagnetic radiation.

Recent reports of Wi-Fi and mobile phone-induced radiation on oxidative stress and reproductive signaling pathways in females and males.

The radioprotective effects of Moringa oleifera against mobile phone electromagnetic radiation-induced infertility in rats.

Protective Effects of Zinc on 2.45 GHz Electromagnetic Radiation-Induced Oxidative Stress and Apoptosis in HEK293 Cells.

Long-term exposure to 4G smartphone radiofrequency electromagnetic radiation diminished male reproductive potential by directly disrupting Spock3-MMP2-BTB axis in the testes of adult rats.

The effect of pulsed electromagnetic radiation from mobile phone on the levels of monoamine neurotransmitters in four different areas of rat brain.

Long-term exposure of 2450MHz electromagnetic radiation induces stress and anxiety like behavior in rats.

The impact of electromagnetic radiation (2.45 GHz, Wi-Fi) on the female reproductive system: The role of vitamin C.

Electromagnetic radiation influence on nonlinear charge and energy transport in biosystems.

900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: protection by vitamins E and C.

Probing the Origins of 1,800 MHz Radio Frequency Electromagnetic Radiation Induced Damage in Mouse Immortalized Germ Cells and Spermatozoa in vitro.

Effects of the exposure to mobile phones on male reproduction: a review of the literature.

Neurobiological effects of microwave exposure: a review focused on morphological findings in experimental animals.

Impact of 2.45 GHz microwave radiation on the testicular inflammatory pathway biomarkers in young rats: The role of gallic acid.

Liver antioxidant stores protect the brain from electromagnetic radiation (900 and 1800 MHz)-induced oxidative stress in rats during pregnancy and the development of offspring.

Effects of electromagnetic radiation from a cellular telephone on epidermal Merkel cells.

Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits.

Electromagnetic radiation 2450 MHz exposure causes cognition deficit with mitochondrial dysfunction and activation of intrinsic pathway of apoptosis in rats.

Protective effects of beta-glucan against oxidative injury induced by 2.45-GHz electromagnetic radiation in the skin tissue of rats.

[Possible modification of radiation injury using radio frequency electromagnetic radiation].

[Role of phospholipase A2 and epoxygenase in inhibition of respiration burst in neutrophils by low intensity radiation of extremely high frequency].

Effects of acute and chronic exposure to both 900 MHz and 2100 MHz electromagnetic radiation on glutamate receptor signaling pathway.

Behavior and memory evaluation of Wistar rats exposed to 1.8 GHz radiofrequency electromagnetic radiation.

[Biological effects of electromagnetic radiation of extremely high frequencies combined with physiologically active compounds].

Non-ionizing electromagnetic radiation and cancer--is there a relationship?

Benefits and hazards of electromagnetic waves, telecommunication, physical and biomedical: a review.

[Suppression of nonspecific resistance of the body under the effect of extremely high frequency electromagnetic radiation of low intensity].

The impact of exposure of diabetic rats to 900 MHz electromagnetic radiation emitted from mobile phone antenna on hepatic oxidative stress.

Analysis of emotionality and locomotion in radio-frequency electromagnetic radiation exposed rats.

[Effects of extremely high-frequency electromagnetic radiation on the immune system and systemic regulation of homeostasis].

Challenging cell phone impact on reproduction: a review.

Radiofrequency electromagnetic radiation-induced behavioral changes and their possible basis.

Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells.

[Electromagnetic radiations from computer video terminals and their effect on health].

Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro.

[FEATURES OF MODIFYING EFFECT OF LOW-INTENSITY ELECTROMAGNETIC RADIATION OF NATURAL AND TECHNOGENIC ORIGIN ON VIABILITY AND FUNCTIONAL STATUS OF NEUTROPHILIC GRANULOCYTES].

Effects of electromagnetic radiation from a cellular phone on human sperm motility: an in vitro study.

[Intracellular regeneration of adrenocorticocytes in response to the prophylactic application of low-intensity electromagnetic radiation under the conditions of radiation (an experimental study)].

Electromagnetic radiation--parameters for risk assessment.

Effect of 1.8 GHz radiofrequency electromagnetic radiation on novel object associative recognition memory in mice.

[Epidemiologic studies of the effect of microwaves (neurophysiologic, hematologic and ophthalmologic aspects)].

[The activity of prooxidant-antioxidant system in loach embryos under the action of microwave radiation].

The effects of electromagnetic radiation (2450 MHz wireless devices) on the heart and blood tissue: role of melatonin.

Immune responses of a wall lizard to whole-body exposure to radiofrequency electromagnetic radiation.

Features of anti-inflammatory effects of modulated extremely high-frequency electromagnetic radiation.

Effect of exposure and withdrawal of 900-MHz-electromagnetic waves on brain, kidney and liver oxidative stress and some biochemical parameters in male rats.

Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation.

Possible cause for altered spatial cognition of prepubescent rats exposed to chronic radiofrequency electromagnetic radiation.

Polarization: A Key Difference between Man-made and Natural Electromagnetic Fields, in regard to Biological Activity.

Exposure to acute electromagnetic radiation of mobile phone exposure range alters transiently skin homeostasis of a model of pigmented reconstructed epidermis.

Structural and ultrastructural study of rat liver influenced by electromagnetic radiation.

[Effects of low-intensity electromagnetic radiation of extremely high frequency on the animal body within the framework of total low-dose x-ray irradiation].

[Metabolic and ultrastructural adaptation mechanisms during the primary prophylactic action of low-intensity electromagnetic radiation under normal and radiation conditions].

Exposure of tumor-bearing mice to extremely high-frequency electromagnetic radiation modifies the composition of fatty acids in thymocytes and tumor tissue.

The antioxidant effect of Green Tea Mega EGCG against electromagnetic radiation-induced oxidative stress in the hippocampus and striatum of rats.

The chronic effect of pulsed 1800 MHz electromagnetic radiation on amino acid neurotransmitters in three different areas of juvenile and young adult rat brain.

[Decrease in the intensity of the cellular immune response and nonspecific inflammation upon exposure to extremely high frequency electromagnetic radiation].

Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.

Model analysis of nonlinear modification of neutrophil calcium homeostasis under the influence of modulated electromagnetic radiation of extremely high frequencies.

Hippocampal lipidome and transcriptome profile alterations triggered by acute exposure of mice to GSM 1800 MHz mobile phone radiation: An exploratory study.

[The specific features of the development of metabolic and regenerative processes under the action of low-intensity electromagnetic radiation in radiation exposure conditions (an experimental study)].

Impact of electromagnetic radiation emitted by monitors on changes in the cellular membrane structure and protective antioxidant effect of vitamin A - In vitro study.

Mobile Phone Radiation: Physiological & Pathophysiologcal Considerations.

Fatty Acid Content and Tumor Growth Changes in Mice After Exposure to Extremely High-Frequency Electromagnetic Radiation and Consumption of N-3 Fatty Acids.

Disordered redox metabolism of brain cells in rats exposed to low doses of ionizing radiation or UHF electromagnetic radiation.

[Modulated extremely high frequency electromagnetic radiation of low intensity activates or inhibits respiratory burst in neutrophils depending on modulation frequency].

The role of fatty acids in anti-inflammatory effects of low-intensity extremely high-frequency electromagnetic radiation.

[Effect of low intensity pulse-modulated electromagnetic radiation on activity of alkaline phosphatase in blood serum].

The effect of electromagnetic radiation on the rat brain: an experimental study.

[Protective action of electromagnetic radiation (40.68 MHz) on Saccharomyces cerevisiae UCM Y-517].

Radiofrequency electromagnetic radiation exposure effects on amygdala morphology, place preference behavior and brain caspase-3 activity in rats.

[Influence of electromagnetic fields on the emotional behaviour of rats].

Testicular apoptosis and histopathological changes induced by a 2.45 GHz electromagnetic field.

[The application of low-intensity electromagnetic radiation under immobilization stress conditions (an experimental study)].

Extremely high-frequency electromagnetic radiation enhances neutrophil response to particulate agonists.

Inhibition by Egb761 of the effect of cellphone radiation on the male reproductive system.

Variations in amino acid neurotransmitters in some brain areas of adult and young male albino rats due to exposure to mobile phone radiation.

[Effect of radio-frequency electromagnetic radiation on physiological features of Saccharomyces cerevisiae strain UCM Y-517].

Variations of melatonin and stress hormones under extended shifts and radiofrequency electromagnetic radiation.

[Effect of radiofrequency range electromagnetic radiation on chemoreceptor structure].

Effects of intensive cell phone (Philips Genic 900) use on the rat kidney tissue.

Effects of prenatal and postnatal exposure of Wi-Fi on development of teeth and changes in teeth element concentration in rats. [corrected].

Changes in mitochondrial functioning with electromagnetic radiation of ultra high frequency as revealed by electron paramagnetic resonance methods.

Effect of electromagnetic waves on human reproduction.

Effects of short-duration electromagnetic radiation on early postnatal neurogenesis in rats: Fos and NADPH-d histochemical studies.

[The effect of electromagnetic radiation on the monoamine oxidase A activity in the rat brain].

[Changes in the immune status under the influence of high-frequency electromagnetic radiation].

The effect of low frequency electromagnetic radiation on the morphology of dental and periodontal tissues (experimental investigation).

Structural and ultrastructural study of rat testes influenced by electromagnetic radiation.

[The combined action of drinking mineral water and low-intensity electromagnetic radiation under the immobilization stress conditions (an experimental study)].

Effects of low-intensity ultrahigh frequency electromagnetic radiation on inflammatory processes.

[Effect of Low-Intensity 900 MHz Frequency Electromagnetic Radiation on Rat Brain Enzyme Activities Linked to Energy Metabolism].

Adverse cutaneous effects of ionizing and non-ionizing electromagnetic radiation.

[The influence of electromagnetic radiation of industrial frequency on Daphnia magna (Straus)].

Effects of electromagnetic radiation from 3G mobile phone on heart rate, blood pressure and ECG parameters in rats.

[Mechanism of radiobiological effects of low intensity nonionizing electromagnetic radiation].

Transdermal patches loaded with L-cysteine HCL as a strategy for protection from mobile phone emitting electromagnetic radiation hazards.

Effect of delta-rhythm-modulated extremely high frequency electromagnetic radiation on rats.

[Effect of hypokinetic stress and low intensity electromagnetic field of extremely high frequency on changes of cytokine concentration in rat blood].

Non-ionizing electromagnetic radiations, emitted by a cellular phone, modify cutaneous blood flow.

Low frequency electromagnetic waves increase human sperm motility - A pilot study revealing the potent effect of 43 kHz radiation.

Biological effects of electromagnetic fields and radiation.

[Status quo of the researches on the biological effect of electromagnetic radiation on the testis and epididymal sperm].

[Effect of low intensity electromagnetic waves from cell phones on human health].

[Impact of cell phone radiation on male reproduction].

Mobile phones electromagnetic radiation and NAD(+)-dependent isocitrate dehydrogenase as a mitochondrial marker in asthenozoospermia.

Electromagnetic radiation emitted from video computer terminals.

[Effect of low intensity and very high frequency electromagnetic radiation on occupationally exposed personnel].

[Effects of electromagnetic radiation in metropolis environment on teenagers' electrocardiogram and blood cells].

Influence of electromagnetic radiation produced by mobile phone on some biophysical blood properties in rats.

[Influence of light and electromagnetic radiation of Sun on circadian rhythms of the total antioxidant capacity of human saliva in the North].

[Radar radiation damages sperm quality].

[Influence of electromagnetic radiation on toxicity of Vipera lebetina obtusa venom].

[On prevention of a combined impact of electromagnetic radiation and climatic/weather factors on worker's organism].

Propagation of electromagnetic radiation in mitochondria?

The effect of low level radiofrequency electromagnetic radiation on the excretion rates of stress hormones in operators during 24-hour shifts.

[Physical factors and stress].

[Effect of weak electromagnetic radiation on learning in the grain beetle Tenebrio monitor].

[Disturbances of glucose tolerance in workers exposed to electromagnetic radiation].

--Leaf Cluster 26 (129)

Theme - Oxidative stress effects from mobile phone radiofrequency radiation

Titles

Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain.

Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants N-acetyl cysteine and epigallocatechin-gallate.

The link between radiofrequencies emitted from wireless technologies and oxidative stress.

The protective effects of N-acetyl-L-cysteine and epigallocatechin-3-gallate on electric field-induced hepatic oxidative stress.

Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin.

Effect of 900 MHz radiofrequency radiation on oxidative stress in rat brain and serum.

The protective effect of caffeic acid phenethyl ester (CAPE) on oxidative stress in rat liver exposed to the 900 MHz electromagnetic field.

Exposure to radiofrequency radiation induces oxidative stress in duckweed Lemna minor L.

The prophylactic effect of vitamin C on oxidative stress indexes in rat eyes following exposure to radiofrequency wave generated by a BTS antenna model.

Vitamin C protects rat cerebellum and encephalon from oxidative stress following exposure to radiofrequency wave generated by a BTS antenna model.

Effects of Electromagnetic Radiation Use on Oxidant/Antioxidant Status and DNA Turn-over Enzyme Activities in Erythrocytes and Heart, Kidney, Liver, and Ovary Tissues From Rats: Possible Protective Role of Vitamin C.

Long-term exposure to electromagnetic radiation from mobile phones and Wi-Fi devices decreases plasma prolactin, progesterone, and estrogen levels but increases uterine oxidative stress in pregnant rats and their offspring.

Oxidative stress and prevention of the adaptive response to chronic iron overload in the brain of young adult rats exposed to a 150 kilohertz electromagnetic field.

Effect of 900-, 1800-, and 2100-MHz radiofrequency radiation on DNA and oxidative stress in brain.

Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain.

Selenium supplementation ameliorates electromagnetic field-induced oxidative stress in the HEK293 cells.

Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats.

Effects of mobile phones on oxidant/antioxidant balance in cornea and lens of rats.

Effects of acute exposure to the radiofrequency fields of cellular phones on plasma lipid peroxide and antioxidase activities in human erythrocytes.

Exposure to static magnetic field of pregnant rats induces hepatic GSH elevation but not oxidative DNA damage in liver and kidney.

Effects of 837 and 1950 MHz radiofrequency radiation exposure alone or combined on oxidative stress in MCF10A cells.

Oxidative stress effects on the central nervous system of rats after acute exposure to ultra high frequency electromagnetic fields.

A cross-sectional study on oxidative stress in workers exposed to extremely low frequency electromagnetic fields.

Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats.

The effect of melatonin on the liver of rats exposed to microwave radiation.

Oxidative stress-mediated alterations on sperm parameters in male Wistar rats exposed to 3G mobile phone radiation.

Wi-Fi (2.45 GHz)- and mobile phone (900 and 1800 MHz)-induced risks on oxidative stress and elements in kidney and testis of rats during pregnancy and the development of offspring.

Effects of Low-Frequency Electromagnetic Field on Oxidative Stress in Selected Structures of the Central Nervous System.

Effect of low level microwave radiation exposure on cognitive function and oxidative stress in rats.

900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues.

Exposure to mobile phone (900-1800 MHz) during pregnancy: tissue oxidative stress after childbirth.

The Effects of Melatonin on Oxidative Stress Parameters and DNA Fragmentation in Testicular Tissue of Rats Exposed to Microwave Radiation.

Exposure to cell phone induce oxidative stress in mice preantral follicles during in vitro cultivation: An experimental study.

[Effect of American Ginseng Capsule on the liver oxidative injury and the Nrf2 protein expression in rats exposed by electromagnetic radiation of frequency of cell phone].

Effects of acute electromagnetic field exposure and movement restraint on antioxidant system in liver, heart, kidney and plasma of Wistar rats: a preliminary report.

Effect of mobile phone exposure on apoptotic glial cells and status of oxidative stress in rat brain.

Effects of third generation mobile phone-emitted electromagnetic radiation on oxidative stress parameters in eye tissue and blood of rats.

Pathological Findings Observed in the Kidneys of Postnatal Male Rats Exposed to the 2100 MHz Electromagnetic Field.

Effects of exposure to 50 Hz electric field at different strengths on oxidative stress and antioxidant enzyme activities in the brain tissue of guinea pigs.

Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain.

Biochemical modifications and neuronal damage in brain of young and adult rats after long-term exposure to mobile phone radiations.

The preventive effect of lotus seedpod procyanidins on cognitive impairment and oxidative damage induced by extremely low frequency electromagnetic field exposure.

900-MHz microwave radiation promotes oxidation in rat brain.

Influence of extremely-low-frequency magnetic field on antioxidative melatonin properties in AT478 murine squamous cell carcinoma culture.

2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, Mus musculus.

In vitro free radical scavenging activities and effect of synthetic oligosaccharides on antioxidant enzymes and lipid peroxidation in aged mice.

The physiopathological effects of quercetin on oxidative stress in radiation of 4.5 g mobile phone exposed liver tissue of rat.

Assessment of oxidant/antioxidant status in saliva of cell phone users.

Effects of 900-MHz electromagnetic field emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs.

Chronic exposure to 50Hz magnetic fields causes a significant weakening of antioxidant defence systems in aged rat brain.

Effects of static magnetic field and cadmium on oxidative stress and DNA damage in rat cortex brain and hippocampus.

Melatonin protects rat thymus against oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes.

Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation.

The effects of N-acetylcysteine and epigallocatechin-3-gallate on liver tissue protein oxidation and antioxidant enzyme levels after the exposure to radiofrequency radiation.

Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin.

Long term exposure to cell phone frequencies (900 and 1800 MHz) induces apoptosis, mitochondrial oxidative stress and TRPV1 channel activation in the hippocampus and dorsal root ganglion of rats.

Impacts of exposure to 900 MHz mobile phone radiation on liver function in rats.

Static magnetic field affects oxidative stress in mouse cochlea.

Effects of cell phone radiation on lipid peroxidation, glutathione and nitric oxide levels in mouse brain during epileptic seizure.

The effect of electromagnetic radiation emitted by display screens on cell oxygen metabolism - in vitro studies.

Effects of electromagnetic radiation produced by 3G mobile phones on rat brains: magnetic resonance spectroscopy, biochemical, and histopathological evaluation.

Role of Mitochondria in the Oxidative Stress Induced by Electromagnetic Fields: Focus on Reproductive Systems.

Effect of selenium pre-treatment on plasma antioxidant vitamins A (retinol) and E (alpha-tocopherol) in static magnetic field-exposed rats.

Effect of cell phone use on salivary total protein, enzymes and oxidative stress markers in young adults: a pilot study.

Radiofrequency radiation emitted from Wi-Fi (2.4 GHz) causes impaired insulin secretion and increased oxidative stress in rat pancreatic islets.

[Corrective effects of electromagnetic radiation in a millimeter wavelength range on the parameters of oxidative stress after standard anti-helicobacterial therapy in patients with ulcer disease].

Oxidative effects of extremely low frequency magnetic field and radio frequency radiation on testes tissues of diabetic and healthy rats.

Investigation of the effects of distance from sources on apoptosis, oxidative stress and cytosolic calcium accumulation via TRPV1 channels induced by mobile phones and Wi-Fi in breast cancer cells.

Radiofrequency electromagnetic radiation from cell phone causes defective testicular function in male Wistar rats.

Effect of 950 MHz UHF electromagnetic radiation on biomarkers of oxidative damage, metabolism of UFA and antioxidants in the livers of young rats of different ages.

The influence of microwave radiation from cellular phone on fetal rat brain.

Effects of chronic exposure to 950 MHz ultra-high-frequency electromagnetic radiation on reactive oxygen species metabolism in the right and left cerebral cortex of young rats of different ages.

Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats.

Radiations and male fertility.

Electromagnetic radiation (Wi-Fi) and epilepsy induce calcium entry and apoptosis through activation of TRPV1 channel in hippocampus and dorsal root ganglion of rats.

Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices.

[Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA)--in vitro study].

[Effects of extremely low frequency electromagnetic field and its combination with lead on the antioxidant system in mouse].

Mobile phone (1800MHz) radiation impairs female reproduction in mice, Mus musculus, through stress induced inhibition of ovarian and uterine activity.

[Interference of vitamin E on the brain tissue damage by electromagnetic radiation of cell phone in pregnant and fetal rats].

Evaluation of genotoxic effects in male Wistar rats following microwave exposure.

1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, Mus musculus.

The 2100MHz radiofrequency radiation of a 3G-mobile phone and the DNA oxidative damage in brain.

[On the mechanism of cytogenetic effect of electromagnetic radiation: a role of oxidation homeostasis].

Neuroprotective effects of dietary supplement Kang-fu-ling against high-power microwave through antioxidant action.

Effect of extremely low frequency magnetic field on antioxidant activity in plasma and red blood cells in spot welders.

Antioxidants alleviate electric field-induced effects on lung tissue based on assays of heme oxygenase-1, protein carbonyl content, malondialdehyde, nitric oxide, and hydroxyproline.

Cell phone electromagnetic field radiations affect rhizogenesis through impairment of biochemical processes.

GSM base station electromagnetic radiation and oxidative stress in rats.

The Effects of Cell Phone Waves (900 MHz-GSM Band) on Sperm Parameters and Total Antioxidant Capacity in Rats.

Extremely low frequency electromagnetic field reduces oxidative stress during the rehabilitation of post-acute stroke patients.

Evaluation of selected biochemical parameters in the saliva of young males using mobile phones.

Protein oxidation under extremely low frequency electric field in guinea pigs. Effect of N-acetyl-L-cysteine treatment.

Selenium supplementation ameliorates static magnetic field-induced disorders in antioxidant status in rat tissues.

In vitro effects of 50 Hz magnetic fields on oxidatively damaged rabbit red blood cells.

[Protective effect of Liuweidihuang Pills against cellphone electromagnetic radiation-induced histomorphological abnormality, oxidative injury, and cell apoptosis in rat testes].

The impact of electromagnetic radiation of different parameters on platelet oxygen metabolism - in vitro studies.

Melatonin attenuates radiofrequency radiation (900 MHz)-induced oxidative stress, DNA damage and cell cycle arrest in germ cells of male Swiss albino mice.

The effect of 50 hz magnetic field of different shape on oxygen metabolism in blood platelets: in vitro studies.

[Protective effect of melatonin and vitamin E against prooxidative action of iron ions and static magnetic field].

The role of zinc supplementation in the inhibition of tissue damage caused by exposure to electromagnetic field in rat lung and liver tissues.

The influence of 1800 MHz GSM-like signals on hepatic oxidative DNA and lipid damage in nonpregnant, pregnant, and newly born rabbits.

Effect of rosmarinic acid on sertoli cells apoptosis and serum antioxidant levels in rats after exposure to electromagnetic fields.

Effects of electromagnetic radiation exposure on bone mineral density, thyroid, and oxidative stress index in electrical workers.

[Electromagnetic radiation of the terahertz range at the nitric oxide frequency in correction and prophylaxis of functional activity disorders in thrombocytes of white rats under long-term stress].

Wi-Fi is an important threat to human health.

The influence of 1800 MHz GSM-like signals on blood chemistry and oxidative stress in non-pregnant and pregnant rabbits.

[Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1)--in vitro researches].

[Effects of nano-selenium on cognition performance of mice exposed in 1800 MHz radiofrequency fields].

Mobile phone usage and male infertility in Wistar rats.

Evidence of oxidative stress in American kestrels exposed to electromagnetic fields.

[Use of terahertz electromagnetic radiation at nitric oxide frequencies for the correction of thyroid functional state during stress].

Correction of microcirculatory disturbances with terahertz electromagnetic radiation at nitric oxide frequencies in albino rats under conditions of acute stress.

The effect of Wi-Fi electromagnetic waves on neuronal response properties in rat barrel cortex.

Electromagnetic wave emitting products and "Kikoh" potentiate human leukocyte functions.

[Influence of 900 MHz frequency electromagnetic radiation on some blood indices].

Effects of electromagnetic waves emitted from 3G+wi-fi modems on human semen analysis.

Reactive oxygen species elevation and recovery in Drosophila bodies and ovaries following short-term and long-term exposure to DECT base EMF.

[PARAMETERS OF SPERMATOGENESIS IN MEN EXPOSED TO DIFFICULT ENVIRONMENTS].

Metal, EMF, and brain energy metabolism.

[Changes in the functional state of rat liver and kidney mitochondria under the effect of electromagnetic fields].

[Effect of low-intensity 900 MHz frequency electromagnetic radiation on rat liver and blood serum enzyme activities].

Ultra-wideband pulses increase nitric oxide production by RAW 264.7 macrophages incubated in nitrate.

[Some regularities of morphological changes in liver tissue exposed to electricity].

Microwave effects on immobilized peroxidase chemiluminescence.

Sympathetic Resonance Technology: scientific foundation and summary of biologic and clinical studies.

Examination of electric field effects on tissues by using back propagation neural network.

A novel method to estimate changes in stress-induced salivary alpha-amylase using heart rate variability and respiratory rate, as measured in a non-contact manner using a single radar attached to the back of a chair.

Effects of new Phoneutria spider toxins on glutamate release and [Ca2+]i in rat cortical synaptosomes.

--Leaf Cluster 37 (140)

Theme - Effect of radiofrequency exposure, especially prenatal exposure, on rats

Titles

Maternal exposure to a continuous 900-MHz electromagnetic field provokes neuronal loss and pathological changes in cerebellum of 32-day-old female rat offspring.

The effects of prenatal exposure to a 900-MHz electromagnetic field on the 21-day-old male rat heart.

The effects of exposure to electromagnetic field on rat myocardium.

900 MHz electromagnetic field exposure affects qualitative and quantitative features of hippocampal pyramidal cells in the adult female rat.

The effect of prenatal exposure to 1800 MHz electromagnetic field on calcineurin and bone development in rats.

Exposure to a 900 MHz electromagnetic field for 1 hour a day over 30 days does change the histopathology and biochemistry of the rat testis.

Effects of a unique electromagnetic field system on the fertility of rats.

Protective effects of luteolin on rat testis following exposure to 900 MHz electromagnetic field.

Evaluation of testicular degeneration induced by low-frequency electromagnetic fields.

Maternal mobile phone exposure alters intrinsic electrophysiological properties of CA1 pyramidal neurons in rat offspring.

Electromagnetic fields promote severe and unique vascular calcification in an animal model of ectopic calcification.

Effects of exposure to electromagnetic field (1.8/0.9 GHz) on testicular function and structure in growing rats.

The effects of prenatal long-duration exposure to 900-MHz electromagnetic field on the 21-day-old newborn male rat liver.

Common behaviors alterations after extremely low-frequency electromagnetic field exposure in rat animal model.

Pathological effects of prenatal exposure to a 900 MHz electromagnetic field on the 21-day-old male rat kidney.

Deleterious impacts of a 900-MHz electromagnetic field on hippocampal pyramidal neurons of 8-week-old Sprague Dawley male rats.

Pernicious effects of long-term, continuous 900-MHz electromagnetic field throughout adolescence on hippocampus morphology, biochemistry and pyramidal neuron numbers in 60-day-old Sprague Dawley male rats.

The effect of exposure of rats during prenatal period to radiation spreading from mobile phones on renal development.

Effects of extremely low frequency electromagnetic fields (100muT) on behaviors in rats.

Biological and morphological effects on the reproductive organ of rats after exposure to electromagnetic field.

An evaluation of the effects of long-term cell phone use on the testes via light and electron microscope analysis.

Effects of 900-MHz electromagnetic fields exposure throughout middle/late adolescence on the kidney morphology and biochemistry of the female rat.

Effects of electromagnetic field (1.8/0.9 GHz) exposure on growth plate in growing rats.

Effects of low-intensity electromagnetic fields on behavioral activity of rats.

Anxiety-like behavioural effects of extremely low-frequency electromagnetic field in rats.

Biochemical and pathological changes in the male rat kidney and bladder following exposure to continuous 900-MHz electromagnetic field on postnatal days 22-59.

The effects of an electromagnetic field on the boundary tissue of the seminiferous tubules of the rat: A light and transmission electron microscope study.

The effect of prenatal exposure to 900-MHz electromagnetic field on the 21-old-day rat testicle.

Testicular development evaluation in rats exposed to 60 Hz and 1 mT electromagnetic field.

Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.

Lasting hepatotoxic effects of prenatal mobile phone exposure.

Nonthermal effects of lifelong high-frequency electromagnetic field exposure on social memory performance in rats.

Effects of prenatal 900 MHz electromagnetic field exposures on the histology of rat kidney.

Neuroprotective effects of melatonin and omega-3 on hippocampal cells prenatally exposed to 900 MHz electromagnetic fields.

Microwave exposure affecting reproductive system in male rats.

Morphological and antioxidant impairments in the spinal cord of male offspring rats following exposure to a continuous 900MHz electromagnetic field during early and mid-adolescence.

Whole-body microwave exposure emitted by cellular phones and testicular function of rats.

Purkinje cell number decreases in the adult female rat cerebellum following exposure to 900 MHz electromagnetic field.

Effect of electromagnetic waves from mobile phone on immune status of male rats: possible protective role of vitamin D.

Changes in antioxidant capacity of blood due to mutual action of electromagnetic field (1800 MHz) and opioid drug (tramadol) in animal model of persistent inflammatory state.

Evaluation of hormonal change, biochemical parameters, and histopathological status of uterus in rats exposed to 50-Hz electromagnetic field.

Stress-related endocrinological and psychopathological effects of short- and long-term 50Hz electromagnetic field exposure in rats.

Impact of microwave at X-band in the aetiology of male infertility.

The effect on rat thymocytes of the simultaneous in vivo exposure to 50-Hz electric and magnetic field and to continuous light.

Effects on rat testis of 1.95-GHz W-CDMA for IMT-2000 cellular phones.

Disruption of the ovarian follicle reservoir of prepubertal rats following prenatal exposure to a continuous 900-MHz electromagnetic field.

Influence of electromagnetic field (1800 MHz) on lipid peroxidation in brain, blood, liver and kidney in rats.

[Effect of Guilingji Capsule on the fertility, liver functions, and serum LDH of male SD rats exposed by 900 mhz cell phone].

Zinc supplementation ameliorates electromagnetic field-induced lipid peroxidation in the rat brain.

A histopathological and biochemical evaluation of oxidative injury in the sciatic nerves of male rats exposed to a continuous 900-megahertz electromagnetic field throughout all periods of adolescence.

Effects of short-term exposure to powerline-frequency electromagnetic field on the electrical activity of the heart.

Altered operant behavior of adult rats after perinatal exposure to a 60-Hz electromagnetic field.

[Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 5. Impact of the blood serum from rats exposed to low-level electromagnetic fields on pregnancy, foetus and offspring development of intact female rats].

The effects of long-term exposure to a 2450 MHz electromagnetic field on growth and pubertal development in female Wistar rats.

Effects of mobile phone radiation on serum testosterone in Wistar albino rats.

Effects of exposure to 2100MHz GSM-like radiofrequency electromagnetic field on auditory system of rats.

Maternal mobile phone exposure adversely affects the electrophysiological properties of Purkinje neurons in rat offspring.

[Female genital toxicities of high-frequency electromagnetic field on rats].

Effects of exposure to electromagnetic field from mobile phone on serum hepcidin and iron status in male albino rats.

[State of the reproductive systemin male rats of 1st generation obtained from irradiated parents and exposed to electromagnetic radiation (897 MHz) during embryogenesis and postnatal development].

The effects of microwave emitted by cellular phones on ovarian follicles in rats.

Effects of prenatal exposure to a 900 MHz electromagnetic field on the dentate gyrus of rats: a stereological and histopathological study.

Effect of Modified Wuzi Yanzong Pill () on Tip60-Mediated Apoptosis in Testis of Male Rats after Microwave Radiation.

Influence of electromagnetic fields on reproductive system of male rats.

Effect of 910-MHz electromagnetic field on rat bone marrow.

Effects of electromagnetic radiation exposure on stress-related behaviors and stress hormones in male wistar rats.

Chronic prenatal exposure to the 900 megahertz electromagnetic field induces pyramidal cell loss in the hippocampus of newborn rats.

Effect of short duration electromagnetic field exposures on rat mass.

Hypospermatogenesis and spermatozoa maturation arrest in rats induced by mobile phone radiation.

[The delayed effects of modulated and non-modulated electromagnetic field on epileptiformic activity in rats].

Effect of extremely low frequency electromagnetic field on brain histopathology of Caspian Sea Cyprinus carpio.

Effects of chronic exposure to electromagnetic waves on the auditory system.

Influence of a 60 Hz, 3 microT, electromagnetic field on the somatic maturation of wistar rat offspring fed a regional basic diet during pregnancy.

The effect of extremely low-frequency electromagnetic fields on skin and thyroid amine- and peptide-containing cells in rats: an immunohistochemical and morphometrical study.

Effects of the electromagnetic field, 60 Hz, 3 microT, on the hormonal and metabolic regulation of undernourished pregnant rats.

[Early and Delayed Effects of Radio Frequency Electromagnetic Fields on the Reproductive Function and Functional Status of the Offspring of Experimental Animals].

The effects of electromagnetic waves emitted by the cell phones on the testicular tissue.

2.1 GHz electromagnetic field does not change contractility and intracellular Ca2+ transients but decreases beta-adrenergic responsiveness through nitric oxide signaling in rat ventricular myocytes.

The influence of electromagnetic radiation generated by a mobile phone on the skeletal system of rats.

Inhibitory effects of low doses of melatonin on induction of preneoplastic liver lesions in a medium-term liver bioassay in F344 rats: relation to the influence of electromagnetic near field exposure.

Effects of cellular phone emissions on sperm motility in rats.

[The physiological mechanisms of the regulation of zoosocial behavior in rats exposed to low-frequency electromagnetic fields].

The therapeutic effect of a pulsed electromagnetic field on the reproductive patterns of male Wistar rats exposed to a 2.45-GHz microwave field.

Postnatal development and behavior effects of in-utero exposure of rats to radiofrequency waves emitted from conventional WiFi devices.

Effects of prenatal exposure to WIFI signal (2.45GHz) on postnatal development and behavior in rat: Influence of maternal restraint.

Effect of Electromagnetic Waves from Mobile Phones on Spermatogenesis in the Era of 4G-LTE.

Exposure to radio-frequency electromagnetic waves alters acetylcholinesterase gene expression, exploratory and motor coordination-linked behaviour in male rats.

Post-continuous whole body exposure of rabbits to 650 MHz electromagnetic fields: effects on liver, spleen, and brain.

Effect of low-intensity extremely high frequency radiation on reproductive function in wistar rats.

[The neurotropic effects of low-intensity electromagnetic waves in rats with different typological characteristics of higher nervous activity].

[The progeny of male rats subjected to chronic exposure to a permanent magnetic field].

The influence of electric field exposure on bone growth and fracture repair in rats.

Effects of pulsed and sinusoidal electromagnetic fields on MMP-2, MMP-9, collagen type IV and E-cadherin expression levels in the rat kidney: an immunohistochemical study.

[Study on effects of bioelectric parameters of rats in electromagnetic radiation of HV transmission line].

Effect of chronic exposure to cellular telephone electromagnetic fields on hearing in rats.

[Bioeffects of chronic exposure to radiofrequency electromagnetic fields of low intensity (standardization strategy)].

[cts of prenatal exposure of 850-1900MHz mobile phone on the expression of PCNA and DCX in dentate gyrus of offspring rats].

Short-Term Exposure to Electromagnetic Fields Generated by Mobile Phone Jammers Decreases the Fasting Blood Sugar in Adult Male Rats.

Learning ability of young rats is unaffected by repeated exposure to a static electromagnetic field in early life.

Effects of exposure to electromagnetic field radiation (EMFR) generated by activated mobile phones on fasting blood glucose.

Histological characteristics of cutaneous and thyroid mast cell populations in male rats exposed to power-frequency electromagnetic fields.

Neural and behavioral teratological evaluation of rats exposed to ultra-wideband electromagnetic fields.

Effect of 50-Hz electromagnetic field on the retention of toxic radionuclides in rat tissues.

Influence of electromagnetic fields on bone mass and growth in developing rats: a morphometric, densitometric, and histomorphometric study.

[Cellphone electromagnetic radiation damages the testicular ultrastructure of male rats].

Effect of the pulsed electromagnetic field on the release of inflammatory mediators from adipose-derived stem cells (ADSCs) in rats.

Prevention of mobile phone induced skin tissue changes by melatonin in rat: an experimental study.

[The effects of extremely low frequency electromagnetic field exposure on the pH of the adult male semen and the motoricity parameters of spermatozoa in vitro].

Congenital anomalies in the offspring of rats after exposure of the testis to an electrostatic field.

Effect of a 1800 MHz electromagnetic field emitted during embryogenesis on chick development and hatchability.

[Systemic effects of the interaction of an organism and microwaves].

Prenatal exposure to non-ionizing radiation: effects of WiFi signals on pregnancy outcome, peripheral B-cell compartment and antibody production.

[Morphological structure of rat epiphysis exposed to electromagnetic radiation from communication devices].

The effects of microwave frequency electromagnetic fields on the development of Drosophila melanogaster.

Effects of low level electromagnetic field exposure at 2.45 GHz on rat cornea.

[Reaction of Reproductive System and Epididymal Spermatozoa .of Rats to Electromagnetic Radiation from Mobile Phone (1745 MHz) of Various Duration].

Excretion and tissue distribution of selenium following treatment of male F344 rats with benzylselenocyanate or sodium selenite.

[Immunomorphologic changes in the testes upon exposure to a microwave electromagnetic field].

Effects of 900 MHz electromagnetic field emitted by cellular phones on electrocardiograms of guinea pigs.

Effect of whole-body exposure to high-frequency electromagnetic field on the brain electrogeny in neurodefective and healthy mice.

Effect of Electromagnetic Wave on Bone Healing in Fixed and Unfixed Conditions.

Some immunological responses of common carp (Cyprinus carpio) fingerling to acute extremely low-frequency electromagnetic fields (50 Hz).

Effects of 60 Hz electromagnetic fields on early growth in three plant species and a replication of previous results.

Influence of 400, 900, and 1900 MHz electromagnetic fields on Lemna minor growth and peroxidase activity.

[Effect of fluctuating electromagnetic fields on the processes of growth and blastomogenesis].

Effects of broad band electromagnetic fields on HSP70 expression and ischemia-reperfusion in rat hearts.

[The effect of electromagnetic radiation in the millimeter range on the development of disorders in the liver induced by ether anesthesia (experimental research)].

Cell Phone Radiation Effect on Bone-to-Implant Osseointegration: A Preliminary Histologic Evaluation in Rabbits.

The Effects of Electromagnetic Fields Generated from 1800 MHz Cell Phones on Erythrocyte Rheological Parameters and Zinc Level in Rats.

[The biological activity of a decameter-range electromagnetic field with a frequency of 24 MHz].

[Effect of discontinuous short-wave electromagnetic field irradiation on the state of the endocrine glands].

Effects of microwaves (950 MHZ mobile phone) on morphometric and apoptotic changes of rabbit epididymis.

Effect of high frequency electromagnetic wave stimulation on muscle injury in a rat model.

[Respondence to electricity in the muscles of rat's jaw].

Hematological and toxicogenomic effects of ferromagnetic screening of natural electromagnetic fields.

The effects of 910-MHz electromagnetic field on rat cranial arachnoid and dura mater collagen. The axial periodicity of collagen fibrils.

Effect of electromagnetic radiation modulated by biostructures on the course of alloxan-induced diabetes mellitus in rats.

[The interaction of changes in the genitalia in the pathogenesis of sterility in men].

[Evaluation of magnesium, zinc, copper and calcium levels in workers exposed to organic solvents, hydrogen cyanide and harmful physical factors].

Induction of macrophage migration inhibitory factor precedes the onset of acute tonsillitis.

--Leaf Cluster 38 (133)

Theme - Effect of radiofrequency radiation on rat brain

Titles

Effects of early-onset radiofrequency electromagnetic field exposure (GSM 900 MHz) on behavior and memory in rats.

Mobile phone radiation and the developing brain: behavioral and morphological effects in juvenile rats.

GFAP expression in the rat brain following sub-chronic exposure to a 900 MHz electromagnetic field signal.

Blood-brain barrier permeability and nerve cell damage in rat brain 14 and 28 days after exposure to microwaves from GSM mobile phones.

Effect of a chronic GSM 900 MHz exposure on glia in the rat brain.

Effect of mobile telephony on blood-brain barrier permeability in the fetal mouse brain.

Biochemical and histological studies on adverse effects of mobile phone radiation on rat's brain.

Blood-brain barrier and electromagnetic fields: effects of scopolamine methylbromide on working memory after whole-body exposure to 2.45 GHz microwaves in rats.

Effects of GSM modulated radio-frequency electromagnetic radiation on permeability of blood-brain barrier in male & female rats.

Effect of 900 MHz radio frequency radiation on beta amyloid protein, protein carbonyl, and malondialdehyde in the brain.

Histopathological examinations of rat brains after long-term exposure to GSM-900 mobile phone radiation.

Effects of GSM and UMTS mobile telephony signals on neuron degeneration and blood-brain barrier permeation in the rat brain.

Cognitive impairment in rats after long-term exposure to GSM-900 mobile phone radiation.

Genotoxic potential of 1.6 GHz wireless communication signal: in vivo two-year bioassay.

One-year, simultaneous combined exposure of CDMA and WCDMA radiofrequency electromagnetic fields to rats.

A confirmation study of Russian and Ukrainian data on effects of 2450 MHz microwave exposure on immunological processes and teratology in rats.

Long term and excessive use of 900 MHz radiofrequency radiation alter microRNA expression in brain.

Radio frequency radiation effects on protein kinase C activity in rats' brain.

Confirmation studies of Soviet research on immunological effects of microwaves: Russian immunology results.

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Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones.

Biological and morphological effects on the brain after exposure of rats to a 1439 MHz TDMA field.

Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8GHz GSM base station environmental emission.

8-Oxo-7, 8-dihydro-2'-deoxyguanosine as a biomarker of DNA damage by mobile phone radiation.

Glial markers and emotional memory in rats following acute cerebral radiofrequency exposures.

Histological and cytological examination of rat reproductive tissue after short-time intermittent radiofrequency exposure.

Acute exposure to GSM 900-MHz electromagnetic fields induces glial reactivity and biochemical modifications in the rat brain.

[Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 3. The effect of the long-term non-thermal RF EMF exposure on complement-fixation antibodies against homologenous tissue].

Effects of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on microRNA expression in brain tissue.

Blood-brain barrier disruption by continuous-wave radio frequency radiation.

Effect of long-term (2 years) exposure of mouse brains to global system for mobile communication (GSM) radiofrequency fields on astrocytic immunoreactivity.

Effect of long-term mobile communication microwave exposure on vascular permeability in mouse brain.

The effects of pulsed 860 MHz radiofrequency radiation on the promotion of neurogenic tumors in rats.

Micronucleus frequency in erythrocytes of mice after long-term exposure to radiofrequency radiation.

Fifty-gigahertz microwave exposure effect of radiations on rat brain.

The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats.

Effect of an acute 900MHz GSM exposure on glia in the rat brain: a time-dependent study.

Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce DNA damage in various tissues of rats?

The effect of radiofrequency radiation generated by a Global System for Mobile Communications source on cochlear development in a rat model.

Mutagenic response of 2.45 GHz radiation exposure on rat brain.

Effect of GSM-900 and -1800 signals on the skin of hairless rats. I: 2-hour acute exposures.

Long-term study of 435 MHz radio-frequency radiation on blood-borne end points in cannulated rats. Part II: methods, results, and summary.

Exposure to GSM 900 MHz electromagnetic fields affects cerebral cytochrome c oxidase activity.

GSM and DCS wireless communication signals: combined chronic toxicity/carcinogenicity study in the Wistar rat.

Evidence for mobile phone radiation exposure effects on reproductive pattern of male rats: role of ROS.

Microglial activation as a measure of stress in mouse brains exposed acutely (60 minutes) and long-term (2 years) to mobile telephone radiofrequency fields.

DNA damage in rat brain cells after in vivo exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia.

Commentary on the utility of the National Toxicology Program study on cell phone radiofrequency radiation data for assessing human health risks despite unfounded criticisms aimed at minimizing the findings of adverse health effects.

Effect of in utero wi-fi exposure on the pre- and postnatal development of rats.

Cerebral radiofrequency exposures during adolescence: Impact on astrocytes and brain functions in healthy and pathologic rat models.

The effect of 2100 MHz radiofrequency radiation of a 3G mobile phone on the parotid gland of rats.

Expression of the water channel protein, aquaporin-4, in mouse brains exposed to mobile telephone radiofrequency fields.

Effects of mobile phone radiation (900 MHz radiofrequency) on structure and functions of rat brain.

Effects of 900 MHz radiofrequency on corticosterone, emotional memory and neuroinflammation in middle-aged rats.

Circadian rhythmicity of antioxidant markers in rats exposed to 1.8 GHz radiofrequency fields.

[Effects of electromagnetic pulse on blood-brain barrier permeability and tight junction proteins in rats].

Effects of head-only exposure of rats to GSM-900 on blood-brain barrier permeability and neuronal degeneration.

Effects of 900 MHz radiofrequency radiation on skin hydroxyproline contents.

Effects of gestational exposure to 1.95-GHz W-CDMA signals for IMT-2000 cellular phones: Lack of embryotoxicity and teratogenicity in rats.

In utero and early-life exposure of rats to a Wi-Fi signal: screening of immune markers in sera and gestational outcome.

The effect of radiofrequency radiation on DNA and lipid damage in female and male infant rabbits.

Effect of global system for mobile communication (gsm)-like radiofrequency fields on vascular permeability in mouse brain.

Does head-only exposure to GSM-900 electromagnetic fields affect the performance of rats in spatial learning tasks?

The effects of mobile phones on apoptosis in cerebral tissue: an experimental study on rats.

Survival and cancer in laboratory mammals exposed to radiofrequency energy.

The effects of simultaneous combined exposure to CDMA and WCDMA electromagnetic fields on rat testicular function.

Teratogenic effects of 27.12 MHz radiofrequency radiation in rats.

The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumors and other neoplasms in rats.

Effect of long-term exposure of 2.4 GHz radiofrequency radiation emitted from Wi-Fi equipment on testes functions.

Effects of continuous low-level exposure to radiofrequency radiation on intrauterine development in rats.

Electromagnetic fields and the blood-brain barrier.

Effects of 20-MHz radiofrequency radiation on rat hematology, splenic function, and serum chemistry.

Effects of GSM-like radiofrequency irradiation during the oogenesis and spermiogenesis of Xenopus laevis.

Effects of mobile phone electromagnetic fields at nonthermal SAR values on melatonin and body weight of Djungarian hamsters (Phodopus sungorus).

Expression of the immediate early gene, c-fos, in fetal brain after whole of gestation exposure of pregnant mice to global system for mobile communication microwaves.

Electromagnetic field effect or simply stress? Effects of UMTS exposure on hippocampal longterm plasticity in the context of procedure related hormone release.

Effect of GSM-900 and -1800 signals on the skin of hairless rats. II: 12-week chronic exposures.

Exposure to cell phone radiofrequency changes corticotrophin hormone levels and histology of the brain and adrenal glands in male Wistar rat.

Neurodegenerative changes and apoptosis induced by intrauterine and extrauterine exposure of radiofrequency radiation.

Rat fertility and embryo fetal development: influence of exposure to the Wi-Fi signal.

Effects of prenatal and postnatal exposure to GSM-like radiofrequency on blood chemistry and oxidative stress in infant rabbits, an experimental study.

Heat shock protein induction in fetal mouse brain as a measure of stress after whole of gestation exposure to mobile telephony radiofrequency fields.

Micronucleus induction after whole-body microwave irradiation of rats.

Effects of GSM-Frequency Electromagnetic Radiation on Some Physiological and Biochemical Parameters in Rats.

Effects of simultaneous combined exposure to CDMA and WCDMA electromagnetic fields on serum hormone levels in rats.

The differential effects of 200, 591, and 2,450 MHz radiation on rat brain energy metabolism.

RAPD Profiling, DNA Fragmentation, and Histomorphometric Examination in Brains of Wistar Rats Exposed to Indoor 2.5 Ghz Wi-Fi Devices Radiation.

[Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 4. Manifestation of oxidative intracellular stress-reaction after long-term non-thermal EMF exposure of rats].

Exposure to an 890-MHz mobile phone-like signal and serum levels of S100B and transthyretin in volunteers.

Effects of electromagnetic radiation on spatial memory and synapses in rat hippocampal CA1.

Developmental toxicity interactions of salicylic acid and radiofrequency radiation or 2-methoxyethanol in rats.

The effect of radiofrequency radiation on DNA and lipid damage in non-pregnant and pregnant rabbits and their newborns.

Effect of Short-term 900 MHz low level electromagnetic radiation exposure on blood serotonin and glutamate levels.

Effect of electromagnetic pulse exposure on brain micro vascular permeability in rats.

Alteration of adaptive behaviors of progeny after maternal mobile phone exposure.

Survivability and long-term stress reactivity levels following repeated exposure to nuclear magnetic resonance imaging procedures in rats.

[Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 2. General scheme and conditions of the experiment. Development of RF exposure conditions complying with experimental tasks. Animal's status during the long-term exposure].

Multigenerational effects of whole body exposure to 2.14 GHz W-CDMA cellular phone signals on brain function in rats.

Detrimental effect of electromagnetic pulse exposure on permeability of in vitro blood-brain-barrier model.

Effects of exposure to electromagnetic field from 915 MHz radiofrequency identification system on circulating blood cells in the healthy adult rat.

[Effects of 2000 muW/cm2; electromagnetic radiation on expression of immunoreactive protein and mRNA of NMDA receptor 2A subunit in rats hippocampus].

Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats.

Effects of exposure of the ear to GSM microwaves: in vivo and in vitro experimental studies.

Age-Related Modulations of AQP4 and Caveolin-1 in the Hippocampus Predispose the Toxic Effect of Phoneutria nigriventer Spider Venom.

GSM radiation triggers seizures and increases cerebral c-Fos positivity in rats pretreated with subconvulsive doses of picrotoxin.

[Studies on the injury effects of hippocampus induced by high power microwave radiation in rat].

GSM-like radiofrequency exposure induces apoptosis via caspase-dependent pathway in infant rabbits.

Exposure to GSM 900-MHz mobile radiation impaired inhibitory avoidance memory consolidation in rat: Involvements of opioidergic and nitrergic systems.

Electromagnetic pulse exposure induces overexpression of beta amyloid protein in rats.

Effects of intrauterine and extrauterine exposure to GSM-like radiofrequency on distortion product otoacoustic emissions in infant male rabbits.

Effects of whole-body exposure to 915 MHz RFID on secretory functions of the thyroid system in rats.

[The assessment of modulated radiofrequence electromagnetic radiation on cognitive function in rats of different ages].

Life-Time Dosimetric Assessment for Mice and Rats Exposed in Reverberation Chambers of the 2-Year NTP Cancer Bioassay Study on Cell Phone Radiation.

Exposure setup to study potential adverse effects at GSM 1800 and UMTS frequencies on the auditory systems of rats.

The effects of 2100-MHz radiofrequency radiation on nasal mucosa and mucociliary clearance in rats.

[A comparative histochemical study of cytochrome oxidase activity in the somatosensory and auditory brain centers in the normal rat and after exposure to superhigh-frequency electromagnetic fields].

Estimates of absorption of radiofrequency radiation by the embryo and fetus during pregnancy.

MRI gradient fields increase brain mannitol space.

The identification of an intensity 'window' on the bioeffects of mobile telephony radiation.

Effects of 7 Hz-modulated 450 MHz electromagnetic radiation on human performance in visual memory tasks.

Dataset on significant role of Candesartan on cognitive functions in rats having memory impairment induced by electromagnetic waves.

Effects of electromagnetic radiation on morphology and TGF-beta3 expression in mouse testicular tissue.

Effects of radiofrequency exposure on the GABAergic system in the rat cerebellum: clues from semi-quantitative immunohistochemistry.

Metabolomic study of urinary polyamines in rat exposed to 915 MHz radiofrequency identification signal.

An international project to confirm Soviet-era results on immunological and teratological effects of RF field exposure in Wistar rats and comments on Grigoriev et al. [2010].

Radiotelemetry and wildlife: Highlighting a gap in the knowledge on radiofrequency radiation effects.

Non-thermal continuous and modulated electromagnetic radiation fields effects on sleep EEG of rats.

The Radiofrequency Radiation Dosimetry Handbook: reminiscences.

Effects of acute exposure to ultrahigh radiofrequency radiation on three antenna engineers.

Bioeffects of mobile telephony radiation in relation to its intensity or distance from the antenna.

Mediastinal fibrosis and radiofrequency radiation exposure: is there an association?

Effects of GSM-like radiofrequency on distortion product otoacoustic emissions in pregnant adult rabbits.

[The biological action of physical factors in the critical periods of embryogenesis].

Fourth Level Cluster 84 (692)
Theme - Genotoxic effects of radiofrequency radiation

--Leaf Cluster 20 (126)

Theme - DNA damage after microwave radiation

Titles

Evaluation of basal DNA damage and oxidative stress in Wistar rat leukocytes after exposure to microwave radiation.

The effect of electromagnetic field exposure on the formation of DNA single strand breaks in human cells.

Measurement of DNA damage after exposure to 2450 MHz electromagnetic radiation.

Human fibroblasts and 900 MHz radiofrequency radiation: evaluation of DNA damage after exposure and co-exposure to 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5h)-furanone (MX).

Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells.

Influence of 1.8-GHz (GSM) radiofrequency radiation (RFR) on DNA damage and repair induced by X-rays in human leukocytes in vitro.

DNA Damage of Lymphocytes in Volunteers after 4 hours Use of Mobile Phone.

Studying the synergistic damage effects induced by 1.8 GHz radiofrequency field radiation (RFR) with four chemical mutagens on human lymphocyte DNA using comet assay in vitro.

Effect of superposed electromagnetic noise on DNA damage of lens epithelial cells induced by microwave radiation.

[DNA damage and repair induced by acute exposure of microwave from mobile phone on cultured human lens epithelial cells].

Intermittent extremely low frequency electromagnetic fields cause DNA damage in a dose-dependent way.

50-Hertz electromagnetic fields induce gammaH2AX foci formation in mouse preimplantation embryos in vitro.

Measurement of DNA damage after acute exposure to pulsed-wave 2450 MHz microwaves in rat brain cells by two alkaline comet assay methods.

Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation.

Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line.

Measurements of alkali-labile DNA damage and protein-DNA crosslinks after 2450 MHz microwave and low-dose gamma irradiation in vitro.

Electromagnetic fields and the induction of DNA strand breaks.

Age-related effects on induction of DNA strand breaks by intermittent exposure to electromagnetic fields.

[Influence of 1.8 GHz microwave on DNA damage induced by ultraviolet C ray].

Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro.

DNA and chromosomal damage in response to intermittent extremely low-frequency magnetic fields.

The toxic effects of mobile phone radiofrequency (940 MHz) on the structure of calf thymus DNA.

Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells.

Effects of in vitro exposure to power frequency magnetic fields on UV-induced DNA damage of rat lymphocytes.

Evaluating the combinative effects on human lymphocyte DNA damage induced by ultraviolet ray C plus 1.8 GHz microwaves using comet assay in vitro.

[Influence of 1.8 GHz microwave on DNA damage induced by 4 chemical mutagens].

Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts.

60 Hz magnetic field exposure induces DNA crosslinks in rat brain cells.

Combined effects of 872 MHz radiofrequency radiation and ferrous chloride on reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells.

8-oxoG DNA glycosylase-1 inhibition sensitizes Neuro-2a cells to oxidative DNA base damage induced by 900 MHz radiofrequency electromagnetic radiation.

Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure.

Radioprotective effects of honeybee venom (Apis mellifera) against 915-MHz microwave radiation-induced DNA damage in wistar rat lymphocytes: in vitro study.

Assessment of DNA sensitivity in peripheral blood leukocytes after occupational exposure to microwave radiation: the alkaline comet assay and chromatid breakage assay.

Assessment of genetic damage in peripheral blood of human volunteers exposed (whole-body) to a 200 muT, 60 Hz magnetic field.

Acute exposure to a 60 Hz magnetic field increases DNA strand breaks in rat brain cells.

Influence of a static magnetic field (250 mT) on the antioxidant response and DNA integrity in THP1 cells.

Investigation of co-genotoxic effects of radiofrequency electromagnetic fields in vivo.

Evaluation of genotoxic effects in human leukocytes after in vitro exposure to 1950 MHz UMTS radiofrequency field.

Adaptive response in mouse bone-marrow stromal cells exposed to 900-MHz radiofrequency fields: Gamma-radiation-induced DNA strand breaks and repair.

Short-term exposure to 50 Hz ELF-EMF alters the cisplatin-induced oxidative response in AT478 murine squamous cell carcinoma cells.

Measurement of DNA damage and apoptosis in Molt-4 cells after in vitro exposure to radiofrequency radiation.

Electromagnetic fields and health: DNA-based dosimetry.

Combinative exposure effect of radio frequency signals from CDMA mobile phones and aphidicolin on DNA integrity.

Magnetic-field-induced DNA strand breaks in brain cells of the rat.

Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells.

DNA repair after gamma irradiation in lymphocytes exposed to low-frequency pulsed electromagnetic fields.

Epinephrine, DNA integrity and oxidative stress in workers exposed to extremely low-frequency electromagnetic fields (ELF-EMFs) at 132 kV substations.

Measurement of DNA damage after exposure to electromagnetic radiation in the cellular phone communication frequency band (835.62 and 847.74 MHz).

Effect of Radiofrequency Radiation on Human Hematopoietic Stem Cells.

Mobile phone signal exposure triggers a hormesis-like effect in Atm(+/+) and Atm(-/-) mouse embryonic fibroblasts.

Evaluation of genotoxic effects in human fibroblasts after intermittent exposure to 50 Hz electromagnetic fields: a confirmatory study.

Single strand DNA breaks in rat brain cells exposed to microwave radiation.

DNA damage, cell kinetics and ODC activities studied in CBA mice exposed to electromagnetic fields generated by transmission lines.

Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells.

The effect of electromagnetic field exposure on the formation of DNA lesions.

Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation.

Sensitivity of spiral ganglion neurons to damage caused by mobile phone electromagnetic radiation will increase in lipopolysaccharide-induced inflammation in vitro model.

Oxidative DNA damage in rats exposed to extremely low frequency electro magnetic fields.

Ataxia telangiectasia mutated deficiency does not result in genetic susceptibility to 50 Hz magnetic fields exposure in mouse embryonic fibroblasts.

[Effects of 2,450 MHz microwave on DNA damage induced by three chemical mutagens in vitro].

In vitro assessment of clastogenicity of mobile-phone radiation (835 MHz) using the alkaline comet assay and chromosomal aberration test.

Effects of pulsed electric fields on DNA of human lymphocytes.

[Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields].

Adaptive response in mice exposed to 900 MHz radiofrequency fields: primary DNA damage.

Exposure of mammalian cells to 60-Hz magnetic or electric fields: analysis for DNA single-strand breaks.

Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline.

Effect of GSTM1 and GSTT1 Polymorphisms on Genetic Damage in Humans Populations Exposed to Radiation From Mobile Towers.

DNA damage induced in brain cells of CBA mice exposed to magnetic fields.

[Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells].

Loss of transforming activity of plasmid DNA (pBR322) in E. coli caused by singlet molecular oxygen.

Decreased DNA repair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure.

Cytotoxic and genotoxic effect in RTG-2 cell line exposed to selected biocides used in the disinfection of cooling towers.

Mobile phone specific electromagnetic fields induce transient DNA damage and nucleotide excision repair in serum-deprived human glioblastoma cells.

Adaptive response in mice exposed to 900 MHZ radiofrequency fields: bleomycin-induced DNA and oxidative damage/repair.

14.6 mT ELF magnetic field exposure yields no DNA breaks in model system Salmonella, but provides evidence of heat stress protection.

Effect of Radiofrequency Radiation Emitted from 2G and 3G Cell Phone on Developing Liver of Chick Embryo - A Comparative Study.

Cell type-specific genotoxic effects of intermittent extremely low-frequency electromagnetic fields.

Biological effects of non-ionizing electromagnetic fields: Two sides of a coin.

Radiofrequency exposure and mammalian cell toxicity, genotoxicity, and transformation.

Studying the protein expression in human B lymphoblastoid cells exposed to 1.8-GHz (GSM) radiofrequency radiation (RFR) with protein microarray.

Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin.

An evaluation of genotoxicity in human neuronal-type cells subjected to oxidative stress under an extremely low frequency pulsed magnetic field.

Exposure of rat brain to 915 MHz GSM microwaves induces changes in gene expression but not double stranded DNA breaks or effects on chromatin conformation.

Radiofrequency (microwave) radiation exposure of mammalian cells during UV-induced DNA repair synthesis.

[Effect of low-intensity microwave of on mitomycin C-induced genotoxicity in vitro].

Effects of 1800 MHz RF-EMF exposure on DNA damage and cellular functions in primary cultured neurogenic cells.

Importance of DNA fragmentation in apoptosis with regard to TUNEL specificity.

Oxidative changes and apoptosis induced by 1800-MHz electromagnetic radiation in NIH/3T3 cells.

The genomic effects of cell phone exposure on the reproductive system.

Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons.

Mitochondrial DNA damage and oxidative damage in HL-60 cells exposed to 900MHz radiofrequency fields.

Microwave miniprep of total genomic DNA from fungi, plants, protists and animals for PCR.

Exposure to 915 MHz radiation induces micronuclei in Vicia faba root tips.

[Pulse-modulated Electromagnetic Radiation of Extremely High Frequencies Protects Cellular DNA against Damaging Effect of Physico-Chemical Factors in vitro].

GSM 900 MHz cellular phone radiation can either stimulate or depress early embryogenesis in Japanese quails depending on the duration of exposure.

Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes.

915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons.

Synergism between electricity and ionizing radiation.

[Mechanisms of electromagnetic radiation damaging male reproduction].

Comments on "Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes" by Schwarz et al. (Int Arch Occup Environ Health 2008: doi: 10.1007/s00420-008-0305-5).

Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in HL-60 cells are not reproducible.

[Changes in the chromatin structure of lymphoid cells under the influence of low-intensity extremely high-frequency electromagnetic radiation against the background of inflammatory process].

Effect of 7 mT static magnetic field and iron ions on rat lymphocytes: apoptosis, necrosis and free radical processes.

[Cytophotometry of myelokaryocyte DNA following a single exposure to low-intensity UHF irradiation].

Genotoxicity of radiofrequency radiation. DNA/Genetox Expert Panel.

Effect of Mobile Phone Radiation on Cardiovascular Development of Chick Embryo.

Effects of gamma rays, ultraviolet radiation, sunlight, microwaves and electromagnetic fields on gene expression mediated by human immunodeficiency virus promoter.

Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice.

Effect of exposure to 900 MHz radiofrequency radiation on intrachromosomal recombination in pKZ1 mice.

Evaluation of DNA damage in spinal cord and mutagenic effect of a Phalpha1beta recombinant toxin with analgesic properties from the Phoneutria nigriventer spider.

Microwaves from GSM mobile telephones affect 53BP1 and gamma-H2AX foci in human lymphocytes from hypersensitive and healthy persons.

Effects of co-exposure to extremely low frequency (ELF) magnetic fields and benzene or benzene metabolites determined in vitro by the alkaline comet assay.

Study of low-intensity 2450-MHz microwave exposure enhancing the genotoxic effects of mitomycin C using micronucleus test and comet assay in vitro.

Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study.

Oxidative and mutagenic effects of low intensity GSM 1800 MHz microwave radiation.

Effects of Long-Term Exposure to 60 GHz Millimeter-Wavelength Radiation on the Genotoxicity and Heat Shock Protein (Hsp) Expression of Cells Derived from Human Eye.

Investigation of potential genotoxic effects of low frequency electromagnetic fields on Escherichia coli.

[Impact of mobile phone radiation on the quality and DNA methylation of human sperm in vitro].

[Effects of low-intensity extremely high frequency electromagnetic radiation on chromatin structure of lymphoid cells in vivo and in vitro].

Characterisation of transcriptionally active and inactive chromatin domains in neurons.

Effect of microwave exposure on the ovarian development of Drosophila melanogaster.

Exposure to non-ionizing electromagnetic radiation of public risk prevention instruments threatens the quality of spermatozoids.

The biological effects of radiofrequency radiation: a critical review and recommendations.

Increase in the mitotic recombination frequency in Drosophila melanogaster by magnetic field exposure and its suppression by vitamin E supplement.

[Mechanisms of the combined effect of SHF electromagnetic radiation and hydrogen peroxide on the viability of microorganisms].

RNA-dependent DNA polymerase (reverse transcriptase) from avian myeloblastosis virus: a zinc metalloenzyme.

--Leaf Cluster 28 (100)

Theme - Chromosome damage in lymphocytes exposed to radiofrequency radiation

Titles

Chromosome damage and micronucleus formation in human blood lymphocytes exposed in vitro to radiofrequency radiation at a cellular telephone frequency (847.74 MHz, CDMA).

Cytogenetic studies in human blood lymphocytes exposed in vitro to 2.45 GHz or 8.2 GHz radiofrequency radiation.

Cytogenetic studies in human blood lymphocytes exposed in vitro to radiofrequency radiation at a cellular telephone frequency (835.62 MHz, FDMA).

Effects of modulated microwave radiation at cellular telephone frequency (1.95 GHz) on X-ray-induced chromosome aberrations in human lymphocytes in vitro.

Influence of radiofrequency radiation on chromosome aberrations in CHO cells and its interaction with DNA-damaging agents.

Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes to radiofrequency electromagnetic fields for 72 hours.

Comparison of chromosome aberrations in peripheral blood lymphocytes from people occupationally exposed to ionizing and radiofrequency radiation.

935 MHz cellular phone radiation. An in vitro study of genotoxicity in human lymphocytes.

Evaluation of genotoxic effects in human peripheral blood leukocytes following an acute in vitro exposure to 900 MHz radiofrequency fields.

Genetic damage in mammalian somatic cells exposed to radiofrequency radiation: a meta-analysis of data from 63 publications (1990-2005).

Mutagenic and morphologic impacts of 1.8GHz radiofrequency radiation on human peripheral blood lymphocytes (hPBLs) and possible protective role of pre-treatment with Ginkgo biloba (EGb 761).

Effects of 1-week and 6-week exposure to GSM/DCS radiofrequency radiation on micronucleus formation in B6C3F1 mice.

Influence of GSM signals on human peripheral lymphocytes: study of genotoxicity.

The repair of gamma-ray-induced chromosomal damage in human lymphocytes after exposure to extremely low frequency electromagnetic fields.

Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability.

Genetic damage in human cells exposed to non-ionizing radiofrequency fields: a meta-analysis of the data from 88 publications (1990-2011).

Incidence of micronuclei in human peripheral blood lymphocytes exposed to modulated and unmodulated 2450 MHz radiofrequency fields.

Effects of in vivo exposure to GSM-modulated 900 MHz radiation on mouse peripheral lymphocytes.

Clastogenic effects in human lymphocytes of power frequency electric fields: in vivo and in vitro studies.

Frequency of micronuclei in the blood and bone marrow cells of mice exposed to ultra-wideband electromagnetic radiation.

Micronucleus assay and lymphocyte mitotic activity in risk assessment of occupational exposure to microwave radiation.

Effect of nuclear magnetic resonance on chromosomes of mouse bone marrow cells.

In vitro lymphocyte proliferation induced by radio-frequency electromagnetic radiation under isothermal conditions.

Induction of adaptive response in human blood lymphocytes exposed to 900 MHz radiofrequency fields: influence of cell cycle.

Lymphocytes and low-frequency electromagnetic fields.

Chromosomal effects in lymphocytes of 400 kV-substation workers.

Effects of high-frequency electromagnetic fields on human lymphocytes in vitro.

Assessment of genotoxicity and genomic instability in rat primary astrocytes exposed to 872 MHz radiofrequency radiation and chemicals.

Increased chromatid-type chromosomal aberrations in mouse m5S cells exposed to power-line frequency magnetic fields.

Cytogenetic studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using R-banded karyotyping.

Genotoxic effects of 3 T magnetic resonance imaging in cultured human lymphocytes.

Age-dependent effects of in vitro radiofrequency exposure (mobile phone) on CD95+ T helper human lymphocytes.

Micronuclei in peripheral blood and bone marrow cells of mice exposed to 42 GHz electromagnetic millimeter waves.

Cytogenetic effects of 900 MHz (GSM) microwaves on human lymphocytes.

Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes.

Elevated sister chromatid exchange frequencies in dividing human peripheral blood lymphocytes exposed to 50 Hz magnetic fields.

Chromosomal damage in human diploid fibroblasts by intermittent exposure to extremely low-frequency electromagnetic fields.

Micronuclei in the blood and bone marrow cells of mice exposed to specific complex time-varying pulsed magnetic fields.

Enhanced cytotoxic and genotoxic effects of gadolinium following ELF-EMF irradiation in human lymphocytes.

[Effects of electromagnetic radiation on health and immune function of operators].

[Chromosome abnormalities caused by computer video display monitors' radiation].

Effect of low-level pulsed electromagnetic fields on human chromosomes in vitro: analysis of chromosomal aberrations.

Effect of high-frequency electromagnetic fields with a wide range of SARs on chromosomal aberrations in murine m5S cells.

Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C.

Effect of 900 MHz Electromagnetic Radiation on the Induction of ROS in Human Peripheral Blood Mononuclear Cells.

Analysis of chromosomal aberrations, sister chromatid exchanges and micronuclei among power linesmen with long-term exposure to 50-Hz electromagnetic fields.

A chromosomal study of workers with long-term exposure to radio-frequency radiation.

Induction of adaptive response in mice exposed to 900MHz radiofrequency fields: application of micronucleus assay.

Genetic damage in subjects exposed to radiofrequency radiation.

Investigation of the genotoxic effect of microwave irradiation in rat bone marrow cells: in vivo exposure.

[Effect of electromagnetic radiation on T-lymphocyte subpopulations and immunoglobulin level in human blood serum after occupational exposure].

Effects of low frequency electromagnetic fields on expression of lymphocyte subsets and production of cytokines of men and women employed in a museum.

Terahertz radiation increases genomic instability in human lymphocytes.

Cytogenetic damage in human lymphocytes following GMSK phase modulated microwave exposure.

Aneuploidy studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using FISH.

X-rays, microwaves and vinyl chloride monomer: their clastogenic and aneugenic activity, using the micronucleus assay on human lymphocytes.

[The effect of ultrahigh-frequency radiation on adaptation thresholds and the damages to blood system cells].

Erythropoietic changes in rats after 2.45 GJz nonthermal irradiation.

Cytogenetic observations in human peripheral blood leukocytes following in vitro exposure to THz radiation: a pilot study.

Effect of long-term 50 Hz magnetic field exposure on the micronucleated polychromatic erythrocytes of mice.

Interactive developmental toxicity of radiofrequency radiation and 2-methoxyethanol in rats.

Proflavin and microwave radiation: absence of a mutagenic interaction.

Effects of GSM-modulated 900 MHz radiofrequency electromagnetic fields on the hematopoietic potential of mouse bone marrow cells.

Follow up study on the immune response to low frequency electromagnetic fields in men and women working in a museum.

Adverse and beneficial effects in Chinese hamster lung fibroblast cells following radiofrequency exposure.

Cytogenetic effects of extremely low frequency magnetic field on Wistar rat bone marrow.

[Chromosome studies of personnel exposed to electromagnetic radiation at radar centers].

Evaluation of the cytogenotoxic damage in immature and mature rats exposed to 900 MHz radiofrequency electromagnetic fields.

Clastogenicity and aneuploidy in newborn and adult mice exposed to 50 Hz magnetic fields.

Effects of electromagnetic fields on the immune systems of occupationally exposed humans and mice.

954 MHz microwaves enhance the mutagenic properties of mitomycin C.

[Comparative effectiveness of different tests to determine the mutagenicity of certain factors in mammals. II. Frequency of anomalous sperm head in mice exposed to different factors].

Cytotoxic and genotoxic effects of high-frequency electromagnetic fields (GSM 1800 MHz) on immature and mature rats.

Interactions of radiofrequency radiation on 2-methoxyethanol teratogenicity in rats.

[The cytogenetic action of electromagnetic fields in the short-wave range].

In vitro fertilization of mouse ova by spermatozoa exposed isothermally to radio-frequency radiation.

Effect of Exposure to 900 MHz GSM Mobile Phone Radiofrequency Radiation on Estrogen Receptor Methylation Status in Colon Cells of Male Sprague Dawley Rats.

Adaptive response in mouse bone marrow stromal cells exposed to 900MHz radiofrequency fields: Impact of poly (ADP-ribose) polymerase (PARP).

Effects of electromagnetic fields produced by radiotelevision broadcasting stations on the immune system of women.

Assessment of radio-frequency electromagnetic radiation by the micronucleus test in bovine peripheral erythrocytes.

[Levels of immunoglobulin and subpopulations of T lymphocytes and NK cells in men occupationally exposed to microwave radiation in frequencies of 6-12 GHz].

Association of low job control with a decrease in memory (CD4+ CD45RO+) T lymphocytes in Japanese middle-aged male workers in an electric power plant.

Effects of extremely low-frequency electromagnetic fields on delayed chromosomal instability induced by bleomycin in normal human fibroblast cells.

[Proliferation of bone marrow cells upon exposure to constant magnetic fields of ultra-high strength].

The immune response of women with prolonged exposure to electromagnetic fields produced by radiotelevision broadcasting stations.

Acute exposure to 930 MHz CW electromagnetic radiation in vitro affects reactive oxygen species level in rat lymphocytes treated by iron ions.

The process of myelopoiesis in guinea pigs under conditions of a static magnetic field.

[Effect of electromagnetic radiation of millimetric wave band on genome of somatic cells].

Suppression of T-lymphocyte cytotoxicity following exposure to 60-Hz sinusoidal electric fields.

Reactive oxygen species formation and apoptosis in human peripheral blood mononuclear cell induced by 900 MHz mobile phone radiation.

Occupational exposure to high frequency electromagnetic fields and its effect on human immune parameters.

Does radio frequency radiation induce micronuclei frequency in exfoliated bladder cells of diabetic rats?

Radiofrequency radiation and the immune system. Part 3. In vitro effects on human immunoglobin and on murine T- and B-lymphocytes.

Leukocyte trafficking in response to magnetic resonance imaging.

Combined effects of traffic and electromagnetic fields on the immune system of fertile atopic women.

Developmental toxicity interactions of methanol and radiofrequency radiation or 2-methoxyethanol in rats.

Neoplastic transformation in C3H 10T(1/2) cells after exposure to 835.62 MHz FDMA and 847.74 MHz CDMA radiations.

Adaptive response in animals exposed to non-ionizing radiofrequency fields: some underlying mechanisms.

Probing lymphoma infiltration in spleen of AKR/J mice chronically exposed to electromagnetic fields for risk assessment--toward noninvasive modeling.

Combined exposure of ELF magnetic fields and x-rays increased mutant yields compared with x-rays alone in pTN89 plasmids.

--Leaf Cluster 45 (179)

Theme - Adverse effects of low-frequency EMF on cells

Titles

Extremely low frequency variable electromagnetic fields affect cancer and noncancerous cells in vitro differently: Preliminary study.

Effect of electromagnetic field exposure on chemically induced differentiation of friend erythroleukemia cells.

Extremely low-frequency electromagnetic fields cause G1 phase arrest through the activation of the ATM-Chk2-p21 pathway.

Extremely low frequency electromagnetic field exposure promotes differentiation of pituitary corticotrope-derived AtT20 D16V cells.

Effect of extremely low-frequency electromagnetic fields on antioxidant activity in the human keratinocyte cell line NCTC 2544.

Electromagnetic fields with frequencies of 5, 60 and 120 Hz affect the cell cycle and viability of human fibroblast BJ in vitro.

Bidirectional frequency-dependent effect of extremely low-frequency electromagnetic field on E. coli K-12.

Melatonin protects rat cerebellar granule cells against electromagnetic field-induced increases in Na(+) currents through intracellular Ca(2+) release.

Neuroprotective effects of lotus seedpod procyanidins on extremely low frequency electromagnetic field-induced neurotoxicity in primary cultured hippocampal neurons.

Pulsed Electromagnetic Field Stimulation Promotes Anti-cell Proliferative Activity in Doxorubicin-treated Mouse Osteosarcoma Cells.

Effects of low frequency electromagnetic field on proliferation of human epidermal stem cells: An in vitro study.

Extremely low-frequency electromagnetic field exposure enhances inflammatory response and inhibits effect of antioxidant in RAW 264.7 cells.

[Effects of extremely low frequency pulsed electromagnetic field on different-derived osteoblast-like cells].

Impact of extremely low frequency electromagnetic fields on CD4 expression in peripheral blood mononuclear cells.

[Effect of long-term power frequency electromagnetic field exposure on proliferation and apoptosis of SRA01/04 cells].

[Effect of pulsed electromagnetic field with different frequencies on the proliferation, apoptosis and migration of human ovarian cancer cells].

Extremely low frequency electromagnetic fields affect proliferation and mitochondrial activity of human cancer cell lines.

Effects of extremely low-frequency pulsed electromagnetic fields on morphological and biochemical properties of human breast carcinoma cells (T47D).

Correlation between pulsed electromagnetic fields exposure time and cell proliferation increase in human osteosarcoma cell lines and human normal osteoblast cells in vitro.

Influence of extremely low frequency electromagnetic fields on the swimming behavior of ciliates.

Suppression of a differentiation response in MC-3T3-E1 osteoblast-like cells by sustained, low-level, 30 Hz magnetic-field exposure.

Exposure of rats to extremely low-frequency electromagnetic fields (ELF-EMF) alters cytokines production.

Exposure to extremely low frequency electromagnetic fields alters the calcium dynamics of cultured entorhinal cortex neurons.

Effects of electromagnetic fields on molecules and cells.

Effect of intermittent and continuous exposure to electromagnetic fields on cultured hippocampal cells.

The impact of electromagnetic field at a frequency of 50 Hz and a magnetic induction of 2.5 mT on viability of pineal cells in vitro.

Induction of apoptotic cell death in human leukemic cell line, HL-60, by extremely low frequency electric magnetic fields: analysis of the possible mechanisms in vitro.

Acute effects of low-frequency electromagnetic fields on leukocyte-endothelial interactions in vivo.

Effects of 50 Hz pulsed electromagnetic fields on the growth and cell cycle arrest of mesenchymal stem cells: an in vitro study.

Haemopoietic cell proliferation in murine bone marrow cells exposed to extreme low frequency (ELF) electromagnetic fields.

Extremely low frequency electromagnetic field exposure affects fertilization outcome in swine animal model.

Effects of extremely low frequency electromagnetic fields on human fetal scleral fibroblasts.

Action of a 50 Hz magnetic field on proliferation of cells in culture.

In vitro evaluation of teratogenic effects by time-varying MR gradient fields on fetal human fibroblasts.

Chronic electromagnetic field exposure decreases HSP70 levels and lowers cytoprotection.

Effect of exposure to an extremely low frequency-electromagnetic field on the cellular collagen with respect to signaling pathways in osteoblast-like cells.

Effect of puerarin on matrix metalloproteinase-2 in human fetal scleral fibroblasts treated with low frequency electromagnetic fields.

Effects of extremely low frequency electromagnetic fields on intracellular calcium transients in cardiomyocytes.

[Biological effects of non-ionizing electromagnetic radiation].

Effects of 60 Hz extremely low frequency magnetic fields (EMF) on radiation- and chemical-induced mutagenesis in mammalian cells.

Exposure to ELF-pulse modulated X band microwaves increases in vitro human astrocytoma cell proliferation.

Bioelectromagnetic field effects on cancer cells and mice tumors.

A 700 MHz 1H-NMR study reveals apoptosis-like behavior in human K562 erythroleukemic cells exposed to a 50 Hz sinusoidal magnetic field.

Low intensity and frequency pulsed electromagnetic fields selectively impair breast cancer cell viability.

Cellular effects of electromagnetic fields.

50 Hz extremely low frequency electromagnetic fields enhance protein carbonyl groups content in cancer cells: effects on proteasomal systems.

Pulsed electromagnetic fields accelerate apoptotic rate in osteoclasts.

A short-term extremely low frequency electromagnetic field exposure increases circulating leukocyte numbers and affects HPA-axis signaling in mice.

Delineation of electric and magnetic field effects of extremely low frequency electromagnetic radiation on transcription.

Pulsed or continuous electromagnetic field induce p53/p21-mediated apoptotic signaling pathway in mouse spermatogenic cells in vitro and thus may affect male fertility.

Power-frequency electromagnetic fields and the capacitative calcium entry system in SV40-transformed Swiss 3T3 cells.

Transferrin receptors and natural killer cell lysis. A study using Colo 205 cells exposed to 60 Hz electromagnetic fields.

Electromagnetic fields and cells.

Calcium protects differentiating neuroblastoma cells during 50 Hz electromagnetic radiation.

A review of in vitro studies: low-frequency electromagnetic fields.

Electric and/or magnetic field effects on DNA structure and function in cultured human cells.

Effects of long-term 50Hz power-line frequency electromagnetic field on cell behavior in Balb/c 3T3 cells.

Low-intensity electromagnetic fields induce human cryptochrome to modulate intracellular reactive oxygen species.

Effect of extremely low frequency (ELF) magnetic field exposure on morphological and biophysical properties of human lymphoid cell line (Raji).

Effect of 0.2 T static magnetic field on human neurons: remodeling and inhibition of signal transduction without genome instability.

Exposure to 1.8 GHz electromagnetic fields affects morphology, DNA-related Raman spectra and mitochondrial functions in human lympho-monocytes.

Effects of 60-Hz fields, estradiol and xenoestrogens on human breast cancer cells.

Influence of a 50 hz extra low frequency electromagnetic field on spermatozoa motility and fertilization rates in rabbits.

Semi-quantitative proteomics of mammalian cells upon short-term exposure to non-ionizing electromagnetic fields.

[Experimental data on extremely low frequency (ELF) electromagnetic fields].

[Effect of static magnetic field on development toxicity of rat embryonic midbrain neurons cells].

[Flow cytometric analysis of the effects of 50 Hz magnetic fields on mouse spermatogenesis].

Dose dependence of acetylcholinesterase activity in neuroblastoma cells exposed to modulated radio-frequency electromagnetic radiation.

Increased apoptosis, changes in intracellular Ca2+, and functional alterations in lymphocytes and macrophages after in vitro exposure to static magnetic field.

Biomarkers of induced electromagnetic field and cancer.

IGF-II receptor number is increased in TE-85 osteosarcoma cells by combined magnetic fields.

Nonlinear cell response to strong electric fields.

Mechanisms underlying spontaneous calcium spiking in aequorin-loaded ROS 17/2.8 cells.

The effects of low-energy 60-Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase.

Effects of extremely low frequency electromagnetic fields on turkeys.

The Bioeffects Resulting from Prokaryotic Cells and Yeast Being Exposed to an 18 GHz Electromagnetic Field.

The effect of electromagnetic field on reactive oxygen species production in human neutrophils in vitro.

[Effect of low-frequency electromagnetic fields on the individual functional systems of the body].

[Modeling of the effect of modulated electromagnetic radiation on animal cells].

2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic Ca(2)(+) influx in human leukemia cancer cells.

[Influence of electromagnetic radiation of different ranges on the transmembrane transport of Na+, K+, and Ca2+ ions in normal and tumor cells].

Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field.

Analysis of the effect of a 60 Hz AC field on histamine release by rat peritoneal mast cells.

Intramembrane protein distribution in cell cultures is affected by 50 Hz pulsed magnetic fields.

Calcium homeostasis of isolated heart muscle cells exposed to pulsed high-frequency electromagnetic fields.

Do electromagnetic fields interact directly with DNA?

Extremely low frequency 7 Hz 100 microT electromagnetic radiation promotes differentiation in the human epithelial cell line HaCaT.

Effects of 60 Hz electromagnetic field exposure on testicular germ cell apoptosis in mice.

Antiproliferative effect of millimeter radiation on human erythromyeloid leukemia cell line K562 in culture: ultrastructural- and metabolic-induced changes.

A 3 milliTesla 60 Hz magnetic field is neither mutagenic nor co-mutagenic in the presence of menadione and MNU in a transgenic rat cell line.

Exposure to low frequency pulsed electromagnetic fields increases interleukin-1 and interleukin-6 production by human peripheral blood mononuclear cells.

Enhanced proliferation caused by a low frequency weak magnetic field in chick embryo fibroblasts is suppressed by radical scavengers.

The interaction between electromagnetic fields at megahertz, gigahertz and terahertz frequencies with cells, tissues and organisms: risks and potential.

In vitro evaluation of magnetic resonance imaging at 3.0 tesla on clonogenic ability, proliferation, and cell cycle in human embryonic lung fibroblasts.

Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters.

Subchronic effects on leukocyte-endothelial interactions in mice by whole body exposure to extremely low frequency electromagnetic fields.

Acute and chronic effects of exposure to a 1-mT magnetic field on the cytoskeleton, stress proteins, and proliferation of astroglial cells in culture.

Effects of weak environmental magnetic fields on the spontaneous bioelectrical activity of snail neurons.

Long-term effects of repetitive exposure to a static magnetic field (1.5 T) on proliferation of human fetal lung fibroblasts.

Modification of electrokinetic properties of nuclei in human buccal epithelial cells by electric fields.

Bioeffects induced by exposure to microwaves are mitigated by superposition of ELF noise.

Electromagnetic fields (UHF) increase voltage sensitivity of membrane ion channels; possible indication of cell phone effect on living cells.

Synaptosomal acetylcholinesterase activity variation pattern in the presence of electromagnetic fields.

Chicken embryo fibroblasts exposed to weak, time-varying magnetic fields share cell proliferation, adenosine deaminase activity, and membrane characteristics of transformed cells.

Low-frequency electromagnetic fields alter the replication cycle of MS2 bacteriophage.

Effects of exposure to electromagnetic radiation at 835 MHz on growth, morphology and secretory characteristics of a mast cell analogue, RBL-2H3.

Cell membrane lipid molecular dynamics in a solenoid versus a magnetically shielded room.

Studies on the possible biological effects of 50 Hz electric and/or magnetic fields: evaluation of some glycolytic enzymes, glycolytic flux, energy and oxido-reductive potentials in human erythrocytes exposed in vitro to power frequency fields.

Modelling the internal field distribution in human erythrocytes exposed to MW radiation.

Nonlinear dynamical law governs magnetic field induced changes in lymphoid phenotype.

Extremely low frequency electromagnetic fields and heat shock can increase microvesicle motility in astrocytes.

[Extremely low frequency electromagnetic radiation enhanced energy metabolism and induced oxidative stress in Caenorhabditis elegans].

Exposure to low-frequency pulsed electromagnetic fields increases mitogen-induced lymphocyte proliferation in Down's syndrome.

Injury by electrical forces: pathophysiology, manifestations, and therapy.

Joint actions of environmental nonionizing electromagnetic fields and chemical pollution in cancer promotion.

Cellular communication in clone 9 cells exposed to magnetic fields.

Spindle disturbances in human-hamster hybrid (A(L)) cells induced by the electrical component of the mobile communication frequency range signal.

Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action.

Neoplastic transformation of C3H/10T1/2 cells following exposure to 120-Hz modulated 2.45-GHz microwaves and phorbol ester tumor promoter.

Effects of 50 Hz electromagnetic fields on rat cortical synaptosomes.

Effect of pulsed electromagnetic field exposure on adenosine receptors in rat brain.

[Effect of sinusoidal electricity magnetic fields on the proliferation and differentiation of osteoblasts in vitro].

A study of the electric field distribution in erythrocyte and rod shape cells from direct RF exposure.

Carcinogenesis and initiation of cell cycling by charge-induced membrane clusters may be due to mitogen receptors and Na+/H+ antiports.

The effect of a high frequency electromagnetic field in the microwave range on red blood cells.

[A static magnetic field loading system for in vitro cultured cells].

Mobile phones modulate response patterns of human brain activity.

Cytokine profile of human peripheral blood mononuclear cells exposed to 50 Hz EMF.

Low-Frequency Electromagnetic Field Exposure Enhances Extracellular Trap Formation by Human Neutrophils through the NADPH Pathway.

Alterations in protein kinase activity following exposure of cultured human lymphocytes to modulated microwave fields.

Offset of the vacuolar potential of Characean cells in response to electromagnetic radiation over the range 250 Hz-250 kHz.

Effect of 935-MHz phone-simulating electromagnetic radiation on endometrial glandular cells during mouse embryo implantation.

Human standing balance is affected by exposure to pulsed ELF magnetic fields: light intensity-dependent effects.

Effects of ELF (1-120 Hz) and modulated (50 Hz) RF fields on the efflux of calcium ions from brain tissue in vitro.

Influence of extremely low frequency magnetic fields on Ca2+ signaling and NMDA receptor functions in rat hippocampus.

A 50 Hz sinusoidal magnetic field does not damage MG-63 three-dimensional tumor spheroids but induces changes in their invasive properties.

A mechanism for action of oscillating electric fields on cells.

Electromagnetic fields (1.8 GHz) increase the permeability to sucrose of the blood-brain barrier in vitro.

ELF magnetic fields increase amino acid uptake into Vicia faba L. roots and alter ion movement across the plasma membrane.

Nonlinear determinism in the immune system. In vivo influence of electromagnetic fields on different functions of murine lymphocyte subpopulations.

Modulation of cell death in the rat thymus. Light and electron microscopic investigations.

Spindle disturbances in human-hamster hybrid (AL) cells induced by mobile communication frequency range signals.

Occupational exposure to static, ELF, VF and VLF magnetic fields and immune parameters.

Role of radical pairs and feedback in weak radio frequency field effects on biological systems.

Vacuolar hyperpolarizing offsets in characean cells exposed to mono- and bichromatic CW and to squarewave-modulated electromagnetic radiation in the band 200-1,000 MHz.

Evaluations of Acute and Sub-Acute Biological Effects of Narrowband and Moderate-Band High Power Electromagnetic Waves on Cellular Spheroids.

A 0.5 G, 60 Hz magnetic field suppresses melatonin production in pinealocytes.

Response of the seminiferous epithelium of the mouse exposed to low dose high energy (HZE) and electromagnetic radiation.

Initial studies on the effects of combined 60 Hz electric and magnetic field exposure on the immune system of nonhuman primates.

Induction of stress proteins by electromagnetic fields in cultured HL-60 cells.

Electromagnetic fields may act via calcineurin inhibition to suppress immunity, thereby increasing risk for opportunistic infection: Conceivable mechanisms of action.

The vacuolar potential of Characean cells subjected to electromagnetic radiation in the range 200-8,200 MHz.

Radiation and brain calcium: a review and critique.

Effects of a moderate-intensity static magnetic field on VEGF-A stimulated endothelial capillary tubule formation in vitro.

Low-amplitude, high-frequency electromagnetic field exposure causes delayed and reduced growth in Rosa hybrida.

[The laboratory detection of intra-cellular factors of anti-viral defense under community-acquired pneumonia in evaluation of effects of low-intensity microwave radiation].

Induced mitogenic activity in AML-12 mouse hepatocytes exposed to low-dose ultra-wideband electromagnetic radiation.

Are there modulated electromagnetic field effects on human conscious perception during attentional blink test?

Effects of electromagnetic radiation in the range 20-300 MHz on the vacuolar potential of characean cells.

Frohlich electromagnetic radiation from human leukocytes: implications for leukocyte adherence inhibition test.

Evaluation of health risks caused by radio frequency accelerated carcinogenesis: the importance of processes driven by the calcium ion signal.

Diacetyl and 2,3-pentanedione exposure of human cultured airway epithelial cells: Ion transport effects and metabolism of butter flavoring agents.

[Mechanism of the biological impact of weak electromagnetic fields and in vitro effects of degassing of blood].

Effects of 45-Hz magnetic fields on the functional state of the human brain.

Some characteristics of the glutathione cycle revealed by ionising and non-ionising electromagnetic radiation.

Magnetic Field Reference Levels for Arbitrary Periodic Waveforms for Prevention of Peripheral Nerve Stimulation.

Magnetism and cardiac arrhythmias.

Aluminum, calcium ion and radiofrequency synergism in acceleration of lymphomagenesis.

Low-frequency electromagnetic fields induce a stress effect upon higher plants, as evident by the universal stress signal, alanine.

Electromagnetic Fields and Stem Cell Fate: When Physics Meets Biology.

Inhibition of neuronal high-voltage activated calcium channels by the omega-phoneutria nigriventer Tx3-3 peptide toxin.

Effect of pulsed high frequency electromagnetic radiation on embryonic mouse palate in vitro.

[The physical mechanism of the effect of low-intensity electromagnetic radiation on biological cells].

[Changes in the acaricidal properties of organophosphorus compounds under the influence of magnetic resonance treatment].

Functional changes in human peripheral neutrophils in workers with different exposure to noxious agents.

Relationship between the Contents of Cyclins, Cyclin-Dependent Kinases, and Their Inhibitors in Whole Blood Mononuclear Leukocytes during the Postclinical Stage of Community-Acquired Pneumonia under the Influence of 1-GHz Microwaves.

Circadian locomotor activity of Musca flies: recording method and effects of 10 Hz square-wave electric fields.

The spark of life: electricity and regeneration.

Sensitive model with which to detect athermal effects of non-ionizing electromagnetic radiation.

--Leaf Cluster 24 (111)

Theme - Gene expression alterations following radiofrequency exposure

Titles

2.45 GHz radiofrequency fields alter gene expression in cultured human cells.

Whole-genome expression analysis in primary human keratinocyte cell cultures exposed to 60 GHz radiation.

Analysis of gene expression in a human-derived glial cell line exposed to 2.45 GHz continuous radiofrequency electromagnetic fields.

Analysis of gene expression in mouse brain regions after exposure to 1.9 GHz radiofrequency fields.

Analysis of proto-oncogene and heat-shock protein gene expression in human derived cell-lines exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field.

Characterization of biological effect of 1763 MHz radiofrequency exposure on auditory hair cells.

Using model organism Saccharomyces cerevisiae to evaluate the effects of ELF-MF and RF-EMF exposure on global gene expression.

Expression of cancer-related genes in human cells exposed to 60 Hz magnetic fields.

Effects on protein kinase C and gene expression in a human mast cell line, HMC-1, following microwave exposure.

Gene expression analysis of a human lymphoblastoma cell line exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field.

Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent.

Gene expression changes in human cells after exposure to mobile phone microwaves.

Gene expression profiles in white blood cells of volunteers exposed to a 50 Hz electromagnetic field.

Gene Expression Analysis in Human Peripheral Blood Cells after 900 MHz RF-EMF Short-Term Exposure.

In vitro study of the effects of ELF electric fields on gene expression in human epidermal cells.

Analysis of gene expression in two human-derived cell lines exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field.

[Global gene response to GSM 1800 MHz radiofrequency electromagnetic field in MCF-7 cells].

Biological effects of EMF exposure on Ets genes.

Evaluation of HSP70 expression and DNA damage in cells of a human trophoblast cell line exposed to 1.8 GHz amplitude-modulated radiofrequency fields.

Effects of the exposure to intermittent 1.8 GHz radio frequency electromagnetic fields on HSP70 expression and MAPK signaling pathways in PC12 cells.

2-GHz band CW and W-CDMA modulated radiofrequency fields have no significant effect on cell proliferation and gene expression profile in human cells.

Mobile phone radiation might alter protein expression in human skin.

Radiofrequency radiation (900 MHz) induces Egr-1 gene expression and affects cell-cycle control in human neuroblastoma cells.

Evaluation of bax, bcl-2, p21 and p53 genes expression variations on cerebellum of BALB/c mice before and after birth under mobile phone radiation exposure.

HSP70 expression in human trophoblast cells exposed to different 1.8 Ghz mobile phone signals.

Effects of exposure to a 1950 MHz radio frequency field on expression of Hsp70 and Hsp27 in human glioma cells.

Analysis of Gene Expression in Mice Testes Exposed to 1.765 GHz Microwave in Utero.

Modulation of heat shock protein response in SH-SY5Y by mobile phone microwaves.

Gene expression in human breast epithelial cells exposed to 60 Hz magnetic fields.

Gene and protein expression following exposure to radiofrequency fields from mobile phones.

Influence of high-frequency electromagnetic fields on different modes of cell death and gene expression.

Biological stress responses to radio frequency electromagnetic radiation: are mobile phones really so (heat) shocking?

A Genome-Wide mRNA Expression Profile in Caenorhabditis elegans under Prolonged Exposure to 1750MHz Radiofrequency Fields.

Impact of 60-GHz millimeter waves on stress and pain-related protein expression in differentiating neuron-like cells.

[Effects of GSM 1800 MHz radiofrequency electromagnetic fields on protein expression profile of human breast cancer cell MCF-7].

Gene expression and reproductive abilities of male Drosophila melanogaster subjected to ELF-EMF exposure.

Biological monitoring of non-thermal effects of mobile phone radiation: recent approaches and challenges.

Mobile-phone radiation-induced perturbation of gene-expression profiling, redox equilibrium and sporadic-apoptosis control in the ovary of Drosophila melanogaster.

The genotoxic effect of radiofrequency waves on mouse brain.

Connection between Cell Phone use, p53 Gene Expression in Different Zones of Glioblastoma Multiforme and Survival Prognoses.

Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes.

In vivo modulation of ETS genes induced by electromagnetic fields.

Human health consequences of environmentally-modulated gene expression: potential roles of ELF-EMF induced epigenetic versus mutagenic mechanisms of disease.

Exposure to 2.45 GHz electromagnetic fields induces hsp70 at a high SAR of more than 20 W/kg but not at 5W/kg in human glioma MO54 cells.

Hsp70 is an independent stress marker among frequent users of mobile phones.

Study of p53 expression and post-transcriptional modifications after GSM-900 radiofrequency exposure of human amniotic cells.

Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: molecular mechanism for cancer- and blood-brain barrier-related effects.

Exposure to global system for mobile communication (GSM) cellular phone radiofrequency alters gene expression, proliferation, and morphology of human skin fibroblasts.

Expression analysis of human HL60 cells exposed to 60 Hz square- or sine-wave magnetic fields.

p53, Rb and bcl-2 expression during the cell cycle: a study in phytohaemagglutinin stimulated lymphocytes and microwave irradiated lymphoid tissue sections.

Human skin cell stress response to GSM-900 mobile phone signals. In vitro study on isolated primary cells and reconstructed epidermis.

In vitro study of the stress response of human skin cells to GSM-1800 mobile phone signals compared to UVB radiation and heat shock.

Proteomic analysis on the alteration of protein expression in the early-stage placental villous tissue of electromagnetic fields associated with cell phone exposure.

Activity and expression of acetylcholinesterase in PC12 cells exposed to intermittent 1.8 GHz 217-GSM mobile phone signal.

Effect of 900 MHz electromagnetic fields on nonthermal induction of heat-shock proteins in human leukocytes.

In vitro effect of cell phone radiation on motility, DNA fragmentation and clusterin gene expression in human sperm.

Electromagnetic fields at a mobile phone frequency (900 MHz) trigger the onset of general stress response along with DNA modifications in Eisenia fetida earthworms.

Effects of a 2450 MHz high-frequency electromagnetic field with a wide range of SARs on the induction of heat-shock proteins in A172 cells.

Effect of GSM-900 and -1800 signals on the skin of hairless rats. III: Expression of heat shock proteins.

Analysis of the cellular stress response in MCF10A cells exposed to combined radio frequency radiation.

The Effect of Radiation Emitted by Cell Phone on The Gelatinolytic Activity of Matrix Metalloproteinase-2 and -9 of Mouse Pre-Antral Follicles during In Vitro Culture.

Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the hsp27/p38MAPK stress pathway in rat brain.

[Responses of thymocytes and splenocytes to low-intensity extremely high-frequency electromagnetic radiation in normal mice and in mice with systemic inflammation].

Analysis of proteome response to the mobile phone radiation in two types of human primary endothelial cells.

Mobile phone electromagnetic radiation activates MAPK signaling and regulates viability in Drosophila.

Novel electric power-driven hydrodynamic injection system for gene delivery: safety and efficacy of human factor IX delivery in rats.

Proto-oncogene mRNA levels and activities of multiple transcription factors in C3H 10T 1/2 murine embryonic fibroblasts exposed to 835.62 and 847.74 MHz cellular phone communication frequency radiation.

Effect of cell phone-like electromagnetic radiation on primary human thyroid cells.

Expression of the immediate early gene, c-fos, in mouse brain after acute global system for mobile communication microwave exposure.

[Effects of high power microwave on the expressions of Bcl-2 and C-myc proteins in the rat testis].

[The Impact of Electroacupuncture Intervention on Expression of 5-HTR 1 B/2 C Genes in Mice under Radiation Stimulation from Mobile Phone].

Cell phone use and parotid salivary gland alterations: no molecular evidence.

Electromagnetic fields may act directly on DNA.

Stimulation of ubiquitin-proteasome pathway through the expression of amidohydrolase for N-terminal asparagine (Ntan1) in cultured rat hippocampal neurons exposed to static magnetism.

Proteomic analysis of human lens epithelial cells exposed to microwaves.

[Effects of electromagnetic pulses on apoptosis and TGF-beta3 expression of mouse testis tissue].

[Changes in Ca(2+)concentration and caspase-3 expression and their relationship in Raji cells exposed to electromagnetic radiation].

Effects of pulsed electromagnetic fields on cartilage apoptosis signalling pathways in ovariectomised rats.

Upregulation of specific mRNA levels in rat brain after cell phone exposure.

Meta-proteomic analysis of protein expression distinctive to electricity-generating biofilm communities in air-cathode microbial fuel cells.

Response of Caenorhabditis elegans to wireless devices radiation exposure.

[The role of heat shock proteins HSP90 in the response of immune cells to centimeter microwaves].

Mechanism of short-term ERK activation by electromagnetic fields at mobile phone frequencies.

[Effects of electromagnetic radiation on RAF/MEK/ERK signaling pathway in rats hippocampus].

Electromagnetic-pulse-induced activation of p38 MAPK pathway and disruption of blood-retinal barrier.

Electromagnetic fields at mobile phone frequency induce apoptosis and inactivation of the multi-chaperone complex in human epidermoid cancer cells.

Electromagnetic wave irradiation promotes osteoblastic cell proliferation and up-regulates growth factors via activation of the ERK1/2 and p38 MAPK pathways.

Millimeter-wave exposure promotes the differentiation of bone marrow stromal cells into cells with a neural phenotype.

Electromagnetic pulse activated brain microglia via the p38 MAPK pathway.

Cytotoxicity of temozolomide on human glioblastoma cells is enhanced by the concomitant exposure to an extremely low-frequency electromagnetic field (100Hz, 100G).

Exposure to 50 Hz electromagnetic radiation promote early maturation and differentiation in newborn rat cerebellar granule neurons.

Analysis of the novel excretory cell expressed ECP-1 protein and its proposed ECP-1/IFC-2 fusion protein EXC-2 in the nematode Caenorhabditis elegans.

Microwave induced alteration in the neuron specific enolase gene expression.

Cytosolic calreticulin inhibits microwave radiation-induced microvascular endothelial cell injury through the integrin-focal adhesion kinase pathway.

Experimental study of millimeter wave-induced differentiation of bone marrow mesenchymal stem cells into chondrocytes.

Effect of 72 Hz pulsed magnetic field exposure on ras p21 expression in CCRF-CEM cells.

Bcl-2 and p53 immunoprofile in Kaposi's sarcoma.

The amelioration of phagocytic ability in microglial cells by curcumin through the inhibition of EMF-induced pro-inflammatory responses.

Cell phone use is associated with an inflammatory cytokine profile of parotid gland saliva.

Calreticulin protects rat microvascular endothelial cells against microwave radiation-induced injury by attenuating endoplasmic reticulum stress.

Qualitative effect on mRNAs of injury-associated proteins by cell phone like radiation in rat facial nerves.

p53 immunoreactivity in cutaneous PUVA tumors is similar to that in other non-melanoma skin neoplasms.

Effects of 2.45 GHz electromagnetic fields with a wide range of SARs on bacterial and HPRT gene mutations.

Microglia M1/M2 polarization contributes to electromagnetic pulse-induced brain injury.

900-MHz microwave radiation enhances gamma-ray adverse effects on SHG44 cells.

[The role of RKIP mediated ERK pathway in hippocampus neurons injured by electromagnetic radiation].

Isoflurane preconditioning ameliorates electromagnetic pulse-induced neural damage by shifting microglia polarization toward anti-inflammatory phenotype via upregulation of SOCS1.

Effects of prolonged exposure to moderate static magnetic field and its synergistic effects with alkaline pH on Enterococcus faecalis.

Abnormal physical architecture of the lipophilic domains of human sperm membrane in oligospermia: a logical cause for low fertility profiles.

[Ecological and biological characteristics of Drosophila melanogaster features depending on the dose of electromagnetic radiation of various types].

Construction and clinical significance of a predictive system for prognosis of hepatocellular carcinoma.

--Leaf Cluster 11 (51)

Theme - Adverse impacts of radiofrequency fields on sleep

Titles

Stimulation of the brain with radiofrequency electromagnetic field pulses affects sleep-dependent performance improvement.

Exposure to radiofrequency electromagnetic fields and sleep quality: a prospective cohort study.

Effects of mobile phone exposure (GSM 900 and WCDMA/UMTS) on polysomnography based sleep quality: An intra- and inter-individual perspective.

Environmental Radiofrequency Electromagnetic Fields Exposure at Home, Mobile and Cordless Phone Use, and Sleep Problems in 7-Year-Old Children.

Radio frequency electromagnetic field exposure in humans: Estimation of SAR distribution in the brain, effects on sleep and heart rate.

Wireless communication fields and non-specific symptoms of ill health: a literature review.

Cohort study on the effects of everyday life radio frequency electromagnetic field exposure on non-specific symptoms and tinnitus.

Memory performance, wireless communication and exposure to radiofrequency electromagnetic fields: A prospective cohort study in adolescents.

Exposure to pulse-modulated radio frequency electromagnetic fields affects regional cerebral blood flow.

Symptoms and the use of wireless communication devices: A prospective cohort study in Swiss adolescents.

Sleep after mobile phone exposure in subjects with mobile phone-related symptoms.

Cognitive performance measures in bioelectromagnetic research--critical evaluation and recommendations.

Sleep duration, quality, and timing and their associations with age in a community without electricity in Haiti.

Radiofrequency electromagnetic field exposure and non-specific symptoms of ill health: a systematic review.

Children's health and RF EMF exposure. Views from a risk assessment and risk communication perspective.

Human sleep under the influence of pulsed radiofrequency electromagnetic fields: a polysomnographic study using standardized conditions.

Conduct of a personal radiofrequency electromagnetic field measurement study: proposed study protocol.

Effects of electromagnetic fields emitted from W-CDMA-like mobile phones on sleep in humans.

International policy and advisory response regarding children's exposure to radio frequency electromagnetic fields (RF-EMF).

Effects of short- and long-term pulsed radiofrequency electromagnetic fields on night sleep and cognitive functions in healthy subjects.

Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG.

Effects of electromagnetic fields emitted by mobile phones (GSM 900 and WCDMA/UMTS) on the macrostructure of sleep.

Individual variation in temporal relationships between exposure to radiofrequency electromagnetic fields and non-specific physical symptoms: A new approach in studying 'electrosensitivity'.

Mobile phone use, behavioural problems and concentration capacity in adolescents: A prospective study.

Towards 5G communication systems: Are there health implications?

Acute effects of electromagnetic fields emitted by GSM mobile phones on subjective well-being and physiological reactions: a meta-analysis.

A Prospective Cohort Study of Adolescents' Memory Performance and Individual Brain Dose of Microwave Radiation from Wireless Communication.

[Investigation of sleep disorders in the vicinity of high frequency transmitters].

Effect of a single 30 min UMTS mobile phone-like exposure on the thermal pain threshold of young healthy volunteers.

Quality Matters: Systematic Analysis of Endpoints Related to "Cellular Life" in Vitro Data of Radiofrequency Electromagnetic Field Exposure.

The effect of electromagnetic fields emitted by mobile phones on human sleep.

Mobile phone 'talk-mode' signal delays EEG-determined sleep onset.

The response of human bacteria to static magnetic field and radiofrequency electromagnetic field.

Exposure to radio-frequency electromagnetic fields and behavioural problems in Bavarian children and adolescents.

Human sleep EEG under the influence of pulsed radio frequency electromagnetic fields. Results from polysomnographies using submaximal high power flux densities.

[Effects of radio- and microwaves emitted by wireless communication devices on the functions of the nervous system selected elements].

Effects of radiation emitted by WCDMA mobile phones on electromagnetic hypersensitive subjects.

Electromagnetic radiation and behavioural response of ticks: an experimental test.

Effects of Sleep Quality on the Association between Problematic Mobile Phone Use and Mental Health Symptoms in Chinese College Students.

Investigating short-term exposure to electromagnetic fields on reproductive capacity of invertebrates in the field situation.

Could myelin damage from radiofrequency electromagnetic field exposure help explain the functional impairment electrohypersensitivity? A review of the evidence.

Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study.

Cochlear implants in the etiopathogenesis of glioblastoma--an interesting observation or independent finding?

Terrestrial Trunked Radio (TETRA) exposure and its impact on slow cortical potentials.

Influence of electromagnetic fields emitted by GSM-900 cellular telephones on the circadian patterns of gonadal, adrenal and pituitary hormones in men.

[Prevalence of insomnia in adults aged 18 to 60 years and exposure to electromagnetic fields in households of Barranquilla, Colombia].

"Triple M" Effect: A Proposed Mechanism to Explain Increased Dental Amalgam Microleakage after Exposure to Radiofrequency Electromagnetic Radiation.

The Effect of a Single 30-Min Long Term Evolution Mobile Phone-Like Exposure on Thermal Pain Threshold of Young Healthy Volunteers.

Pain, pain intensity and pain disability in high school students are differently associated with physical activity, screening hours and sleep.

Long-Term Evolution Electromagnetic Fields Exposure Modulates the Resting State EEG on Alpha and Beta Bands.

Microwaves emitted by cellular telephones affect human slow brain potentials.

--Leaf Cluster 41 (125)

Theme - Adverse effects of radiofrequency fields on cells

Titles

The effects of radiofrequency fields on cell proliferation are non-thermal.

Effects of RF-EMF Exposure from GSM Mobile Phones on Proliferation Rate of Human Adipose-derived Stem Cells: An In-vitro Study.

Effects of radiofrequency exposure emitted from a GSM mobile phone on proliferation, differentiation, and apoptosis of neural stem cells.

Are the young more sensitive than adults to the effects of radiofrequency fields? An examination of relevant data from cellular and animal studies.

Comparative study of cell cycle kinetics and induction of apoptosis or necrosis after exposure of human Mono Mac 6 cells to radiofrequency radiation.

Apoptosis induced by ultraviolet radiation is enhanced by amplitude modulated radiofrequency radiation in mutant yeast cells.

Review of possible modulation-dependent biological effects of radiofrequency fields.

An in vitro study of the effects of exposure to a GSM signal in two human cell lines: monocytic U937 and neuroblastoma SK-N-SH.

Ornithine decarboxylase activity is affected in primary astrocytes but not in secondary cell lines exposed to 872 MHz RF radiation.

Disturbance of cell proliferation in response to mobile phone frequency radiation.

Effects of chronic exposure to radiofrequency electromagnetic fields on energy balance in developing rats.

Exposure to 835 MHz radiofrequency electromagnetic field induces autophagy in hippocampus but not in brain stem of mice.

Continuous exposure to 900MHz GSM-modulated EMF alters morphological maturation of neural cells.

Enhancement of X-ray Induced Apoptosis by Mobile Phone-Like Radio-Frequency Electromagnetic Fields in Mouse Spermatocyte-Derived Cells.

Cell oxidation-reduction imbalance after modulated radiofrequency radiation.

Pulse modulated 900 MHz radiation induces hypothyroidism and apoptosis in thyroid cells: a light, electron microscopy and immunohistochemical study.

1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.

Non-thermal biomarkers of exposure to radiofrequency/microwave radiation.

In vitro non-thermal oxidative stress response after 1800 MHz radiofrequency radiation.

Effect of radiofrequency electromagnetic field exposure on in vitro models of neurodegenerative disease.

Influence of a 902.4 MHz GSM signal on the human visual system: investigation of the discrimination threshold.

Biological indicators in response to radiofrequency/microwave exposure.

The protective effect of autophagy on mouse spermatocyte derived cells exposure to 1800MHz radiofrequency electromagnetic radiation.

Proliferation and apoptosis in a neuroblastoma cell line exposed to 900 MHz modulated radiofrequency field.

p25/CDK5 is partially involved in neuronal injury induced by radiofrequency electromagnetic field exposure.

Microwave exposure of neuronal cells in vitro: Study of apoptosis.

Apoptosis is induced by radiofrequency fields through the caspase-independent mitochondrial pathway in cortical neurons.

[Impact of radiofrequency/microwave radiation on cell and cytoskeleton structure].

Reaction of the immune system to low-level RF/MW exposures.

Exposure to Global System for Mobile Communication 900 MHz Cellular Phone Radiofrequency Alters Growth, Proliferation and Morphology of Michigan Cancer Foundation-7 Cells and Mesenchymal Stem Cells.

Exposure to 900 MHz radiofrequency radiation induces caspase 3 activation in proliferating human lymphocytes.

Proteomic analysis of continuous 900-MHz radiofrequency electromagnetic field exposure in testicular tissue: a rat model of human cell phone exposure.

Anthropogenic Radio-Frequency Electromagnetic Fields Elicit Neuropathic Pain in an Amputation Model.

Effect of high SARs produced by cell phone like radiofrequency fields on mollusk single neuron.

Investigation of the effects of 2.1 GHz microwave radiation on mitochondrial membrane potential (DeltaPsim), apoptotic activity and cell viability in human breast fibroblast cells.

Comparison of 864 MHz and 935 MHz microwave radiation effects on cell culture.

Free radical release and HSP70 expression in two human immune-relevant cell lines after exposure to 1800 MHz radiofrequency radiation.

Effects of GSM-modulated radiofrequency electromagnetic fields on B-cell peripheral differentiation and antibody production.

Effect of radiofrequency radiation in cultured mammalian cells: A review.

Effect of 835 MHz radiofrequency radiation exposure on calcium binding proteins in the hippocampus of the mouse brain.

Viability and phagocytosis of neutrophils exposed in vitro to 100-MHz radiofrequency radiation.

Effects of 3G cell phone exposure on the structure and function of the human cytochrome P450 reductase.

Possible effects of radiofrequency electromagnetic fields on in vivo C6 brain tumors in Wistar rats.

In-vitro exposure of neuronal networks to the GSM-1800 signal.

Mitochondrial hyperpolarization and cytochrome-c release in microwave-exposed MCF-7 cells.

Does MW Radiation Affect Gene Expression, Apoptotic Level, and Cell Cycle Progression of Human SH-SY5Y Neuroblastoma Cells?

Effect of exposure to the edge signal on oxidative stress in brain cell models.

Impact of 864 MHz or 935 MHz radiofrequency microwave radiation on the basic growth parameters of V79 cell line.

Effect of a 2.45-GHz radiofrequency electromagnetic field on neutrophil chemotaxis and phagocytosis in differentiated human HL-60 cells.

Effects of low intensity radiofrequency electromagnetic fields on electrical activity in rat hippocampal slices.

Effects of mobile phone type signals on calcium levels within human leukaemic T-cells (Jurkat cells).

Does exposure to a radiofrequency electromagnetic field modify thermal preference in juvenile rats?

Measurement of the 100MHz EMF radiation in vivo effects on zebrafish D. rerio embryonic development: A multidisciplinary study.

Microwave effects on the nervous system.

[Effects of radiofrequency electromagnetic fields on mammalian spermatogenesis].

Effects of simultaneous combined exposure to CDMA and WCDMA electromagnetic field on immune functions in rats.

Microwaves from Mobile Phones Inhibit 53BP1 Focus Formation in Human Stem Cells More Strongly Than in Differentiated Cells: Possible Mechanistic Link to Cancer Risk.

Effects of 900-MHz radio frequencies on the chemotaxis of human neutrophils in vitro.

Problems in assessment of risks from exposures to microwaves of mobile communication.

Bioassay for assessing cell stress in the vicinity of radio-frequency irradiating antennas.

Heart rate variability affected by radiofrequency electromagnetic field in adolescent students.

Mobile phone radiation alters proliferation of hepatocarcinoma cells.

A new in vitro exposure device for the mobile frequency of 900 MHz.

A radio-frequency system for in vivo pilot experiments aimed at the studies on biological effects of electromagnetic fields.

Modeling cell dynamics under mobile phone radiation.

Evaluation of the potential of mobile phone specific electromagnetic fields (UMTS) to produce micronuclei in human glioblastoma cell lines.

Exposure to radiation from single or combined radio frequencies provokes macrophage dysfunction in the RAW 264.7 cell line.

Responses of neurons to an amplitude modulated microwave stimulus.

Pathophysiology of cell phone radiation: oxidative stress and carcinogenesis with focus on male reproductive system.

An HF exposure system for mice with improved efficiency.

Cell phone radiation effects on cytogenetic abnormalities of oral mucosal cells.

Sleep EEG alterations: effects of different pulse-modulated radio frequency electromagnetic fields.

Effects of exposure to DAMPS and GSM signals on ornithine decarboxylase (ODC) activity: II. SH-SY5Y human neuroblastoma cells.

Design, optimization, realization, and analysis of an in vitro system for the exposure of embryonic stem cells at 1.71 GHz.

Cell physiological effects of radiofrequency electromagnetic fields.

Acute effect of exposure of mollusk single neuron to 900-MHz mobile phone radiation.

[Dependence of the non-thermal radiofrequency electromagnetic field bioeffects on the typological features of electroencephalogram in humans].

Age-dependent acute interference with stem and progenitor cell proliferation in the hippocampus after exposure to 1800 MHz electromagnetic radiation.

Biological effects of radiofrequency radiation: concepts and criteria.

Effects of radiofrequency electromagnetic fields on seed germination and root meristematic cells of Allium cepa L.

Effects of exposure to DAMPS and GSM signals on ornithine decarboxylase (ODC) activity: I. L-929 mouse fibroblasts.

An investigation of the effects of TETRA RF fields on intracellular calcium in neurones and cardiac myocytes.

Apoptotic cell death during Drosophila oogenesis is differentially increased by electromagnetic radiation depending on modulation, intensity and duration of exposure.

Biological effects of amplitude-modulated radiofrequency radiation.

Cell phone radiations affect early growth of Vigna radiata (mung bean) through biochemical alterations.

The use of FDTD in establishing in vitro experimentation conditions representative of lifelike cell phone radiation on the spermatozoa.

Effects of RF fields emitted from smart phones on cardio-respiratory parameters: a preliminary provocation study.

Exposure to 900 MHz electromagnetic field induces an unbalance between pro-apoptotic and pro-survival signals in T-lymphoblastoid leukemia CCRF-CEM cells.

Basis for optimization of in vitro exposure apparatus for health hazard evaluations of mobile communications.

In vitro effect of pulsed 900 MHz GSM radiation on mitochondrial membrane potential and motility of human spermatozoa.

The effect of pulsed 900-MHz GSM mobile phone radiation on the acrosome reaction, head morphometry and zona binding of human spermatozoa.

Prenatal exposure to radiofrequencies: effects of WiFi signals on thymocyte development and peripheral T cell compartment in an animal model.

Combined effects of flow-induced shear stress and electromagnetic field on neural differentiation of mesenchymal stem cells.

Long-term electromagnetic exposure of developing neuronal networks: A flexible experimental setup.

Influence of radiofrequency-electromagnetic waves from 3rd-generation cellular phones on fertilization and embryo development in mice.

Simulation of electromagnetic fields in the human body using Finite Integration Technique (FIT).

The implications of non-linear biological oscillations on human electrophysiology for electrohypersensitivity (EHS) and multiple chemical sensitivity (MCS).

Comparison of biological effects between continuous and intermittent exposure to GSM-900-MHz mobile phone radiation: Detection of apoptotic cell-death features.

[Effects of mobile phones and radar radiofrequencies on the eye].

Exposure to 1950-MHz TD-SCDMA electromagnetic fields affects the apoptosis of astrocytes via caspase-3-dependent pathway.

Intrauterine effects in animals exposed to radiofrequency and microwave fields.

Effect of cell phone radiation on neutrophil of mice.

In vitro effects of radiofrequency electromagnetic waves on bovine spermatozoa motility.

[Role of modulation in biological effects of electromagnetic radiation].

Numerical and experimental dosimetry of Petri dish exposure setups.

Effects of 2.45-GHz electromagnetic fields with a wide range of SARs on micronucleus formation in CHO-K1 cells.

Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study.

Modulation of oxidative phosphorylation (OXPHOS) by radiation- induced biophotons.

The possible global hazard of cell phone radiation on thyroid cells and hormones: a systematic review of evidences.

Radiofrequency-induced carcinogenesis: cellular calcium homeostasis changes as a triggering factor.

Mobile phone electromagnetic radiation affects Amyloid Precursor Protein and alpha-synuclein metabolism in SH-SY5Y cells.

Exposure to cell phone radiations produces biochemical changes in worker honey bees.

Human mesenchymal stem cells are sensitive to abnormal gravity and exhibit classic apoptotic features.

[Surface markers and functions of human dendritic cells exposed to mobile phone 1800 MHz electromagnetic fields].

Activation of the TRPV1 Thermoreceptor Induced by Modulated or Unmodulated 1800 MHz Radiofrequency Field Exposure.

Low power radiofrequency electromagnetic radiation for the treatment of pain due to osteoarthritis of the knee.

Calcium-binding proteins and GFAP immunoreactivity alterations in murine hippocampus after 1 month of exposure to 835 MHz radiofrequency at SAR values of 1.6 and 4.0 W/kg.

Alteration of glycine receptor immunoreactivity in the auditory brainstem of mice following three months of exposure to radiofrequency radiation at SAR 4.0 W/kg.

Radiofrequency (RF) effects on blood cells, cardiac, endocrine, and immunological functions.

Influence of electromagnetic waves, with maxima in the green or red range, on the morphofunctional properties of multipotent stem cells.

Can exposure to a terrestrial trunked radio (TETRA)-like signal cause symptoms? A randomised double-blind provocation study.

Potential protection of green tea polyphenols against 1800 MHz electromagnetic radiation-induced injury on rat cortical neurons.

Effects of 2450 MHz electromagnetic fields with a wide range of SARs on methylcholanthrene-induced transformation in C3H10T1/2 cells.

Assessment of intermittent UMTS electromagnetic field effects on blood circulation in the human auditory region using a near-infrared system.

Skin changes in "screen dermatitis" versus classical UV- and ionizing irradiation-related damage--similarities and differences.

Fourth Level Cluster 81 (673)
Theme - Adverse impacts of power-line EMF

--Leaf Cluster 9 (43)

Theme - Adverse effects of ELF magnetic field exposures

Titles

[Electromagnetic noise blocks the gap-junctional intercellular communication suppression induced by 50 Hz magnetic field].

Developmental effects of magnetic field (50 Hz) in combination with ionizing radiation and chemical teratogens.

The effect of extremely low frequency magnetic field on the conjunctiva and goblet cells.

Chronic exposure to an extremely low-frequency magnetic field induces depression-like behavior and corticosterone secretion without enhancement of the hypothalamic-pituitary-adrenal axis in mice.

Effect of coexposure to 50 Hz magnetic fields and an aneugen on human lymphocytes, determined by the cytokinesis block micronucleus assay.

[Superposition of noise magnetic fields inhibits clustering of fibroblast membrane surface receptors induced by 50 Hz magnetic fields in Chinese hamster lungs].

Extremely low frequency magnetic field induces hyperalgesia in mice modulated by nitric oxide synthesis.

The cardiovascular response to an acute 1800-microT, 60-Hz magnetic field exposure in humans.

Mouse early embryos obtained by natural breeding or in vitro fertilization display a differential sensitivity to extremely low-frequency electromagnetic fields.

Interaction of MF 50 Hz, 10 mT with high dose of X-rays: evaluation of embryotoxicity in chick embryos.

Effects on micronuclei formation of 60-Hz electromagnetic field exposure with ionizing radiation, hydrogen peroxide, or c-Myc overexpression.

Rodent cell transformation and immediate early gene expression following 60-Hz magnetic field exposure.

[A study on dose-effect of suppression to gap junctional intercellular communication function by 50-Hz magnetic fields].

Effect of magnetic field exposure on anchorage-independent growth of a promoter-sensitive mouse epidermal cell line (JB6).

Exposure to 60-Hz magnetic fields and proliferation of human astrocytoma cells in vitro.

Activation of Signaling Cascades by Weak Extremely Low Frequency Electromagnetic Fields.

Effects of ELF magnetic fields on protein expression profile of human breast cancer cell MCF7.

[Noise magnetic fields block co-suppression effect induced by power frequency magnetic field and phorbol ester].

The response of the human circulatory system to an acute 200-muT, 60-Hz magnetic field exposure.

Exposure of Drosophila melanogaster embryonic cell cultures to 60-Hz sinusoidal magnetic fields: assessment of potential teratogenic effects.

Non-thermal effects of power-line magnetic fields (50 Hz) on gene expression levels of pluripotent embryonic stem cells-the role of tumour suppressor p53.

[Abnormal shift of connexin 43 gap-junction protein induced by 50 Hz electromagnetic fields in Chinese hamster lung cells].

[Effects of power frequency magnetic field on gap junction intercellular communication of astrocytes].

Mutation induction by high-density, 50-Hz magnetic fields in human MeWo cells exposed in the DNA synthesis phase.

Micronucleus induction in Syrian hamster embryo cells following exposure to 50 Hz magnetic fields, benzo(a)pyrene, and TPA in vitro.

[Estimation of magnetic radiation effects on leucocytes].

[Effects of electromagnetic noise on the enhancement of stress-activated protein kinase(SAPK) phosphorylation induced by 50 Hz magnetic fields].

Micronucleus formation in human amnion cells after exposure to 50 Hz MF applied horizontally and vertically.

Immune function and host defense in rodents exposed to 60-Hz magnetic fields.

Effects of 50-Hz magnetic field exposure on hormone secretion and apoptosis-related gene expression in human first trimester villous trophoblasts in vitro.

Effects of whole-body 50-Hz magnetic field exposure on mouse Leydig cells.

Superposition of an incoherent magnetic field inhibited EGF receptor clustering and phosphorylation induced by a 1.8 GHz pulse-modulated radiofrequency radiation.

Effects of 50 Hz sinusoidal magnetic fields on Hsp27, Hsp70, Hsp90 expression in porcine aortic endothelial cells (PAEC).

Effect of 60 Hz magnetic field exposure on c-fos expression in stimulated PC12 cells.

Immune markers and ornithine decarboxylase activity among electric utility workers.

Neural mass modeling of power-line magnetic fields effects on brain activity.

Modulation of natural killer cell function after exposure to 60 Hz magnetic fields: confirmation of the effect in mature B6C3F1 mice.

Alteration of tight and adherens junctions on 50-Hz magnetic field exposure in Madin Darby canine kidney (MDCK) cells.

Genome-wide transcription analysis of Escherichia coli in response to extremely low-frequency magnetic fields.

Real-time detection of stimulus response in cultured neurons by high-intensity intermediate-frequency magnetic field exposure.

Magnetic field desensitizes 5-HT(1B) receptor in brain: pharmacological and functional studies.

Influence of combined AC-DC magnetic fields on free radicals in organized and biological systems. Development of a model and application of the radical pair mechanism to radicals in micelles.

A 1.8-GHz radiofrequency radiation induces EGF receptor clustering and phosphorylation in cultured human amniotic (FL) cells.

--Leaf Cluster 17 (55)

Theme - Adverse impacts of EMF on mammary cancer development

Titles

Acceleration of mammary tumorigenesis by exposure of 7,12-dimethylbenz[a]anthracene-treated female rats in a 50-Hz, 100-microT magnetic field: replication study.

A histopathological study on alterations in DMBA-induced mammary carcinogenesis in rats with 50 Hz, 100 muT magnetic field exposure.

Effects of magnetic fields on mammary tumor development induced by 7,12-dimethylbenz(a)anthracene in rats.

Significant differences in the effects of magnetic field exposure on 7,12-dimethylbenz(a)anthracene-induced mammary carcinogenesis in two substrains of Sprague-Dawley rats.

Effects of weak alternating magnetic fields on nocturnal melatonin production and mammary carcinogenesis in rats.

Do cocarcinogenic effects of ELF electromagnetic fields require repeated long-term interaction with carcinogens? Characteristics of positive studies using the DMBA breast cancer model in rats.

Effect of 26 week magnetic field exposures in a DMBA initiation-promotion mammary gland model in Sprague-Dawley rats.

Effect of 13 week magnetic field exposures on DMBA-initiated mammary gland carcinomas in female Sprague-Dawley rats.

Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats.

[Effects of mobile-phone microwave on dimethylbenz (a) anthracene induced mammary carcinoma development in rats].

Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats.

Developmental toxicity evaluation of ELF magnetic fields in Sprague-Dawley rats.

Developmental toxicity study of 60 Hz (power frequency) magnetic fields in rats.

In vivo exposure of rats to a weak alternating magnetic field increases ornithine decarboxylase activity in the mammary gland by a similar extent as the carcinogen DMBA.

A study on skin tumour formation in mice with 50 Hz magnetic field exposure.

Rat liver foci study on coexposure with 50 Hz magnetic fields and known carcinogens.

5-Iododeoxyuridine-125I incorporation in vivo after exposure to a 50 Hz magnetic field.

Study on potential effects of "902-MHz GSM-type Wireless Communication Signals" on DMBA-induced mammary tumours in Sprague-Dawley rats.

Anxiogenic effect of chronic exposure to extremely low frequency magnetic field in adult rats.

Acute and subchronic toxicity of 20 kHz and 60 kHz magnetic fields in rats.

Results of lifespan exposure to continuous and intermittent extremely low frequency electromagnetic fields (ELFEMF) administered alone to Sprague Dawley rats.

Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves.

Acute effects of 50 Hz magnetic field exposure on human visual task and cardiovascular performance.

Cancer promotion in a mouse-skin model by a 60-Hz magnetic field: II. Tumor development and immune response.

Repeated exposure of C3H/HeJ mice to ultra-wideband electromagnetic pulses: lack of effects on mammary tumors.

[Effects of chronic exposure of power frequency magnetic field on neurobehavior in rats].

Multigeneration reproductive toxicity assessment of 60-Hz magnetic fields using a continuous breeding protocol in rats.

Assessing the potential carcinogenic activity of magnetic fields using animal models.

Evaluation of the potential carcinogenicity of 60 Hz linear sinusoidal continuous-wave magnetic fields in Fischer F344 rats.

Effect of exposure to extremely low electro-magnetic field during prenatal period on mice spleen.

Effects of magnetic field exposure on the development of lung fibrosis elicited by industrial pollutants.

Effect of radiofrequency radiation exposure on mouse skin tumorigenesis initiated by 7,12-dimethybenz[alpha]anthracene.

Recent experimental data on Extremely Low Frequency (ELF) magnetic field carcinogenic risk: open questions.

Extremely low-frequency magnetic fields modulate nitric oxide signaling in rat brain.

[Modifying effect of light and electromagnetic field on development of mammary tumors induced by N-nitrosomethyl urea in female rats].

Effects of subchronic extremely low-frequency electromagnetic field exposure on biochemical parameters in rats.

Effects of gestational exposure to a video display terminal-like magnetic field (20-kHz) on CBA/S mice.

Evaluation of the developmental toxicity of 60 Hz magnetic fields and harmonic frequencies in Sprague-Dawley rats.

Testing electromagnetic fields for potential carcinogenic activity: a critical review of animal models.

Effects of aluminum and extremely low frequency electromagnetic radiation on oxidative stress and memory in brain of mice.

Skeletal muscle HSP72 and norepinephrine response to static magnetic field in rat.

Effect of chronic exposure to a GSM-like signal (mobile phone) on survival of female Sprague-Dawley rats: modulatory effects by month of birth and possibly stage of the solar cycle.

Long-term exposure of Sprague Dawley rats to 20 kHz triangular magnetic fields.

Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves.

In vivo studies of the effect of magnetic field exposure on ontogeny of choline acetyltransferase in the rat brain.

Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats exposed to frequency-modulated microwave fields.

Toxicity bioassay in Sprague-Dawley rats exposed to 20 kHz triangular magnetic field for 90 days.

A cerebral primitive neuroectodermal tumor in a squirrel monkey (Saimiri sciureus).

Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model.

A case of hepatocellular carcinoma rupturing after angiography.

A case of recurring hepatocellular carcinoma with a solitary Virchow's lymph node metastasis.

Benzodiazepine system is involved in hyperalgesia in rats induced by the exposure to extremely low frequency magnetic fields.

[A case report of primary hepatic carcinoid with lymph node metastasis--treatment of hepatic arterial infusion to post-reoperative liver and radiation to metastasis of para-aortic lymph nodes].

Effects of mobile phone radiation on UV-induced skin tumourigenesis in ornithine decarboxylase transgenic and non-transgenic mice.

[A case of renal cell carcinoma in a horseshoe kidney].

--Leaf Cluster 6 (67)

Theme - Adverse health effects of magnetic fields associated with magnetic resonance imaging

Titles

Menometrorrhagia in magnetic resonance imaging operators with copper intrauterine contraceptive devices (IUDS): a case report.

Safety issues in magnetic resonance imaging.

[Magnetic resonance imaging : Recent studies on biological effects of static magnetic and highfrequency electromagnetic fields].

A review of the current use of magnetic resonance imaging in pregnancy and safety implications for the fetus.

Retrospective assessment of exposure to static magnetic fields during production and development of magnetic resonance imaging systems.

[Safety of magnetic resonance imaging in patients with implanted cardiovascular devices].

Implantable pulse generators (pacemakers) and electrodes: safety in the magnetic resonance imaging scanner environment.

Health risk assessment of occupational exposure to a magnetic field from magnetic resonance imaging devices.

Calculation of radiofrequency electromagnetic fields and their effects in MRI of human subjects.

Biological effects of exposure to magnetic resonance imaging: an overview.

The safety of MRI. Considerations for site planning and clinical use.

Interference with cardiac pacemakers by magnetic resonance imaging: are there irreversible changes at 0.5 Tesla?

Evaluation of occupational exposure in magnetic resonance sites.

Safety of strong, static magnetic fields.

Guidelines and recommendations for MR imaging safety and patient management. III. Questionnaire for screening patients before MR procedures. The SMRI Safety Committee.

Safety concerns related to magnetic field exposure.

Occupational exposure of healthcare and research staff to static magnetic stray fields from 1.5-7 Tesla MRI scanners is associated with reporting of transient symptoms.

Magnetic resonance imaging safety: implications for cardiovascular patients.

Exposure classification of MRI workers in epidemiological studies.

MRI magnetic field stimulates rotational sensors of the brain.

[Exposure to static magnetic field and health hazards during the operation of magnetic resonance scanners].

The effects of 1.5T magnetic resonance imaging on early murine in-vitro embryo development.

Effect of electromagnetic field accompanying the magnetic resonance imaging on human heart rate variability - a pilot study.

A comprehensive analysis of MRI research risks: in support of full disclosure.

Exposure to time varying magnetic fields associated with magnetic resonance imaging reduces fentanyl-induced analgesia in mice.

MRI effects on craniofacial size and crown-rump length in C57BL/6J mice in 1.5T fields.

RF-EMF exposure of fetus and mother during magnetic resonance imaging.

Effects of static magnetic fields on cognition, vital signs, and sensory perception: a meta-analysis.

Pilot study investigating the effect of the static magnetic field from a 9.4-T MRI on the vestibular system.

Exposure to static and time-varying magnetic fields from working in the static magnetic stray fields of MRI scanners: a comprehensive survey in the Netherlands.

Safety considerations in MR imaging.

Human exposure to 4.0-Tesla magnetic fields in a whole-body scanner.

Vestibular stimulation by magnetic fields.

EMF exposure variation among MRI sequences from pediatric examination protocols.

Adaptive suppression of power line interference in ultra-low field magnetic resonance imaging in an unshielded environment.

Exposure, health complaints and cognitive performance among employees of an MRI scanners manufacturing department.

[ECG changes caused by the effect of static magnetic fields of nuclear magnetic resonance tomography using magnets with a field power of 0.5 to 4.0 Telsa].

Development of hypertension after long-term exposure to static magnetic fields among workers from a magnetic resonance imaging device manufacturing facility.

An improved quasi-static finite-difference scheme for induced field evaluation in MRI based on the biconjugate gradient method.

A trail of artificial vestibular stimulation: electricity, heat, and magnet.

Prediction of specific absorption rate in mother and fetus associated with MRI examinations during pregnancy.

Effect of 1.5 tesla nuclear magnetic resonance imaging scanner on implanted permanent pacemakers.

MRI safety: everyone's job.

MR procedures: biologic effects, safety, and patient care.

Operational safety issues in MRI.

Biologic effects and potential hazards of nuclear magnetic imaging.

Novel mechanistic model and computational approximation for electromagnetic safety evaluations of electrically short implants.

[Possible mutagenic effects of magnetic fields].

[Effect of a static magnetic field (3.5 T) on the reproductive behavior of mice, on the embryo and fetal development and on selected hematologic parameters].

[Bacterial mutation in high magnetic fields and radiofrequency radiation].

Neurophysiology: vertigo in MRI machines.

Complex magnetic field exposure system for in vitro experiments at intermediate frequencies.

Magnetic-field-induced vertigo: a theoretical and experimental investigation.

Effects of magnetic stray fields from a 7 tesla MRI scanner on neurocognition: a double-blind randomised crossover study.

Effect of a 0.5-T static magnetic field on conduction in guinea pig spinal cord.

Cognitive, cardiac, and physiological safety studies in ultra high field magnetic resonance imaging.

Magnetic resonance imaging of the chest. Where we stand.

Exposure to static magnetic fields and risk of accidents among a cohort of workers from a medical imaging device manufacturing facility.

MR safety: past, present, and future from a historical perspective.

Pacemaker reed switch behavior in 0.5, 1.5, and 3.0 Tesla magnetic resonance imaging units: are reed switches always closed in strong magnetic fields?

Offline impedance measurements for detection and mitigation of dangerous implant interactions: an RF safety prescreen.

[Do strong static magnetic fields in NMR tomography modify tissue perfusion?].

Aneurysm clips: evaluation of magnetic field interactions and translational attraction by use of "long-bore" and "short-bore" 3.0-T MR imaging systems.

Modeling of the internal fields distribution in human inner hearing system exposed to 900 and 1800 MHz.

Safety aspects of switched gradient fields.

Effect on germination and early growth characteristics in sunflower (Helianthus annuus) seeds exposed to static magnetic field.

INFLUENCE OF STATIC ELECTRICITY ON RADON MEASUREMENT USING PASSIVE DETECTORS.

--Leaf Cluster 32 (139)

Theme - Health risks of power-line electromagnetic fields on humans

Titles

An examination of underlying physical principles. The interaction of power-line electromagnetic fields with the human body.

The establishment of frequency dependent limits for electric and magnetic fields and evaluation of indirect effects.

Health risks of electric and magnetic fields caused by high-voltage systems in Finland.

Electric field induced in the human body by uniform 50 Hz electric or magnetic fields: bibliography analysis and method for conservatively deriving measurable limits.

LEVELS OF EXTREMELY LOW-FREQUENCY ELECTRIC AND MAGNETIC FIELDS FROM OVERHEAD POWER LINES IN THE OUTDOOR ENVIRONMENT OF RAMALLAH CITY-PALESTINE.

Exposure of workers in the electric power industry to electric and magnetic fields.

Comparison of cardiac-induced endogenous fields and power frequency induced exogenous fields in an anatomical model of the human body.

Influence of 50 Hz electric and magnetic fields on the human heart.

Physiologic and dosimetric considerations for limiting electric fields induced in the body by movement in a static magnetic field.

The influence of 50 Hz electric and magnetic fields on the extrasystoles of human heart.

Computational estimation of magnetically induced electric fields in a rotating head.

[Evaluation of reports on environmental measurements of electromagnetic fields generated by high voltage transmission lines and substations].

Intensity of electric and magnetic fields from power lines within the business district of 60 Ontario communities.

Current densities in a pregnant woman model induced by simultaneous ELF electric and magnetic field exposure.

Basic restrictions in EMF exposure guidelines.

Numerical dosimetry at power-line frequencies using anatomically based models.

Impedance method computation of induced currents in a simple model of a child exposed to electromagnetic fields of an electric blanket.

[Effects of electromagnetic field emitted by electric blankets on brain catecholamine in fetal mice].

[Practical aspects of taking measurements of electromagnetic fields in the surrounding of overhead transmission lines].

Electric and magnetic field exposures for people living near a 735-kilovolt power line.

Nerves in a human body exposed to low-frequency electromagnetic fields.

Current densities and total contact currents for 110 and 220 kV power line tasks.

Evaluation and measurement of magnetic field exposure at a typical high-voltage substation and its power lines.

Current densities measured in human models exposed to 60-Hz electric fields.

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Fetal exposure to low frequency electric and magnetic fields.

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Electric field prediction for a human body-electric machine system.

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Effects of high-intensity power-frequency electric fields on implanted modern multiprogrammable cardiac pacemakers.

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A system for simultaneous exposure of small animals to 60-Hz electric and magnetic fields.

Comparison of electric field exposure measurement methods under power lines.

Quandaries in the application of the ICNIRP low frequency basic restriction on current density.

Human body exposure to power lines: relation of induced quantities to external magnetic fields.

Measurement and Modeling of Personal Exposure to the Electric and Magnetic Fields in the Vicinity of High Voltage Power Lines.

Effects of electric and magnetic fields from high-power lines on female urinary excretion of 6-sulfatoxymelatonin.

60-Hertz electric-field exposures in transmission line towers.

Possible health effects of 50/60Hz electric and magnetic fields: review of proposed mechanisms.

Pacemaker interference by magnetic fields at power line frequencies.

Non-Hodgkin's lymphoma among electric utility workers in Ontario: the evaluation of alternate indices of exposure to 60 Hz electric and magnetic fields.

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Comparison of various safety guidelines for electronic article surveillance devices with pulsed magnetic fields.

Influence of human model resolution on computed currents induced in organs by 60-Hz magnetic fields.

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The interference threshold of cardiac pacemakers in electric 50 Hz fields.

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Current densities and total contact currents during forest clearing tasks under 400 kV power lines.

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Clinical study of interference with cardiac pacemakers by a magnetic field at power line frequencies.

Effects of a high-voltage direct-current transmission line on beef cattle production.

Acute effects of ELF electromagnetic fields: a field study of linesmen working with 400 kV power lines.

[Diseases in animals associated with exposure to electric and magnetic fields of 50/60 Hz: report of a case].

Powerline frequency electric and magnetic fields: a pilot study of risk perception.

[Duration of conscious reactions in persons exposed to an electric field of 50 Hz frequency].

Implantable cardioverter defibrillators in electric and magnetic fields of 400 kV power lines.

Effects of low frequency electric fields on synaptic integration in hippocampal CA1 pyramidal neurons: implications for power line emissions.

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Theoretical limits on the threshold for the response of long cells to weak extremely low frequency electric fields due to ionic and molecular flux rectification.

An In Situ and In Silico Evaluation of Biophysical Effects of 27 MHz Electromagnetic Whole Body Humans Exposure Expressed by the Limb Current.

Interference with the pacemakers of two workers at electricity substations.

Sensitivity to electricity in the catfish, Parasilurus asotus.

Electric and magnetic fields generated by ac power lines: an application of advanced modelling tools in order to predict exposure levels.

[The characteristics of the electromagnetic situation close to overhead electric power transmission lines in St. Petersburg].

Human perception of electric fields and ion currents associated with high-voltage DC transmission lines.

[An experimental study of the sciatic nerve injury by high voltage electricity in rabbits].

Magnetic induction at 60 Hz in the human heart: a comparison between the in situ and isolated scenarios.

Exposure guidelines for low-frequency electric and magnetic fields: report from the Brussels workshop.

Power lines and ionizing radiation.

COMPUTATIONAL ASSESSMENT OF PREGNANT WOMAN MODELS EXPOSED TO UNIFORM ELF-MAGNETIC FIELDS: COMPLIANCE WITH THE EUROPEAN CURRENT EXPOSURE REGULATIONS FOR THE GENERAL PUBLIC AND OCCUPATIONAL EXPOSURES AT 50 Hz.

[Exposure of workers to electric and magnetic fields from radiofrequency dielectric heaters to process polyvinyl chloride material].

Effects of 50 Hz electric currents on vigilance and concentration.

Can disturbances in the atmospheric electric field created by powerline corona ions disrupt melatonin production in the pineal gland?

Experimental and numeric investigation about electromagnetic interference between implantable cardiac pacemaker and magnetic fields at power line frequency.

SAR changes in a human head model for plane wave exposure (500 - 2500 MHz) and a comparison with IEEE 2005 safety limits.

The possible consequences for cognitive functions of external electric fields at power line frequency on hippocampal CA1 pyramidal neurons.

Interference of 16.7-Hz electromagnetic fields on measured electrocardiogram.

Biological effects of a 765-kV transmission line: exposures and thresholds in honeybee colonies.

Power-frequency magnetic fields from electric blankets.

Electric fields in bone marrow substructures at power-line frequencies.

Analysis of the relationship between electromagnetic radiation characteristics and urban functions in highly populated urban areas.

Chronic exposure of primates to 60-Hz electric and magnetic fields: III. Neurophysiologic effects.

Exposure of the human body to professional and domestic induction cooktops compared to the basic restrictions.

Electricity and the heart.

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Biological effects of exposure to static electric fields in humans and vertebrates: a systematic review.

Biological effects of electric and magnetic fields on productivity of dairy cows.

Computer screens and brain cancer.

[Clinical analysis of brain injury in patients injured by high voltage electricity].

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A Shear-Mode Piezoelectric Heterostructure for Electric Current Sensing in Electric Power Grids.

Provocation of electric hypersensitivity under everyday conditions.

Chronic exposure of primates to 60-Hz electric and magnetic fields: I. Exposure system and measurements of general health and performance.

Low-voltage electricity-induced lung injury.

Biophysical cancer transformation pathway.

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[Pathomorphological constellation in death resulting from high voltage electricity].

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[Injuries caused by electricity].

Compound injury from high-voltage electricity.

Exposure to static electric fields leads to changes in biogenic amine levels in the brains of Drosophila.

Effects of electric field reduction in visual display units on skin symptoms.

Do induction loops pose a hazard to health?

Effects of concurrent exposure to 60 Hz electric and magnetic fields on the social behavior of baboons.

Assessment of motor pathways by magnetic stimulation in human and veterinary medicine.

Cow sensitivity to electricity during milking.

Characteristics and potential human health hazards of charged aerosols generated by high-voltage power lines.

Elimination of power line interference from ECG signals using recurrent neural networks.

Neuroelectric mechanisms applied to low frequency electric and magnetic field exposure guidelines--part II: non sinusoidal waveforms.

Electromagnetic field strength levels surrounding electronic article surveillance (EAS) systems.

Suppression of power-line interference by analog notch filtering in the ECG signal for heart rate variability analysis: to do or not to do?

Return to arc welding following defibrillator implantation.

Fracture due to shock from domestic electricity supply.

Cascading failures in ac electricity grids.

Renal artery thrombosis due to high voltage electricity.

The potential of electricity transmission corridors in forested areas as bumblebee habitat.

--Leaf Cluster 34 (188)

Theme - Adverse effects of low-frequency electromagnetic fields on humans

Titles

An evaluation of the existing evidence on the carcinogenic potential of extremely low frequency magnetic fields.

Possible mechanisms by which extremely low frequency magnetic fields affect opioid function.

Effects of 60 Hz magnetic fields on teenagers and adults.

Do extremely low frequency magnetic fields enhance the effects of environmental carcinogens? A meta-analysis of experimental studies.

Biological interactions and potential health effects of extremely-low-frequency magnetic fields from power lines and other common sources.

Effects of extremely low-frequency magnetic field on growth and differentiation of human mesenchymal stem cells.

[Morphological characteristics and various theories on the mechanism of biological effect of magnetic fields].

Developmental effects of extremely low frequency electric and magnetic fields.

[Very low frequency electric and magnetic fields and the immune system].

Concern that "EMF" magnetic fields from power lines cause cancer.

Effects of low-frequency magnetic fields on embryonic development and pregnancy.

Perspectives on health effects of electric and magnetic fields.

Assessment of ELF magnetic fields produced by independent power lines.

Low-frequency magnetic fields and cancer. What you should know and what to tell your patients.

Extremely low frequency magnetic field (50 Hz, 0.5 mT) modifies fitness components and locomotor activity of Drosophila subobscura.

Extremely low frequency magnetic field effects on metabolite of Aspergillus niger.

Extremely-low frequency magnetic field effects on sulfate reducing bacteria viability.

Extremely low frequency (ELF) magnetic fields and apoptosis: a review.

Variability and consistency of electric and magnetic field occupational exposure measurements.

The genotoxic potential of electric and magnetic fields: an update.

Power frequency electromagnetic fields and health. Where's the evidence?

Interaction of static and extremely low frequency electric and magnetic fields with living systems: health effects and research needs.

[Biological influences of electromagnetic fields].

[Biological and health effects on electric and magnetic fields at extremely low frequencies].

[Biological effects of nonionizing radiation: low frequency electromagnetic fields].

Extremely low-frequency magnetic fields of transformers and possible biological and health effects.

EMF and health.

[Evaluation of the effects of electric and magnetic fields in humans].

Induction of kinetochore-positive and kinetochore-negative micronuclei in CHO cells by ELF magnetic fields and/or X-rays.

Epidemiological studies of work with video display terminals and adverse pregnancy outcomes (1984-1992).

Possible health hazards from exposure to power-frequency electric and magnetic fields--a COMAR Technical Information Statement.

Extremely low frequency (ELF) magnetic fields enhance chemically induced formation of apurinic/apyrimidinic (AP) sites in A172 cells.

Occupational exposure to intermediate frequency and extremely low frequency magnetic fields among personnel working near electronic article surveillance systems.

[The role of free radicals in mechanisms of biological function exposed to weak, constant and net magnetic fields].

Behavioural evidence that magnetic field effects in the land snail, Cepaea nemoralis, might not depend on magnetite or induced electric currents.

The effect of a 50 Hz magnetic field on cognitive function in humans.

Exposure assessment for electric and magnetic fields.

Assessment of occupational exposure to extremely low frequency magnetic fields in hospital personnel.

Origins of electromagnetic hypersensitivity to 60 Hz magnetic fields: A provocation study.

Exposure to magnetic fields of railway engine drivers: a case study in Italy.

Acute exposure to 50-Hz magnetic fields increases interleukin-6 in young healthy men.

A review of the literature on potential reproductive and developmental toxicity of electric and magnetic fields.

[Influence of low magnetic field on lipid peroxidation].

50-Hz magnetic field exposure system for small animals.

Typical exposure of children to EMF: exposimetry and dosimetry.

[Risks of electromagnetic fields for humans].

Low-frequency pulsed electromagnetic field exposure can alter neuroprocessing in humans.

A critical review of the genotoxic potential of electric and magnetic fields.

Occupational exposures of pharmacists and pharmaceutical assistants to 60 Hz magnetic fields.

Evaluation of in vitro effects of 50 and 60 Hz magnetic fields in regional EMF exposure facilities.

Exposure system to study hypotheses of ELF and RF electromagnetic field interactions of mobile phones with the central nervous system.

Exposure of welders and other metal workers to ELF magnetic fields.

Safety of the magnetic field generated by a neuronal magnetic stimulator: evaluation of possible mutagenic effects.

Exposure of high resolution fetuses in advanced pregnant woman models at different stages of pregnancy to uniform magnetic fields at the frequency of 50 Hz.

The epidemiology of electric and magnetic field exposures in the power frequency range and reproductive outcomes.

Food and Drug Administration low-level extremely-low-frequency magnetic field exposure facility.

Analyses of magnetic-field peak-exposure summary measures.

Increased resorptions in CBA mice exposed to low-frequency magnetic fields: an attempt to replicate earlier observations.

Cancer promotion in a mouse-skin model by a 60-Hz magnetic field: I. Experimental design and exposure system.

Influence of weak static and 50 Hz magnetic fields on the redox activity of cytochrome-C oxidase.

Evaluating alternative exposure indices in epidemiologic studies on extremely low-frequency magnetic fields.

Amyotrophic lateral sclerosis (ALS) and extremely-low frequency (ELF) magnetic fields: a study in the SOD-1 transgenic mouse model.

Can low-level 50/60 Hz electric and magnetic fields cause biological effects?

Effects of information and 50 Hz magnetic fields on cognitive performance and reported symptoms.

Electromagnetic radiation from VDT units: study of the effectiveness of an active shielding device.

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The wonders of magnetism.

Human exposure to 60-Hz magnetic fields: neurophysiological effects.

Effects of extremely low-frequency magnetic field exposure on cognitive functions: results of a meta-analysis.

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Human electrophysiological and cognitive effects of exposure to ELF magnetic and ELF modulated RF and microwave fields: a review of recent studies.

Biophysical mechanisms: a component in the weight of evidence for health effects of power-frequency electric and magnetic fields.

Cardiac autonomic control mechanisms in power-frequency magnetic fields: a multistudy analysis.

Magnetic fields of video display terminals and pregnancy outcome.

[Exposure to low electromagnetic fields and the carcinogenesis process].

[Sister chromatid exchange (SCE)and high-frequency cells in workers professionally exposed to extremely low-frequency magnetic fields (ELF)].

Bio-effects of high magnetic fields: a study using a simple animal model.

Exposure to ELF magnetic and ELF-modulated radiofrequency fields: the time course of physiological and cognitive effects observed in recent studies (2001-2005).

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Individual subject sensitivity to extremely low frequency magnetic field.

Assessment of magnetic field exposures for a mortality study at a uranium enrichment plant.

Human cognitive performance in a 3 mT power-line frequency magnetic field.

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Biological effects of extremely low-frequency electromagnetic fields: in vivo studies.

Effects in rodents of a 1-month exposure to magnetic fields (200-1200 Gauss).

Symptoms of the musculoskeletal system and exposure to magnetic fields in an aluminium plant.

Children's exposure to magnetic fields produced by U.S. television sets used for viewing programs and playing video games.

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Implantable cardioverter defibrillator and 50-Hz electric and magnetic fields exposure in the workplace.

A study of heart rate and heart rate variability in human subjects exposed to occupational levels of 50 Hz circularly polarised magnetic fields.

Measuring exposed magnetic fields of welders in working time.

Intermittent exposures to nanoTesla range, 7 Hz, amplitude-modulated magnetic fields increase regeneration rates in planarian.

Cancer from exposure to 50/60 Hz electric and magnetic fields--a major scientific debate.

Chronic or intractable medical problems associated with prolonged exposure to unsuspected harmful environmental electric, magnetic or electro-magnetic fields radiating in the bedroom or workplace and their exacerbation by intake of harmful light and heavy metals from common sources.

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Possible cocarcinogenic effects of ELF electromagnetic fields may require repeated long-term interaction with known carcinogenic factors.

Psychological effects of chronic exposure to 50 Hz magnetic fields in humans living near extra-high-voltage transmission lines.

Effects of short term exposure to 60 Hz electromagnetic fields on interleukin 1 and interleukin 6 production by peritoneal exudate cells.

Apoptosis in haemopoietic progenitor cells exposed to extremely low-frequency magnetic fields.

ELF magnetic fields in a city environment.

Assessing compliance with 60-hertz magnetic-field exposure guidelines.

[Epidemiological study of populations exposed to high levels of 50 Hz magnetic fields].

Temporally incoherent magnetic fields mitigate the response of biological systems to temporally coherent magnetic fields.

Evaluation of potential health effects of 10 kHz magnetic fields: a short-term mouse toxicology study.

Magnetic fields and radical reactions: recent developments and their role in nature.

Ambient 60-Hz magnetic flux density in an urban neighborhood.

Brief exposure to a 50 Hz, 100 microT magnetic field: effects on reaction time, accuracy, and recognition memory.

The influence of a temporary magnetic field on chicken hatching.

Characterization of Children's Exposure to Extremely Low Frequency Magnetic Fields by Stochastic Modeling.

Natural killer cell activity decreases in workers occupationally exposed to extremely low frequency magnetic fields exceeding 1 microT.

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Measurement of low frequency magnetic fields from digital cellular telephones.

Magnetic fields of video display terminals and spontaneous abortion.

Epidemiologic studies of electric and magnetic fields and cancer: a case study of distortions by the media.

Non-ionizing electromagnetic radiation: a study of carcinogenic and cancer treatment potential.

Electromagnetic field exposure and cancer: a review of epidemiologic evidence.

[State of peripheral blood of technical personnel exposed to constant magnetic fields].

Magnetic field exposure and arrythmic risk: evaluation in railway drivers.

Prevalence of self-reported hypersensitivity to electric or magnetic fields in a population-based questionnaire survey.

Involvement of eddy currents in the mutagenicity of ELF magnetic fields.

The effects of weak magnetic fields on radical pairs.

Urban exposure to ELF magnetic field due to high-, medium- and low-voltage electricity supply networks.

[The effect of low-frequency electromagnetic fields on the development of experimental mammary tumors].

ECG changes in humans exposed to 50 Hz magnetic fields.

On the role of the interactions of ions with external magnetic fields in physiologic processes and their importance in chronobiology.

Relative-risk-estimate bias and loss of power in the Mantel test for trend resulting from the use of magnetic-field point-in-time ("spot") measurements in epidemiological studies based on an ordinal exposure scale.

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Some non neoplastic effects of ELF magnetic fields in experimental animals.

Alternate indices of electric and magnetic field exposures among Ontario electrical utility workers.

[The effect of electromagnetic fields on living organisms: plants, birds and animals].

Calcium homeostasis and low-frequency magnetic and electric field exposure: A systematic review and meta-analysis of in vitro studies.

Exposure to magnetic field harmonics in the vicinity of indoor distribution substations.

Comparison between personal exposure to 60 Hz magnetic fields and stationary home measurements for people living near and away from a 735 kV power line.

A 60 Hz electric and magnetic field exposure facility for nonhuman primates: design and operational data during experiments.

Reduction of laser-induced retinal injury applying the combination of the 3D variable electric and magnetic fields in "vivo".

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Speculations on the influence of electromagnetism on genomic and associated structures.

Methodology of a study on the French population exposure to 50 Hz magnetic fields.

Hypersensitivity of human subjects to environmental electric and magnetic field exposure: a review of the literature.

MYC mRNA abundance is unchanged in subcultures of HL60 cells exposed to power-line frequency magnetic fields.

Do naturally occurring magnetic nanoparticles in the human body mediate increased risk of childhood leukaemia with EMF exposure?

Free radical mechanism for the effects of environmental electromagnetic fields on biological systems.

Facility for chronic exposure of rats to ELF magnetic fields.

Dynamic characteristics of membrane ions in multifield configurations of low-frequency electromagnetic radiation.

Flight deck magnetic fields in commercial aircraft.

Cardiovascular alterations in Macaca monkeys exposed to stationary magnetic fields: experimental observations and theoretical analysis.

Personal power-frequency magnetic field exposure in women recruited at an infertility clinic: association with physical activity and temporal variability.

Evaluating exposure cutpoint bias in epidemiologic studies of electric and magnetic fields.

Specific patterns of weak (1 microTesla) transcerebral complex magnetic fields differentially affect depression, fatigue, and confusion in normal volunteers.

Effects of 50 Hz magnetic field exposure on human heart rate variability with passive tilting.

Comment on "designing EMF experiments: what is required to characterize 'exposure'?".

[Effect of high intensity magnetic field on the processes of early growth in plant seeds and development of honeybees].

Low-frequency electromagnetic radiation enhances the induction of rat mammary tumors by nitrosomethyl urea.

Possible disruption of remote viewing by complex weak magnetic fields around the stimulus site and the possibility of accessing real phase space: a pilot study.

60 Hertz magnetic field exposure assessment for an investigation of leukemia in telephone lineworkers.

The heliogeophysical aspects of circumpolar health.

The effects of continuous exposure to 20-kHz sawtooth magnetic fields on the litters of CD-1 mice.

[Magnetic field on the deranged accommodation of visual detector terminal operators].

Macro- and trace element concentrations in blood plasma and cerebrospinal fluid of dairy cows exposed to electric and magnetic fields.

Assessment of occupational exposure patterns by frequency-domain analysis of time series data.

Field enhancement of nonreciprocal electromagnetic wave supported by magnetic surface plasmon.

Residential magnetic field measurements in France: comparison of indoor and outdoor measurements.

Sleep EEG alterations: effects of pulsed magnetic fields versus pulse-modulated radio frequency electromagnetic fields.

Hypothesis on a casual link between EMF and an evolutionary class of cancer and spontaneous abortion.

Exposure to alternating electromagnetic fields and effects on the visual and visuomotor systems.

Statistical review of the henhouse experiments: the effects of a pulsed magnetic field on chick embryos.

A pilot study on the reproductive risks of maternal exposure to magnetic fields from electronic article surveillance systems.

How do honeybees use their magnetic compass? Can they see the North?

Temporal trends and misclassification in residential 60 Hz magnetic field measurements.

The precautionary principle and electric and magnetic fields.

[Assessment of exposure to extremely low frequency magnetic field emitted from monitors].

Anthropogenic electromagnetic noise disrupts magnetic compass orientation in a migratory bird.

A comparison of rheumatoid arthritis and fibromyalgia patients and healthy controls exposed to a pulsed (200 microT) magnetic field: effects on normal standing balance.

An Investigation on the Effect of Extremely Low Frequency Pulsed Electromagnetic Fields on Human Electrocardiograms (ECGs).

Patient reactions to some electromagnetic fields from dental chair and unit: a pilot study.

Occupational 50 Hz magnetic field exposure measurements among female sewing machine operators in Hungary.

Anatomical localization of human detection of weak electromagnetic radiation: experiments with dowsers.

Paroxysmal itching in multiple sclerosis during treatment with external magnetic fields.

Natural very-low-frequency sferics and headache.

Nanoscale Design of Nano-Sized Particles in Shape-Memory Polymer Nanocomposites Driven by Electricity.

Effect of the alternative magnetic stimulation on peripheral circulation for regenerative medicine.

Influence of 50-Hz electromagnetic field on anurian (Xenopus laevis) metamorphosis.

--Leaf Cluster 40 (116)

Theme - Adverse effects of low-frequency magnetic fields on rodents

Titles

Influence of 60-Hertz magnetic fields on leukemia.

Long-term exposure of male and female mice to 50 Hz magnetic field: effects on fertility.

Effects of extremely low-frequency electromagnetic fields (ELF-EMF) exposure on B6C3F1 mice.

Deficits in spatial learning after exposure of mice to a 50 Hz magnetic field.

Eight-week toxicity study of 60 Hz magnetic fields in F344 rats and B6C3F1 mice.

Dominant lethal studies in male mice after exposure to a 50-Hz electric field.

Long term effects of a 50 Hz electric field on the life-expectancy of mice.

Effect of a 9 mT pulsed magnetic field on C3H/Bi female mice with mammary carcinoma. A comparison between the 12 Hz and the 460 Hz frequencies.

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in F344/N rats.

Effects of power frequency alternating magnetic fields on reproduction and pre-natal development of mice.

Hematopoietic neoplasia in C57BL/6 mice exposed to split-dose ionizing radiation and circularly polarized 60 Hz magnetic fields.

A gross morphologic, histologic, hematologic, and blood chemistry study of adult and neonatal mice chronically exposed to high magnetic fields.

Behavioral effects of long-term exposure to magnetic fields in rats.

Behavioral studies with mice exposed to DC and 60-Hz magnetic fields.

Carcinogenicity test of 50 Hz sinusoidal magnetic fields in rats.

Dominant lethal studies in male mice after exposure to a 50 Hz magnetic field.

Toxic effects of 50 Hz electromagnetic field on memory consolidation in male and female mice.

Behavioral sensitivity of rats to extremely-low-frequency magnetic fields.

Rats are not aversive when exposed to 60-Hz magnetic fields at 3.03 mT.

Effects of low-frequency magnetic fields on fetal development in rats.

Effect of pulsed magnetic fields on leukemia-prone AKR mice. No-effect on mortality through five generations.

Teratogenic effect of broad-band electromagnetic field on neonatal mice (Mus musculus).

Effect of a magnetic field on ascorbate system in mice.

Spatial learning deficit in the rat after exposure to a 60 Hz magnetic field.

Biologic effects of prolonged exposure to ELF electromagnetic fields in rats: II. 50 Hz magnetic fields.

Neurodevelopmental anomalies of the hippocampus in rats exposed to weak intensity complex magnetic fields throughout gestation.

Assessment of biological changes of continuous whole body exposure to static magnetic field and extremely low frequency electromagnetic fields in mice.

[VDT pulse magnetic field enhances teratogenic effect of ara-c in mice].

Electric field exposure and evidence of stress in mice.

Subchronic in vivo effects of a high static magnetic field (9.4 T) in rats.

Exposure to a theta-burst patterned magnetic field impairs memory acquisition and consolidation for contextual but not discrete conditioned fear in rats.

Radial maze proficiency of adult Wistar rats given prenatal complex magnetic field treatments.

Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice.

Teratogenic effects of static magnetic field on mouse fetuses.

Effect of electric field in conditioned aversion response.

Acute exposure to a 50 Hz magnetic field impairs consolidation of spatial memory in rats.

Effects of radiofrequency electromagnetic fields (UMTS) on reproduction and development of mice: a multi-generation study.

Combined effects of complex magnetic fields and agmatine for contextual fear learning deficits in rats.

Enhanced mortality of rat pups following inductions of epileptic seizures after perinatal exposures to 5 nT, 7 Hz magnetic fields.

DEXA analysis on the bones of rats exposed in utero and neonatally to static and 50 Hz electric fields.

Gender- and age-specific impairment of rat performance in the Morris water maze following prenatal exposure to an MRI magnetic field.

Repeated exposure attenuates the behavioral response of rats to static high magnetic fields.

Metallothionein content increased in the liver of mice exposed to magnetic fields.

[Alternating magnetic field damages the reproductive function of murine testes].

Initial exposure to 30 kV/m or 60 kV/m 60 Hz electric fields produces temporary cessation of operant behavior of nonhuman primates.

Extremely low-frequency magnetic fields can impair spermatogenesis recovery after reversible testicular damage induced by heat.

Teratological evaluation of mouse fetuses exposed to a 20 kHz EMF.

Fetal loss in mice exposed to magnetic fields during early pregnancy.

Effects of pulsed magnetic fields on the developing mouse embryo.

The effect of the prenatal and post-natal long-term exposure to 50 Hz electric field on growth, pubertal development and IGF-1 levels in female Wistar rats.

Carcinogenicity study of GSM and DCS wireless communication signals in B6C3F1 mice.

Lymphoma induced in mice chronically exposed to very strong low-frequency electromagnetic field.

Short-term memory in mice is affected by mobile phone radiation.

Nonhuman primates will not respond to turn off strong 60 Hz electric fields.

[Effects of 1800 MHz GSM-like exposure on the gonadal function and hematological parameters of male mice].

Detection thresholds for 60 Hz electric fields by nonhuman primates.

Effects of exposure to a 50 Hz electric field on plasma levels of lactate, glucose, free Fatty acids, triglycerides and creatine phosphokinase activity in hind-limb ischemic rats.

Transient and cumulative memory impairments induced by GSM 1.8 GHz cell phone signal in a mouse model.

The effect of very low dose pulsed magnetic waves on cochlea.

Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields.

Effects of combined ferrous sulphate administration and exposure to static magnetic field on spatial learning and motor abilities in rats.

Autism-relevant social abnormalities in mice exposed perinatally to extremely low frequency electromagnetic fields.

Urinary 6-sulphatoxymelatonin excretion is increased in rats after 24 hours of exposure to vertical 50 Hz, 100 microT magnetic field.

Developmental effects of perinatal exposure to extremely weak 7 Hz magnetic fields and nitric oxide modulation in the Wistar albino rat.

Developmental profiles of growth-associated protein (Gap43), Ngfb, Bndf and Ntf4 mRNA levels in the rat forebrain after exposure to 60 Hz magnetic fields.

Prenatal exposures to LTP-patterned magnetic fields: quantitative effects on specific limbic structures and acquisition of contextually conditioned fear.

Effects of exposure to a 60-kV/m, 60-Hz electric field on the social behavior of baboons.

Effects of a 60 Hz magnetic field on central cholinergic systems of the rat.

Electromagnetic waves of 900 MHz in acute pentylenetetrazole model in ontogenesis in mice.

Reduced litter sizes following 48-h of prenatal exposure to 5 nT to 10 nT,0.5 Hz magnetic fields: implications for sudden infant deaths.

Effects of Electromagnetic Radiation from Smartphones on Learning Ability and Hippocampal Progenitor Cell Proliferation in Mice.

Rats avoid exposure to HVdc electric fields: a dose response study.

Biological effects of long-duration, high-field (4 T) MRI on growth and development in the mouse.

Behavioral in-effectiveness of high frequency electromagnetic field in mice.

Does static electric field from ultra-high voltage direct-current transmission lines affect male reproductive capacity? Evidence from a laboratory study on male mice.

Carcinogenicity study of 217 Hz pulsed 900 MHz electromagnetic fields in Pim1 transgenic mice.

Five-tesla static magnetic fields suppress food and water consumption and weight gain in mice.

[The effect of alternating electric field of industrial frequency on testicles of white mice].

Theta-gamma coupling in hippocampus during working memory deficits induced by low frequency electromagnetic field exposure.

[An ultrastructural analysis of the testes in mice subjected to long-term exposure to a 17-kHz electrical field].

Evaluation of mouse embryos produced in vitro after electromagnetic waves exposure; Morphometric study.

Biological accounts emerging from some kinds of electromagnetic waves in the environment.

Effect of electromagnetic waves on sensitivity of fungi of the genus Candida to miconazole.

Effects of exposure to static magnetic field on motor skills and iron levels in plasma and brain of rats.

Morphometric and structural study of the pineal gland of the Wistar rat subjected to the pulse action of a 52 Gauss, (50 Hz) magnetic field. Evolutive analysis over 21 days.

Direct suppressive effects of weak magnetic fields (50 Hz and 16 2/3 Hz) on melatonin synthesis in the pineal gland of Djungarian hamsters (Phodopus sungorus).

Effect of a 20 kHz sawtooth magnetic field exposure on the estrous cycle in mice.

[Pathomorphological reactions of the cerebral cortex nerve elements during treatment with an alternating magnetic field].

[Effect of an electric field of industrial frequency on selected biochemical parameters in the guinea pig liver].

Alterations in the rat electrocardiogram induced by stationary magnetic fields.

Effect of ELF electric field on some on biochemistry characters in the rat serum.

Repeated application of an electric field increases BDNF in the brain, enhances spatial learning, and induces infarct tolerance.

Pulsed magnetic field from video display terminals enhances teratogenic effects of cytosine arabinoside in mice.

Fetal radiofrequency radiation exposure from 800-1900 mhz-rated cellular telephones affects neurodevelopment and behavior in mice.

Liver and spleen morphology, ceruloplasmin activity and iron content in serum of guinea pigs exposed to the magnetic field.

[Effects of pregnant exposure to electromagnetic field emitted by electric blankets on brain catecholamine and behavior in offspring mice].

Variable E-cadherin expression in a MNU-induced colon tumor model in rats which exposed with 50 Hz frequency sinusoidal magnetic field.

Low frequency electromagnetic radiation and hearing.

Pretraining exposure to physiologically patterned electromagnetic stimulation attenuates fear-conditioned analgesia.

Influence of combined DC and AC magnetic fields on rat behavior.

Radiofrequency fields and teratogenesis.

Chronic exposure of primates to 60-Hz electric and magnetic fields: II. Neurochemical effects.

[Biological effects of pulsing electromagnetic fields (PEMFs) on ICR mice].

Effects of Simulated Mobile Phone Electromagnetic Radiation on Fertilization and Embryo Development.

Effect of low frequency, low amplitude magnetic fields on the permeability of cationic liposomes entrapping carbonic anhydrase: I. Evidence for charged lipid involvement.

The influences of extremely low frequency magnetic fields on drug-induced convulsion in mouse.

Effects of pulsed magnetic field treatment of soybean seeds on calli growth, cell damage, and biochemical changes under salt stress.

What is the impact of electromagnetic waves on epileptic seizures?

Intensity threshold for 60-Hz magnetically induced behavioral changes in rats.

Neuritin reverses deficits in murine novel object associative recognition memory caused by exposure to extremely low-frequency (50 Hz) electromagnetic fields.

[Standardization of electromagnetic fields of 3-30 MHz with reference to the time factor].

[Biological effects of the action of permanent magnetic fields of various intensities].

The influence of low intensity 50 Hz electromagnetic field exposure on blood Na, K and Cl concentrations in humans.

Effects of exposure of animals to ultra-wideband pulses.

A controlled trial of daily left prefrontal cortex TMS for treating depression.

[The reaction of the systems of hormonal mediator regulation to a weak geomagnetic field against a background of ionizing radiation exposure].

--Leaf Cluster 2 (27)

Theme - Effects of electromagnetic fields on chicken embryos

Titles

Effects of sinusoidal electromagnetic fields on histopathology and structures of brains of preincubated white Leghorn chicken embryos.

Teratogenic effects of sinusoidal extremely low frequency electromagnetic fields on morphology of 24 hr chick embryos.

Effects of MR exposure at 1.5 T on early embryonic development of the chick.

Histopathological and ultrastructural studies on the effects of electromagnetic fields on the liver of preincubated white Leghorn chicken embryo.

Study of potential health effects of electromagnetic fields of telephony and Wi-Fi, using chicken embryo development as animal model.

Development of preincubated chicken eggs following exposure to 50 Hz electromagnetic fields with 1.33-7.32 mT flux densities.

Effect of electric power network frequency magnetic field on embryonic development of Ascaris suum (Nematoda).

Development of chicken embryos in a pulsed magnetic field.

Influence of continuous electromagnetic fields on the stage, weight and stature of the chick embryo.

Growth Retardation Of Chick Embryo Exposed To A Low Dose Of Electromagnetic Waves.

Effects of the ELF-MFs on the development of spleens of preincubated chicken embryos.

Effects of exposing chicken eggs to a cell phone in "call" position over the entire incubation period.

Effects of 50 Hz electromagnetic fields on the histology, apoptosis, and expression of c-Fos and beta-catenin on the livers of preincubated white Leghorn chicken embryos.

Chick embryo development can be irreversibly altered by early exposure to weak extremely-low-frequency magnetic fields.

Effects of static electromagnetic fields on chick embryo pineal gland development.

Survival Assessment of Mouse Preimplantation Embryos After Exposure to Cell Phone Radiation.

Effect of ambient levels of power-line-frequency electric fields on a developing vertebrate.

Lethal and teratogenic effects of long-term low-intensity radio frequency radiation at 428 MHz on developing chick embryo.

Biological effects of continuous exposure of embryos and young chickens to electromagnetic fields emitted by video display units.

Effects of electromagnetic fields on fecundity in the chicken.

[The influence of ultrasound and constant magnetic field on gametes, zygotes, and embryos of the sea urchin].

Developmental changes in Drosophila melanogaster following exposure to alternating electromagnetic fields.

Effect of exposure to radio frequency radiation emitted by cell phone on the developing dorsal root ganglion of chick embryo: a light microscopic study.

First cell cycles of sea urchin Paracentrotus lividus are dramatically impaired by exposure to extremely low-frequency electromagnetic field.

Assessment of the effects of electromagnetic field modification on egg-laying hens in commercial flocks as indicated by production measures.

Superimposing spatially coherent electromagnetic noise inhibits field-induced abnormalities in developing chick embryos.

Sex-linked recessive lethal test of Drosophila melanogaster after exposure to 50-Hz magnetic fields.

--Leaf Cluster 12 (38)

Theme - Impact of static and low-frequency magnetic fields on melatonin secretion

Titles

Geomagnetic activity and human melatonin metabolite excretion.

Melatonin suppression by static and extremely low frequency electromagnetic fields: relationship to the reported increased incidence of cancer.

Human melatonin during continuous magnetic field exposure.

The influence of long-term exposure of mice to randomly varied power frequency magnetic fields on their nocturnal melatonin secretion patterns.

Geomagnetic disturbances are associated with reduced nocturnal excretion of a melatonin metabolite in humans.

Melatonin metabolite levels in workers exposed to 60-Hz magnetic fields: work in substations and with 3-phase conductors.

Magnetic fields and pineal function in humans: evaluation of nocturnal acute exposure to extremely low frequency magnetic fields on serum melatonin and urinary 6-sulfatoxymelatonin circadian rhythms.

Nocturnal excretion of a urinary melatonin metabolite among electric utility workers.

Multi-night exposure to 60 Hz magnetic fields: effects on melatonin and its enzymatic metabolite.

Chronic exposure to 2.9 mT, 40 Hz magnetic field reduces melatonin concentrations in humans.

Is melatonin the hormonal missing link between magnetic field effects and human diseases?

Chronic exposure to ELF magnetic fields during night sleep with electric sheet: effects on diurnal melatonin rhythms in men.

Reduced excretion of a melatonin metabolite in workers exposed to 60 Hz magnetic fields.

Melatonin and magnetic fields.

Examination of the melatonin hypothesis in women exposed at night to EMF or bright light.

Effects of 60-Hz magnetic field exposure on nocturnal 6-sulfatoxymelatonin, estrogens, luteinizing hormone, and follicle-stimulating hormone in healthy reproductive-age women: results of a crossover trial.

Increases in geomagnetic activity are associated with increases in thyroxine levels in a single patient: implications for melatonin levels.

Rapid-onset/offset, variably scheduled 60 Hz electric and magnetic field exposure reduces nocturnal serum melatonin concentration in nonhuman primates.

Nocturnal 6-hydroxymelatonin sulfate excretion in female workers exposed to magnetic fields.

Acute exposure to 50 Hz magnetic fields with harmonics and transient components: lack of effects on nighttime hormonal secretion in men.

Age-dependent association of exposure to television screen with children's urinary melatonin excretion?

Effects of electric and magnetic fields on nocturnal melatonin concentrations in dairy cows.

Relationship between amyloid beta protein and melatonin metabolite in a study of electric utility workers.

Non-linear relation of heart rate variability during exercise recovery with local geomagnetic activity.

Graded response of heart rate variability, associated with an alteration of geomagnetic activity in a subarctic area.

Evaluation of the nocturnal levels of urinary biogenic amines in men exposed overnight to 50-Hz magnetic field.

Nocturnal exposure to intermittent 60 Hz magnetic fields alters human cardiac rhythm.

[Biological effects produced by the influence of low frequency electromagnetic fields on hormone secretion].

Circasemiannual chronomics: half-yearly biospheric changes in their own right and as a circannual waveform.

Endocrine functions in young men exposed for one night to a 50-Hz magnetic field. A circadian study of pituitary, thyroid and adrenocortical hormones.

Effects of exposure to 16.7 Hz magnetic fields on urinary 6-hydroxymelatonin sulfate excretion of Swiss railway workers.

Chronic exposure to ELF fields may induce depression.

[Dependence of acoustic-motor reaction of healthy individuals from geomagnetic activity].

Is motivation influenced by geomagnetic activity?

Is geomagnetic activity a risk factor for sudden unexplained death in epilepsies?

[Exacerbation of hypertension and disturbances of the geomagnetic field].

Magnetic storm effect on the circulation of rabbits.

Exercise testing in the evaluation of human responses to powerline frequency fields.

Fourth Level Cluster 85 (540)
Theme - Adverse impacts of low-frequency EMF, emphasizing cancer and neurodegenerative diseases

--Leaf Cluster 4 (97)

Theme - Exposure to power lines and risk of childhood cancer

Titles

Distance from residence to power line and risk of childhood leukemia: a population-based case-control study in Denmark.

Living near overhead high voltage transmission power lines as a risk factor for childhood acute lymphoblastic leukemia: a case-control study.

Residential exposure to electric power transmission lines and risk of lymphoproliferative and myeloproliferative disorders: a case-control study.

Epidemiological study of power lines and childhood cancer in the UK: further analyses.

Exposure to Electromagnetic Fields of High Voltage Overhead Power Lines and Female Infertility.

Acute childhood leukemias and exposure to magnetic fields generated by high voltage overhead power lines - a risk factor in Iran.

Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study.

Residential mobility of populations near UK power lines and implications for childhood leukaemia.

Electromagnetic fields and cancer in children residing near Norwegian high-voltage power lines.

Magnetic fields and cancer in children residing near Swedish high-voltage power lines.

Exposure of children to residential magnetic fields in Norway: is proximity to power lines an adequate predictor of exposure?

Residential exposure to magnetic fields generated by 110-400 kV power lines in Finland.

Childhood cancer and magnetic fields from high-voltage power lines in England and Wales: a case-control study.

Estimating magnetic fields of homes near transmission lines in the California Power Line Study.

Proximity to overhead power lines and childhood leukaemia: an international pooled analysis.

Childhood leukaemia and distance from power lines in California: a population-based case-control study.

Childhood leukemia risk in the California Power Line Study: Magnetic fields versus distance from power lines.

Residential distance to high-voltage power lines and risk of neurodegenerative diseases: a Danish population-based case-control study.

Residential proximity to high-voltage power lines and risk of childhood hematological malignancies.

Magnetic fields and leukemia--risk for adults living close to power lines.

Overhead electricity power lines and childhood leukemia: a registry-based, case-control study.

Preterm birth among women living within 600 meters of high voltage overhead Power Lines: a case-control study.

Are children living near high-voltage power lines at increased risk of acute lymphoblastic leukemia?

Health responses to a new high-voltage power line route: design of a quasi-experimental prospective field study in the Netherlands.

Reanalysis of risks of childhood leukaemia with distance from overhead power lines in the UK.

Adult cancers near high-voltage overhead power lines.

Methods used to estimate residential exposure to 50 Hz magnetic fields from overhead power lines in an epidemiological study in France.

Magnetic fields of high voltage power lines and risk of cancer in Finnish adults: nationwide cohort study.

Risks of leukaemia among residents close to high voltage transmission electric lines.

Childhood leukaemia close to high-voltage power lines--the Geocap study, 2002-2007.

Risk of cancer in Finnish children living close to power lines.

Exposure to magnetic fields and childhood acute lymphocytic leukemia in Sao Paulo, Brazil.

"These Power Lines Make Me Ill": A Typology of Residents' Health Responses to a New High-Voltage Power Line.

Increased risk of childhood acute lymphoblastic leukemia (ALL) by prenatal and postnatal exposure to high voltage power lines: a case control study in Isfahan, Iran.

[Childhood leukaemia in a residential area with a high-voltage power line: approach according to the Dutch Community Health Services' guideline 'Cancer Clusters'].

Magnetic fields and childhood cancer: an epidemiological investigation of the effects of high-voltage underground cables.

Epidemiologic study of residential proximity to transmission lines and childhood cancer in California: description of design, epidemiologic methods and study population.

Residence near power lines and the risk of birth defects.

Leukaemia and residence near electricity transmission equipment: a case-control study.

Adult mortality from leukemia, brain cancer, amyotrophic lateral sclerosis and magnetic fields from power lines: a case-control study in Brazil.

Residential and occupational exposure to 50 Hz magnetic fields and malignant melanoma: a population based study.

Distance to high-voltage power lines and risk of childhood leukemia--an analysis of confounding by and interaction with other potential risk factors.

Residential and occupational exposure to 50 Hz magnetic fields and hematological cancers in Norway.

Childhood cancer and residential proximity to power lines. UK Childhood Cancer Study Investigators.

[Environmental exposure to electromagnetic fields and the risk of cancer].

Residential magnetic fields, contact voltage and their relationship: the effects of distribution unbalance and residential proximity to a transmission line.

Magnetic fields, leukemia, and central nervous system tumors in Swedish adults residing near high-voltage power lines.

Residential distance from high-voltage overhead power lines and risk of Alzheimer's dementia and Parkinson's disease: a population-based case-control study in a metropolitan area of Northern Italy.

Maternal exposure to magnetic fields from high-voltage power lines and the risk of birth defects.

Magnetic fields exposure from high-voltage power lines and risk of amyotrophic lateral sclerosis in two Italian populations.

Residence near power lines and mortality from neurodegenerative diseases: longitudinal study of the Swiss population.

Symptom reporting after the introduction of a new high-voltage power line: a prospective field study.

Risk of hematological malignancies associated with magnetic fields exposure from power lines: a case-control study in two municipalities of northern Italy.

Nocebo responses to high-voltage power lines: Evidence from a prospective field study.

Role of Electromagnetic Field Exposure in Childhood Acute Lymphoblastic Leukemia and No Impact of Urinary Alpha- Amylase--a Case Control Study in Tehran, Iran.

Residence near high voltage facilities and risk of cancer in children.

[Residence close to high-tension electric power lines and its association with leukemia in children].

Relation between suicide and the electromagnetic field of overhead power lines.

Methods used to calculate exposures in two epidemiological studies of power lines in the UK.

Risk of selected birth defects by maternal residence close to power lines during pregnancy.

The effects of electric power lines on the breeding ecology of greater sage-grouse.

Birth defects and high voltage power lines: an exploratory study based on registry data.

[Electromagnetic fields from high-voltage installations and cancer in childhood].

Understanding Local Policy Elites' Perceptions on the Benefits and Risks Associated with High-Voltage Power Line Installations in the State of Arkansas.

Maternal residential proximity to sources of extremely low frequency electromagnetic fields and adverse birth outcomes in a UK cohort.

Experimental validation of a statistical model for evaluating the past or future magnetic field exposures of a population living near power lines.

Residential exposure to overhead high-voltage lines and the risk of testicular cancer: results of a population-based case-control study in Hamburg (Germany).

Childhood cancer and exposure to corona ions from power lines: an epidemiological test.

[Epidemiological studies on neurotic disturbances, anxiety and depression disorders in a population living near an overhead high voltage transmission line (400 kV)].

Magnetic fields of transmission lines and depression.

Power lines and the geomagnetic field.

The relationship between residential proximity to extremely low frequency power transmission lines and adverse birth outcomes.

Effect of Power Line Interference on Microphone Calibration Measurements Made at or Near Harmonics of the Power Line Frequency.

Maternal proximity to extremely low frequency electromagnetic fields and risk of birth defects.

Morbidity experience in populations residentially exposed to 50 hz magnetic fields: methodology and preliminary findings of a cohort study.

Case-control study on maternal residential proximity to high voltage power lines and congenital anomalies in France.

Case-only study of interactions between DNA repair genes (hMLH1, APEX1, MGMT, XRCC1 and XPD) and low-frequency electromagnetic fields in childhood acute leukemia.

Environmental justice: a contrary finding for the case of high-voltage electric power transmission lines.

Comparison of Two Methods for Judging Distances Near Overhead Power Lines.

Relative contribution of residential and occupational magnetic field exposure over twenty-four hours among people living close to and far from a power line.

Depressive symptomatology in women and residential proximity to high-voltage transmission lines.

Childhood cancer occurrence in relation to power line configurations: a study of potential selection bias in case-control studies.

Power lines, roads, and avian nest survival: effects on predator identity and predation intensity.

Symptom prevalence and worry about high voltage transmission lines.

Re-examining the association between residential exposure to magnetic fields from power lines and childhood asthma in the Danish National Birth Cohort.

Comparison of three different ways of measuring distances between residences and high voltage power lines.

Association between high voltage overhead transmission lines and mental health: a cross-sectional study.

Residential proximity to electromagnetic field sources and birth weight: Minimizing residual confounding using multiple imputation and propensity score matching.

[Health effects of electromagnetic fields].

Association between exposure to electromagnetic fields from high voltage transmission lines and neurobehavioral function in children.

Theory of oncogene activation by chemicals and antioncogene inactivation by radiations - possible carcinogenic effect of power-lines.

Radiofrequency field exposure and cancer: what do the laboratory studies suggest?

Cancer cluster among young Indian adults living near power transmission lines in Bom Jesus do Tocantins, Para, brazil.

The Origin and Role of Trust in Local Policy Elites' Perceptions of High-Voltage Power Line Installations in the State of Arkansas.

A note on the charging of aerosols by overhead line corona.

[Heliogeophysical correlates of early biodemographic variables in the south of western Siberia].

Experimental evidence of a potentially increased thrombo-embolic disease risk by domestic electromagnetic field exposure.

--Leaf Cluster 15 (131)

Theme - Residential magnetic fields and childhood leukemia

Titles

A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia-EMF Study Group.

Residential magnetic fields predicted from wiring configurations: II. Relationships To childhood leukemia.

Childhood leukemia and personal monitoring of residential exposures to electric and magnetic fields in Ontario, Canada.

Residential exposure to magnetic fields and acute lymphoblastic leukemia in children.

A case-control study of childhood leukemia in southern Ontario, Canada, and exposure to magnetic fields in residences.

Do studies of wire code and childhood leukemia point towards or away from magnetic fields as the causal agent?

Case-control study of childhood cancer and exposure to 60-Hz magnetic fields.

Power-frequency electric and magnetic fields and risk of childhood leukemia in Canada.

Exposure to residential electric and magnetic fields and risk of childhood leukemia.

Magnetic field exposure assessment in a case-control study of childhood leukemia.

Assessment of selection bias in the Canadian case-control study of residential magnetic field exposure and childhood leukemia.

Childhood leukemia and electromagnetic fields: results of a population-based case-control study in Germany.

[Infantile leukemia and exposure to 50/60 Hz magnetic fields: review of epidemiologic evidence in 2000].

Factors that explain the power line configuration wiring code-childhood leukemia association: what would they look like?

Electric and magnetic fields at power frequencies.

Residential magnetic field exposure and childhood brain cancer: a meta-analysis.

Childhood cancer in relation to a modified residential wire code.

Occupational and residential magnetic field exposure and leukemia and central nervous system tumors.

A pooled analysis of magnetic fields and childhood leukaemia.

Electrical power lines and childhood leukemia: a study from Greece.

Childhood leukemia, electric and magnetic fields, and temporal trends.

Residential mobility and childhood leukemia.

Residential wire codes: reproducibility and relation with measured magnetic fields.

Childhood leukemia: electric and magnetic fields as possible risk factors.

Childhood leukemia and magnetic fields in Japan: a case-control study of childhood leukemia and residential power-frequency magnetic fields in Japan.

Hypothesis: the risk of childhood leukemia is related to combinations of power-frequency and static magnetic fields.

Selection bias from differential residential mobility as an explanation for associations of wire codes with childhood cancer.

Do confounding or selection factors of residential wiring codes and magnetic fields distort findings of electromagnetic fields studies?

Residential magnetic fields and childhood leukemia: a meta-analysis.

Residential magnetic fields as a risk factor for childhood acute leukaemia: results from a German population-based case-control study.

The residential case-specular method to study wire codes, magnetic fields, and disease.

Wire codes, magnetic fields, and childhood cancer.

Residential EMF exposure and childhood leukemia: meta-analysis and population attributable risk.

The potential impact of bias in studies of residential exposure to magnetic fields and childhood leukemia.

Magnetic fields and childhood cancer--a pooled analysis of two Scandinavian studies.

Do magnetic fields cause increased risk of childhood leukemia via melatonin disruption?

[Electromagnetic residential fields and childhood cancers: state of epidemiological research].

[Synthesis of the epidemiological evidence concerning childhood leukemia in relation to exposure to 50 Hz. electric and magnetic fields].

Childhood leukemia and magnetic fields in infant incubators.

Electromagnetic fields and cancer risks.

[Risk of childhood leukemia and environmental exposure to ELF electromagnetic fields].

Magnetic fields and acute leukemia in children with Down syndrome.

Residential exposure to electromagnetic fields and childhood leukaemia: a meta-analysis.

Combined risk estimates for two German population-based case-control studies on residential magnetic fields and childhood acute leukemia.

Magnetic fields and acute lymphoblastic leukemia in children: a systematic review of case-control studies.

Adult and childhood leukemia near a high-power radio station in Rome, Italy.

Residential proximity to electricity transmission and distribution equipment and risk of childhood leukemia, childhood lymphoma, and childhood nervous system tumors: systematic review, evaluation, and meta-analysis.

Variation in cancer risk estimates for exposure to powerline frequency electromagnetic fields: a meta-analysis comparing EMF measurement methods.

Maternal occupational exposure to extremely low frequency magnetic fields during pregnancy and childhood leukemia.

Residential magnetic fields predicted from wiring configurations: I. Exposure model.

Estimating exposure in studies of residential magnetic fields and cancer: importance of short-term variability, time interval between diagnosis and measurement, and distance to power line.

Childhood leukemia in relation to radio frequency electromagnetic fields in the vicinity of TV and radio broadcast transmitters.

Pooled analysis of recent studies on magnetic fields and childhood leukaemia.

Occupational electric and magnetic field exposure and leukemia. A meta-analysis.

Description of a new computer wire coding method and its application to evaluate potential control selection bias in the Savitz et al. childhood cancer study.

Aetiology of childhood leukemia.

Maternal occupational exposure to electromagnetic fields before, during, and after pregnancy in relation to risks of childhood cancers: findings from the Oxford Survey of Childhood Cancers, 1953-1981 deaths.

Exposure to power-frequency magnetic fields and the risk of childhood cancer. UK Childhood Cancer Study Investigators.

Exposure to magnetic fields and survival after diagnosis of childhood leukemia: a German cohort study.

A case-control pilot study of traffic exposures and early childhood leukemia using a geographic information system.

Influence of power frequency electric and magnetic fields on human health.

Rate of occurrence of transient magnetic field events in U.S. residences.

Epidemiologic studies of electric and magnetic fields and cancer: strategies for extending knowledge.

Suggestion of concomitant changes of electric power consumption and childhood leukemia in Greece.

Association between childhood acute lymphoblastic leukemia and use of electrical appliances during pregnancy and childhood.

Nighttime exposure to electromagnetic fields and childhood leukemia: an extended pooled analysis.

The possible role of contact current in cancer risk associated with residential magnetic fields.

Leukemia in electric utility workers: the evaluation of alternative indices of exposure to 60 Hz electric and magnetic fields.

Exposure to radio-frequency electromagnetic fields from broadcast transmitters and risk of childhood cancer: a census-based cohort study.

Leukemia and lymphoma incidence in rodents exposed to low-frequency magnetic fields.

Do power frequency magnetic fields cause leukemia in children?

Viral contacts confound studies of childhood leukemia and high-voltage transmission lines.

Residential exposure to magnetic fields and risk of canine lymphoma.

[Occupational and residential exposure to electric and magnetic field and its relationship on acute myeloid leukemia in adults - A Meta-analysis].

Acute nonlymphocytic leukemia and residential exposure to power frequency magnetic fields.

Modification of the 1979 "Denver wire code" for different wire or plumbing types.

Risk of childhood leukemia in areas passed by high power lines.

Risk factors for leukemia in Thailand.

Determinants of power-frequency magnetic fields in residences located away from overhead power lines.

Risk of leukemia in children living near high-voltage transmission lines.

Los Angeles study of residential magnetic fields and childhood brain tumors.

Residential electric consumption and childhood cancer in Canada (1971-1986)

Selection bias and its implications for case-control studies: a case study of magnetic field exposure and childhood leukaemia.

Extremely low-frequency magnetic fields and childhood acute lymphoblastic leukemia: an exploratory analysis of alternative exposure metrics.

Childhood brain tumors and residential electromagnetic fields (EMF).

Power-frequency magnetic fields and childhood brain tumors: a case-control study in Japan.

Electromagnetic field exposures and childhood leukaemia in New Zealand.

Childhood cancer in relation to indicators of magnetic fields from ground current sources.

[A review of epidemiological studies on the relationship of residential electromagnetic exposure to cancer].

Investigation of the sources of residential power frequency magnetic field exposure in the UK Childhood Cancer Study.

Electric and magnetic fields and health outcomes--an overview.

Estimation of population attributable fractions from fitted incidence ratios and exposure survey data, with an application to electromagnetic fields and childhood leukemia.

The determinants of Canadian children's personal exposures to magnetic fields.

Correlation of year-to-year magnetic field exposure metrics among children in a leukemia survival study.

Contact voltage measured in residences: implications to the association between magnetic fields and childhood leukemia.

Exposure to power frequency electric fields and the risk of childhood cancer in the UK.

Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations.

Adult glioma in relation to residential power frequency electromagnetic field exposures in the San Francisco Bay area.

A precautionary public health protection strategy for the possible risk of childhood leukaemia from exposure to power frequency magnetic fields.

[Leukemia mortality and incidence of infantile leukemia near the Vatican Radio Station of Rome].

A pooled analysis of extremely low-frequency magnetic fields and childhood brain tumors.

Health effects of magnetic fields generated from power lines: new clues for an old puzzle.

Association of childhood cancer with residential traffic density.

Designs and analyses for exploring the relationship of magnetic fields to childhood leukaemia: a pilot project for the Danish National Birth Cohort.

A Bayesian approach to hazard identification. The case of electromagnetic fields and cancer.

[Risk of neoplastic diseases in conditions of exposure to power magnetic fields--epidemiologic investigations].

Potential motion related bias in the worn dosimeter measurements of two childhood leukemia studies.

Early pregnancy loss and exposure to 50-Hz magnetic fields.

Extra low frequency electric and magnetic fields in the bedplace of children diagnosed with leukaemia: a case-control study.

Environmental factors and childhood acute leukemias and lymphomas.

Are the stray 60-Hz electromagnetic fields associated with the distribution and use of electric power a significant cause of cancer?

An alternate hypothesis for the association between electrical wiring configurations and cancer.

Magnetic field exposure and long-term survival among children with leukaemia.

Decreased survival for childhood leukemia in proximity to television towers.

Exposure to electromagnetic fields and risk of leukemia.

An evaluation of exposure metrics in an epidemiologic study on radio and television broadcast transmitters and the risk of childhood leukemia.

[Meta-analysis and its application in epidemiology].

Does our electricity distribution system pose a serious risk to public health?

Magnetic fields and leukaemia risks in UK electricity supply workers.

Childhood incidence of acute lymphoblastic leukaemia and exposure to broadcast radiation in Sydney--a second look.

50-Hz electromagnetic environment and the incidence of childhood tumors in Stockholm County.

Assessment of non-response bias in a survey of residential magnetic field exposure in Taiwan.

The relative merits of contemporary measurements and historical calculated fields in the Swedish childhood cancer study.

A population-based case-control study of radiofrequency exposure in relation to childhood neoplasm.

A unified approach to the analysis of case-distribution (case-only) studies.

A richer conceptualization of "exposure" for epidemiological studies of the "EMF mixture".

High incidence of acute leukemia in the proximity of some industrial facilities in El Bierzo, northwestern Spain.

Exposure measurement errors, risk estimate and statistical power in case-control studies using dichotomous analysis of a continuous exposure variable.

Deaths from electricity.

RF personal exposimetry on employees of elementary schools, kindergartens and day nurseries as a proxy for child exposures.

Attributable fractions: bias from broad definition of exposure.

--Leaf Cluster 13 (113)

Theme - Electromagnetic fields and cancer, especially breast cancer

Titles

Breast cancer and electromagnetic fields--a review.

Follow-up of radio and telegraph operators with exposure to electromagnetic fields and risk of breast cancer.

Electric power, pineal function, and the risk of breast cancer.

Electric blanket use and breast cancer in the Nurses' Health Study.

Electric blanket or mattress cover use and breast cancer incidence in women 50-79 years of age.

Electric blanket use and breast cancer risk among younger women.

Electric blanket use and breast cancer on Long Island.

Risk of premenopausal breast cancer and use of electric blankets.

Occupational exposure to electromagnetic field and breast cancer risk in a large, population-based, case-control study in the United States.

The relationship between electromagnetic field and light exposures to melatonin and breast cancer risk: a review of the relevant literature.

Residential and occupational exposures to 50-Hz magnetic fields and breast cancer in women: a population-based study.

Use of electric blankets and risk of postmenopausal breast cancer.

Electromagnetic fields and male breast cancer.

Environmental factors and breast cancer.

Electromagnetic fields and female breast cancer.

Environmental risk factors and female breast cancer.

Occupational and residential magnetic field exposure and breast cancer in females.

Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca2+ signaling molecular pathways in breast cancer.

Occupational exposures to extremely low frequency magnetic fields and postmenopausal breast cancer.

Population-based case-control study of occupational exposure to electromagnetic fields and breast cancer.

Exposure to electromagnetic fields from use of electric blankets and other in-home electrical appliances and breast cancer risk.

Residential magnetic field exposure and breast cancer risk: a nested case-control study from a multiethnic cohort in Los Angeles County, California.

Evaluation of potential confounders in planning a study of occupational magnetic field exposure and female breast cancer.

Risk for leukaemia and brain and breast cancer among Danish utility workers: a second follow-up.

Breast cancer and electric power.

Residential exposure to 60-Hertz magnetic fields and adult cancers in Taiwan.

Occupational exposure to magnetic fields in relation to male breast cancer and testicular cancer: a Swedish case-control study.

The melatonin hypothesis: electric power and breast cancer.

Incidence of breast cancer in a Norwegian cohort of women with potential workplace exposure to 50 Hz magnetic fields.

Magnetic fields and breast cancer in Swedish adults residing near high-voltage power lines.

Electromagnetic fields and breast cancer on Long Island: a case-control study.

Occupational magnetic fields and female breast cancer: a case-control study using Swedish population registers and new exposure data.

A meta-analysis of epidemiologic studies of electric and magnetic fields and breast cancer in women and men.

Residential magnetic fields and the risk of breast cancer.

A cluster of male breast cancer in office workers.

Induction of tamoxifen resistance in breast cancer cells by ELF electromagnetic fields.

Electromagnetic field exposure and male breast cancer risk: a meta-analysis of 18 studies.

Relationship between exposure to extremely low-frequency electromagnetic fields and breast cancer risk: a meta-analysis.

Shift work, light at night, and breast cancer on Long Island, New York.

Extremely low-frequency electromagnetic fields exposure and female breast cancer risk: a meta-analysis based on 24,338 cases and 60,628 controls.

Use of electric bedding devices and risk of breast cancer in African-American women.

Epidemiological appraisal of studies of residential exposure to power frequency magnetic fields and adult cancers.

Magnetic fields and mammary cancer in rodents: a critical review and evaluation of published literature.

Breast cancer, occupation, and exposure to electromagnetic fields among Swedish men.

[Risk of cancer among Danish electricity workers. A cohort study].

Meta-analysis of extremely low frequency electromagnetic fields and cancer risk: a pooled analysis of epidemiologic studies.

Occupational magnetic field exposure and site-specific cancer incidence: a Swedish cohort study.

Risk of cancer among Danish utility workers--a nationwide cohort study.

Occupational exposures associated with male breast cancer.

Incidence of cancer in persons with occupational exposure to electromagnetic fields in Denmark.

Cancer incidence in California flight attendants (United States).

Overview of epidemiologic research on electric and magnetic fields and cancer.

Exposure to extremely low frequency magnetic fields among working women and homemakers.

Endometrial cancer incidence in relation to electric blanket use.

[Carcinogenic risk of extremely-low-frequency electromagnetic fields: state of the art].

Increased incidence of cancer in a cohort of office workers exposed to strong magnetic fields.

Socioeconomic status, social mobility and cancer occurrence during working life: a case-control study among French electricity and gas workers.

Extremely low frequency electromagnetic fields (EMF) and brain cancer in adults and children: review and comment.

Incidence of cancer in the vicinity of Korean AM radio transmitters.

Cancer incidence and magnetic field exposure in industries using resistance welding in Sweden.

Use of electric blankets and risk of testicular cancer.

Use of electric blankets and association with prevalence of endometrial cancer.

Personal radio use and cancer risks among 48,518 British police officers and staff from the Airwave Health Monitoring Study.

[Use of cellular telephones and risk of cancer. A Danish cohort study].

Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter.

Cancer incidence among Norwegian airline pilots.

Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer.

Biologically based epidemiological studies of electric power and cancer.

Cancer incidence vs. FM radio transmitter density.

A new electromagnetic exposure metric: high frequency voltage transients associated with increased cancer incidence in teachers in a California school.

Primary brain cancer in adults and the use of common household appliances: a case-control study.

Extremely low frequency electromagnetic fields and cancer: the epidemiologic evidence.

Cancer incidence near radio and television transmitters in Great Britain. II. All high power transmitters.

Epidemiological studies of radio frequency exposures and human cancer.

Cancer mortality and residence near electricity transmission equipment: a retrospective cohort study.

Effects of 50- or 60-hertz, 100 microT magnetic field exposure in the DMBA mammary cancer model in Sprague-Dawley rats: possible explanations for different results from two laboratories.

Brain tumor risk in children in relation to use of electric blankets and water bed heaters. Results from the United States West Coast Childhood Brain Tumor Study.

Electromagnetic fields: a cancer promoter?

Prostate cancer in relation to the use of electric blanket or heated water bed.

Cancer in radar technicians exposed to radiofrequency/microwave radiation: sentinel episodes.

Brain cancer risk and electromagnetic fields (EMFs): assessing the geomagnetic component.

Incidence of Seminoma Cancer in Staffs that Worked in Electromagnetic Waves Station; Three Cases Report.

Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields.

Human cancer from environmental pollutants: the epidemiological evidence.

Electric blanket use during pregnancy in relation to the risk of congenital urinary tract anomalies among women with a history of subfertility.

Magnetic field exposure related to cancer subtypes.

Increasing incidence of thyroid cancer in the Nordic countries with main focus on Swedish data.

[Electromagnetic fields: is there any probability of the risk of cancer?].

Electric Blanket Use and Risk of Thyroid Cancer in the Women's Health Initiative Observational Cohort.

Epidemiology and aetiological factors of male breast cancer: a ten years retrospective study in eastern Turkey.

Radio frequency radiation-related cancer: assessing causation in the occupational/military setting.

Spontaneous abortion and exposure to electric blankets and heated water beds.

[Environment and cancer risk].

[Geomagnetic field variation in early ontogenesis as a risk factor for oncopathology].

Cancer in the electric power industry.

The use of electric bed heaters and the risk of clinically recognized spontaneous abortion.

The role of household electromagnetic fields in the development of mammary tumors in women: clinical case-record observations.

[Evaluation of genotoxic and/or co-genotoxic effects in cells exposed in vitro to extremely-low frequency electromagnetic fields].

Trends in incidence of primary brain cancer in New Zealand, 1995 to 2010.

Myelogenous leukemia and electric blanket use.

Panel exploring pro and con arguments as to whether EMFs cause childhood brain cancer.

[Recent data from the literature on the biological and pathologic effects of electromagnetic radiation, radio waves and stray currents].

Chronic toxicity/oncogenicity evaluation of 60 Hz (power frequency) magnetic fields in B6C3F1 mice.

Biological effects of power-frequency fields as they relate to carcinogenesis.

[Age diseases depending on geomagnetic field activity inside the womb period].

[Enhancement of efficacy of neoadjuvant polychemotherapy in combined treatment of lung cancer].

Cancer versus FM radio polarization types.

Genetic damage in humans exposed to extremely low-frequency electromagnetic fields.

[Male breast tumors in railway engine drivers: investigation of 5 cases].

Exposure to electromagnetic fields during pregnancy with emphasis on electrically heated beds: association with birthweight and intrauterine growth retardation.

Melanoma incidence and frequency modulation (FM) broadcasting.

Malignant melanoma of the skin - not a sunshine story!

Multimodal treatment of hepatocellular carcinoma.

--Leaf Cluster 18 (62)

Theme - Mortality studies of electrical utility workers, focusing on electromagnetic field exposures

Titles

Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers.

A mortality study of electrical utility workers in Quebec.

Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers.

[Cancer mortality among electricity utility workers in a the state of Sao Paulo, Brazil].

Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems.

Magnetic field exposure and neurodegenerative disease mortality among electric utility workers.

Exposure to electromagnetic fields and suicide among electric utility workers: a nested case-control study.

[Preliminary study of cause-specific mortality of a population exposed to 50 Hz magnetic fields, in a district of Rome municipality].

Exposure to electromagnetic fields and suicide among electric utility workers: a nested case-control study.

Mortality from brain cancer and leukaemia among electrical workers.

Mortality in workers exposed to electromagnetic fields.

Comparative analyses of the studies of magnetic fields and cancer in electric utility workers: studies from France, Canada, and the United States.

Leukemia following occupational exposure to 60-Hz electric and magnetic fields among Ontario electric utility workers.

Mortality from amyotrophic lateral sclerosis, other chronic disorders, and electric shocks among utility workers.

Electromagnetic fields and health effects--epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease.

Mortality of workers exposed to ionizing radiation at the French National Electricity Company.

Association between exposure to pulsed electromagnetic fields and cancer in electric utility workers in Quebec, Canada, and France.

[Mortality of personnel operating electric power objects with 500 kV voltage].

Electromagnetic fields, polychlorinated biphenyls, and prostate cancer mortality in electric utility workers.

Mortality of plastic-ware workers exposed to radiofrequencies.

Mortality among workers in the geothermal power plants at Larderello, Italy.

A population-based cohort study of occupational exposure to magnetic fields and cardiovascular disease mortality.

Exposure to 50-Hz electric field and incidence of leukemia, brain tumors, and other cancers among French electric utility workers.

Cancer in Korean war navy technicians: mortality survey after 40 years.

[Occupational exposure to electromagnetic fields and its health effects in electric energy workers].

Leukemia, brain tumors, and exposure to extremely low frequency electromagnetic fields in Swiss railway employees.

Electric and magnetic field exposure and brain cancer: a review.

[Remote effects of occupational and non-occupational exposure to electromagnetic fields of power-line frequency. Epidemiological studies].

Occupational exposures and brain cancer mortality: a preliminary study of east Texas residents.

[Mortality of people residing near electric power supply line with voltage of 500 kV].

Cancer incidence and mortality and proximity to TV towers.

Cardiovascular mortality and exposure to extremely low frequency magnetic fields: a cohort study of Swiss railway workers.

Mortality of persons resident in the vicinity of electricity transmission facilities.

A case cohort study of suicide in relation to exposure to electric and magnetic fields among electrical utility workers.

Mortality from neurodegenerative disease and exposure to extremely low-frequency magnetic fields: 31 years of observations on Swiss railway employees.

Refinements in magnetic field exposure assignment for a case-cohort study of electrical utility workers.

Leukaemia, brain tumours and exposure to extremely low frequency magnetic fields: cohort study of Swiss railway employees.

Magnetic field exposure and cardiovascular disease mortality among electric utility workers.

[Mortality indices for hemoblastoses in Rivno Province before and after the accident at the Chernobyl Atomic Electric Power Station].

Incidence of cancer among workers in Norwegian hydroelectric power companies.

Fatal occupational injuries among electric power company workers.

Exposure to electromagnetic fields and the risk of leukemia.

Biological effects on human health due to radiofrequency/microwave exposure: a synopsis of cohort studies.

Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies.

Risk of severe cardiac arrhythmia in male utility workers: a nationwide danish cohort study.

Occupational magnetic field exposure, cardiovascular disease mortality, and potential confounding by smoking.

Invited commentary: electromagnetic fields and cancer in railway workers.

Multiple sclerosis among utility workers.

Annals of conflicting results: looking back on electromagnetic field research.

Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea.

Feasibility of a cohort study on health risks caused by occupational exposure to radiofrequency electromagnetic fields.

[An epidemiological study of cancer morbidity and mortality among the population living in areas close to thermal and atomic electric power stations].

Practical limitations of epidemiologic methods.

Causes of death among Belgian professional military radar operators: a 37-year retrospective cohort study.

Effects upon health of occupational exposure to microwave radiation (radar).

Leukemia in telephone linemen.

Cancer incidence among welders: possible effects of exposure to extremely low frequency electromagnetic radiation (ELF) and to welding fumes.

Uncertainty in the relation between exposure to magnetic fields and brain cancer due to assessment and assignment of exposure and analytical methods in dose-response modeling.

Accidental deaths caused by electricity in Sweden, 1975-2000.

Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation.

Pregnancy outcomes after paternal radiofrequency field exposure aboard fast patrol boats.

Home and leisure injuries among the French electricity and gas company active employees: circumstances and short-term consequences.

--Leaf Cluster 27 (137)

Theme - Occupational exposure to electromagnetic fields, emphasizing neurodegenerative disease and cancer

Titles

Occupational exposures and the risk of amyotrophic lateral sclerosis.

Dementia and occupational exposure to magnetic fields.

Occupational exposure to magnetic fields in case-referent studies of neurodegenerative diseases.

Amyotrophic lateral sclerosis and occupational exposure to electromagnetic fields.

Occupational Exposures and Neurodegenerative Diseases-A Systematic Literature Review and Meta-Analyses.

Occupational magnetic field exposure and neurodegenerative disease.

Association between extremely low-frequency electromagnetic fields occupations and amyotrophic lateral sclerosis: a meta-analysis.

Paternal occupational exposure to electro-magnetic fields as a risk factor for cancer in children and young adults: a case-control study from the North of England.

Occupational exposure and amyotrophic lateral sclerosis in a prospective cohort.

Neurodegenerative diseases in welders and other workers exposed to high levels of magnetic fields.

Parental occupational exposure to magnetic fields and childhood cancer (Sweden).

Association between occupational exposure to power frequency electromagnetic fields and amyotrophic lateral sclerosis: a review.

Magnetic field exposure and neurodegenerative diseases--recent epidemiological studies.

Occupational and residential exposure to electromagnetic fields and risk of brain tumors in adults: a case-control study in Gironde, France.

Electrical occupations and neurodegenerative disease: analysis of U.S. mortality data.

Occupational exposure to magnetic fields and brain tumours in central Sweden.

Occupational exposure to extremely low frequency electric and magnetic fields and Alzheimer disease: a meta-analysis.

Amyotrophic Lateral Sclerosis and Occupational Exposures: A Systematic Literature Review and Meta-Analyses.

Case-Control Study on Occupational Exposure to Extremely Low-Frequency Electromagnetic Fields and the Association with Meningioma.

Occupational exposure to extremely low frequency magnetic fields and risk of Alzheimer disease: A systematic review and meta-analysis.

Occupational exposure to electromagnetic fields and Alzheimer disease.

Risk of childhood acute lymphoblastic leukaemia following parental occupational exposure to extremely low frequency electromagnetic fields.

Brain cancer and occupational exposure to magnetic fields among men: results from a Canadian population-based case-control study.

Occupational exposure to high-frequency electromagnetic fields and brain tumor risk in the INTEROCC study: An individualized assessment approach.

Occupational exposure to power frequency magnetic fields and risk of non-Hodgkin lymphoma.

Parental occupational exposure to extremely low frequency magnetic fields and childhood cancer: a German case-control study.

[News in occupational cancers].

Exposure to magnetic fields among electrical workers in relation to leukemia risk in Los Angeles County.

Occupational exposure to magnetic fields and the risk of brain tumors.

Occupational electric and magnetic field exposure and brain cancer: a meta-analysis.

Risk factors for Alzheimer disease: a population-based case-control study in Istanbul, Turkey.

Acute leukemia in electrical workers: a New Zealand case-control study.

Brain tumor risk in offspring of men occupationally exposed to electric and magnetic fields.

Work related etiology of amyotrophic lateral sclerosis (ALS): a meta-analysis.

Occupational exposure to low frequency magnetic fields and the risk of low grade and high grade glioma.

Occupational magnetic field exposure among women in Stockholm County, Sweden.

Occupational risk factors for cancer of the central nervous system: a case-control study on death certificates from 24 U.S. states.

Are occupational, hobby, or lifestyle exposures associated with Philadelphia chromosome positive chronic myeloid leukaemia?

Berkson error adjustment and other exposure surrogates in occupational case-control studies, with application to the Canadian INTEROCC study.

Paternal occupational exposure to radiofrequency electromagnetic fields and risk of adverse pregnancy outcome.

Electromagnetic field exposures and childhood cancers in New Zealand.

Occupational exposure to electromagnetic fields and acute leukaemia: analysis of a case-control study.

Maternal occupational exposure to extremely low frequency magnetic fields and the risk of brain cancer in the offspring.

Elevated risk of Alzheimer's disease among workers with likely electromagnetic field exposure.

[Epidemiological risk assessment of pathology development in occupational exposure to radiofrequency electromagnetic fields].

Review of the epidemiologic literature on EMF and Health.

Occupational risk factors in Alzheimer's disease: a review assessing the quality of published epidemiological studies.

Exposure to extremely low frequency electromagnetic fields and the risk of malignant diseases--an evaluation of epidemiological and experimental findings.

Interactions between occupational exposure to extremely low frequency magnetic fields and chemicals for brain tumour risk in the INTEROCC study.

The effect of male occupational exposure in infertile couples in Norway.

[Amyotrophic lateral sclerosis and exposure to metals and other occupational/environmental hazardous materials: state of the art].

Occupational exposure to radio frequency/microwave radiation and the risk of brain tumors: Interphone Study Group, Germany.

Relationships between occupational history and serum concentrations of organochlorine compounds in exocrine pancreatic cancer.

Risk agents related to work and amyotrophic lateral sclerosis: An occupational medicine focus.

[A case-control study on the risk factors of Alzheimer's disease in military elderly men].

Occupational exposures obtained by questionnaire in clinical practice and their association with semen quality.

Leukemia and occupational exposure to electromagnetic fields: review of epidemiologic surveys.

Occupational exposure to ionizing and non-ionizing radiation and risk of non-Hodgkin lymphoma.

Case-control study on occupational exposure to extremely low-frequency electromagnetic fields and glioma risk.

Risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields: a population based study.

Environmental risk factors for non-Hodgkin's lymphoma: a population-based case-control study in Languedoc-Roussillon, France.

Exposure to electromagnetic fields and risk of central nervous system disease in utility workers.

[Exposure to electromagnetic fields and risk of central nervous system diseases among employees at Danish electric companies].

Neurodegenerative diseases, suicide and depressive symptoms in relation to EMF.

Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer's disease.

Occupational risk factors for lung cancer in the French electricity and gas industry: a case-control survey nested in a cohort of active employees.

Parental occupational exposures to electromagnetic fields and radiation and the incidence of neuroblastoma in offspring.

Occupational exposure to ionizing radiation and electromagnetic fields in relation to the risk of thyroid cancer in Sweden.

[Parental occupational exposures and autism spectrum disorder in children].

Acute leukaemia in workers exposed to electromagnetic fields.

Parental heat exposure and risk of childhood brain tumor: a Children's Oncology Group study.

Occupational magnetic field exposure and the risk of acoustic neuroma.

Leukemia risk and occupational electric field exposure in Los Angeles County, California.

Neurodegenerative disease and magnetic field exposure in UK electricity supply workers.

Occupational exposure to low frequency magnetic fields and dementia: a case-control study.

Occupation and malignant lymphoma: a population based case control study in Germany.

A nested case-control study of residential and personal magnetic field measures and miscarriages.

Need for a European approach to the effects of extremely low-frequency electromagnetic fields on cancer. ELF-EMF European Feasibility Study Group.

Incidence of leukaemia and brain tumours in some "electrical occupations".

A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of miscarriage.

Risk of cognitive impairment in relation to elevated exposure to electromagnetic fields.

Occupational factors of anxiety and depressive disorders in the French National Electricity and Gas Company. The Anxiety-Depression Group.

Occupational electromagnetic field exposures associated with sleep quality: a cross-sectional study.

Testicular cancer and electromagnetic fields (EMF) in the workplace: results of a population-based case-control study in Germany.

Occupational exposure to electromagnetic fields and the occurrence of brain tumors. An analysis of possible associations.

Occupational magnetic field exposure and myocardial infarction incidence.

Magnetic fields and brain tumour risks in UK electricity supply workers.

[The potential hazard for the development of leukemia from exposure to electromagnetic radiation (a review of the literature)].

Neuroblastoma and paternal occupation. A case-control analysis.

Self-reported electrical appliance use and risk of adult brain tumors.

Non-specific physical symptoms and electromagnetic field exposure in the general population: can we get more specific? A systematic review.

Occupational risk factors for acute leukaemia: a case-control study.

Occupational hazards for the male reproductive system.

Amyotrophic lateral sclerosis and environmental factors.

Environmental risk factors for brain tumors.

Occupational exposure to electromagnetic fields and sex-differential risk of uveal melanoma.

Maternal cumulative exposure to extremely low frequency electromagnetic fields and pregnancy outcomes in the Elfe cohort.

[Non dietetic environmental risk factors in prostate cancer].

[Environmental risk factors and epidemiologic study].

[Paternal exposure to occupational electromagnetic radiation and sex ratio of the offspring: a meta-analysis].

[Occupational risk and its prophylaxis for female workers engaged in radio-electronic instrument industry].

Interactive effect of chemical substances and occupational electromagnetic field exposure on the risk of gliomas and meningiomas in Swedish men.

[Delayed biological effect of electromagnetic fields action].

Prevalence of musculoskeletal disorders and related occupational causative factors among electricity linemen: A narrative review.

Paternal work in the power industry: effects on children at delivery.

Miscarriages among female physical therapists who report using radio- and microwave-frequency electromagnetic radiation.

Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: a nested case-control study.

Risk factors, health risks, and risk management for aircraft personnel and frequent flyers.

Video display terminal use during pregnancy and reproductive outcome--a meta-analysis.

[Difficulties of expert testimony in microwave disease].

Radiofrequency electromagnetic fields; male infertility and sex ratio of offspring.

Exposure to magnetic fields and the risk of poor sperm quality.

Myeloid leukemias and myelodysplastic syndromes: chemical exposure, histologic subtype and cytogenetics in a case-control study.

[Evaluation of occupational risk caused by exposure to electromagnetic rays].

An apparently incongruous exposure-response relationship resulting from the use of job description to assess magnetic field exposure.

Carcinogenicity test in B6C3F1 mice after parental and prenatal exposure to 50 Hz magnetic fields.

Congenital malformations and exposure to high-frequency electromagnetic radiation among Danish physiotherapists.

[The IARC carcinogenicity evaluation of radio-frequency electromagnetic field: with special reference to epidemiology of mobile phone use and brain tumor risk].

Limitations of interview-based risk assessment of RF exposure from appliances.

Case-control study on uveal melanoma (RIFA): rational and design.

Gender-specific reproductive outcome and exposure to high-frequency electromagnetic radiation among physiotherapists.

Search for teratogenic risks with the aid of malformation registries.

[Effect of early pregnancy electromagnetic field exposure on embryo growth ceasing].

Development and evaluation of a tool for retrospective exposure assessment of selected endocrine disrupting chemicals and EMF in the car manufacturing industry.

Environmental risk factors in the history of male patients of an infertility clinic.

Environmental exposure assessment in European birth cohorts: results from the ENRIECO project.

Maternal exposure to magnetic fields during pregnancy in relation to the risk of asthma in offspring.

Life styles, anxiety, expertise: the perception of risk from electromagnetic fields.

Epidemiologic evidence relevant to radar (microwave) effects.

Exposure to electromagnetic fields during pregnancy.

A literature review of medical side effects from radio-frequency energy in the human environment: involving cancer, tumors, and problems of the central nervous system.

Does exposure to computers affect the routine parameters of semen quality?

Gender ratio of offspring and exposure to shortwave radiation among female physiotherapists.

Clinical teratology.

Electricity and bones.

[A historic case of Wegener's granulomatosis: the physicist who discovered the electromagnetic waves: Heinrich Hertz].

A possible association between fetal/neonatal exposure to radiofrequency electromagnetic radiation and the increased incidence of autism spectrum disorders (ASD).

Fourth Level Cluster 83 (668)
Theme - Adverse effects of mobile phone use, especially brain tumors, and brain and neural function

--Leaf Cluster 30 (321)

Theme - Adverse health symptoms from mobile phone use

Titles

The risk of subjective symptoms in mobile phone users in Poland--an epidemiological study.

Problematic Use of Mobile Phones in Australia...Is It Getting Worse?

Use of mobile phones and cancer risk.

Health hazards of mobile phones: an Indian perspective.

Nomophobia: A Cross-sectional Study to Assess Mobile Phone Usage Among Dental Students.

Evidence-based policy? The use of mobile phones in hospital.

A survey study of the association between mobile phone use and daytime sleepiness in California high school students.

Electroencephalographic, personality, and executive function measures associated with frequent mobile phone use.

[Radiation from mobile phone and the health].

Is human saliva an indicator of the adverse health effects of using mobile phones?

Use of mobile phones by medical staff at Queen Elizabeth Hospital, Barbados: evidence for both benefit and harm.

Mobile phone induced sensorineural hearing loss.

[Mobile phones radiate--risk to the health?].

An international prospective cohort study of mobile phone users and health (COSMOS): Factors affecting validity of self-reported mobile phone use.

Mobile phone use and location of glioma: a case-case analysis.

Exposure to mobile phone electromagnetic fields and subjective symptoms: a double-blind study.

Adverse effects of excessive mobile phone use.

Mobile phone radiation causes brain tumors and should be classified as a probable human carcinogen (2A) (review).

Are mobile phones harmful?

[Subjective symptoms related to mobile phone use--a pilot study].

Mobile phone radiation and the risk of cancer; a review.

Review on health effects related to mobile phones. Part II: results and conclusions.

Effects of thirty minutes mobile phone use on the human sensory cortex.

Significance of micronuclei in buccal smears of mobile phone users: A comparative study.

[Psychophysiological indicators for children using mobile phones. Communication 1. Current state of the problem].

Audiologic disturbances in long-term mobile phone users.

Does chronic exposure to mobile phones affect cognition?

Mobile phones: influence on auditory and vestibular systems.

Cellular phones: are they detrimental?

Analysis of mobile phone use among young patients with brain tumors in Japan.

Association of mobile phone radiation with fatigue, headache, dizziness, tension and sleep disturbance in Saudi population.

Association between vestibular schwannomas and mobile phone use.

Acute effects of 3G mobile phone radiations on frontal haemodynamics during a cognitive task in teenagers and possible protective value of Om chanting.

Ethical considerations of mobile phone use by patients in KwaZulu-Natal: Obstacles for mHealth?

The use of cell phone and insight into its potential human health impacts.

Mobile phones, in combination with a computer locator system, improve the response times of emergency medical services in central London.

Mobile phone use, school electromagnetic field levels and related symptoms: a cross-sectional survey among 2150 high school students in Izmir.

Mobile phone related-hazards and subjective hearing and vision symptoms in the Saudi population.

Thermal effects of mobile phones on human auricle region.

Mobile phones and health: a literature overview.

Radiation from mobile phone systems: Is it perceived as a threat to people's health?

Child and Adolescent Use of Mobile Phones: An Unparalleled Complex Developmental Phenomenon.

Micronucleus frequency in buccal mucosa cells of mobile phone users.

Exposure of magnetic bacteria to simulated mobile phone-type RF radiation has no impact on mortality.

The assessment of electromagnetic field radiation exposure for mobile phone users.

Mobile phones: Reservoirs for the transmission of nosocomial pathogens.

Headache, tinnitus and hearing loss in the international Cohort Study of Mobile Phone Use and Health (COSMOS) in Sweden and Finland.

Analysis of ear side of mobile phone use in the general population of Japan.

Questionnaire-based evaluation of mobile phone interference with medical-electrical equipment in Swedish hospitals.

Mobile Phone-Use Habits Among Adolescents: Predictors of Intensive Use.

[Effect of stress and intesity of mobile phone using on the health and subjective symptoms in GSM workers].

Epidemiological risk assessment of mobile phones and cancer: where can we improve?

Impact of one's own mobile phone in stand-by mode on personal radiofrequency electromagnetic field exposure.

Mobile phones, radiofrequency fields, and health effects in children--epidemiological studies.

Self-reported mobile phone use and semen parameters among men from a fertility clinic.

Recall of mobile phone usage and laterality in young people: The multinational Mobi-Expo study.

Psychological factors associated with self-reported sensitivity to mobile phones.

Real versus Simulated Mobile Phone Exposures in Experimental Studies.

Time trends (1998-2007) in brain cancer incidence rates in relation to mobile phone use in England.

Neurological changes induced by a mobile phone.

Survey of mobile phone use and their chronic effects on the hearing of a student population.

Mobile phone use and stress, sleep disturbances, and symptoms of depression among young adults--a prospective cohort study.

[SAR values of mobile phones. Safety evaluation and risk perception].

Assessment of extremely low frequency magnetic field exposure from GSM mobile phones.

EEG Changes Due to Experimentally Induced 3G Mobile Phone Radiation.

Effects of thirty-minute mobile phone exposure on saccades.

Cell phone radiation exposure on brain and associated biological systems.

Effects of chronic exposure of electromagnetic fields from mobile phones on hearing in rats.

Do mobile 'phones have a detrimental impact on auditory function?

[Hearing level and intensive use of mobile phones].

Preliminary report: symptoms associated with mobile phone use.

Validation of exposure assessment and assessment of recruitment methods for a prospective cohort study of mobile phone users (COSMOS) in Finland: a pilot study.

Studying the effects of mobile phone use on the auditory system and the central nervous system: a review of the literature and future directions.

Mobile-phone pulse triggers evoked potentials.

Association between General Health and Mobile Phone Dependency among Medical University Students: A Cross-sectional Study in Iran.

Mobile phones and children: is precaution warranted?

[Determining health policy for sensible mobile phone use--current world status].

Comparison of cytotoxic and genotoxic effects of plutonium-239 alpha particles and mobile phone GSM 900 radiation in the Allium cepa test.

Mobile phones: time to rethink and limit usage.

Bedtime mobile phone use and sleep in adults.

Mobile phones and seizures: drug-resistant epilepsy is less common in mobile-phone-using patients.

Estimation of head tissue-specific exposure from mobile phones based on measurements in the homogeneous SAM head.

Mobile phones, heat shock proteins and cancer.

Can evidence change belief? Reported mobile phone sensitivity following individual feedback of an inability to discriminate active from sham signals.

Distribution of RF energy emitted by mobile phones in anatomical structures of the brain.

Association between mobile phone use and depressed mood in Japanese adolescents: a cross-sectional study.

Effects of thirty-minute mobile phone use on visuo-motor reaction time.

The controversy about a possible relationship between mobile phone use and cancer.

Association between Excessive Use of Mobile Phone and Insomnia and Depression among Japanese Adolescents.

Electromagnetic interference of GSM mobile phones with the implantable deep brain stimulator, ITREL-III.

Association between overuse of mobile phones on quality of sleep and general health among occupational health and safety students.

Effect of mobile phones on micronucleus frequency in human exfoliated oral mucosal cells.

A study on the effect of prolonged mobile phone use on pure tone audiometry thresholds of medical students of Sikkim.

Factors that influence the radiofrequency power output of GSM mobile phones.

Analysis of mobile phone design features affecting radiofrequency power absorbed in a human head phantom.

Does the Brain Detect 3G Mobile Phone Radiation Peaks? An Explorative In-Depth Analysis of an Experimental Study.

Effects of mobile phone use on brain tissue from the rat and a possible protective role of vitamin C - a preliminary study.

Effects of a 902 MHz mobile phone on cerebral blood flow in humans: a PET study.

Analysis of the influence of handset phone position on RF exposure of brain tissue.

Are thyroid dysfunctions related to stress or microwave exposure (900 MHz)?

Exposure to mobile phone electromagnetic field radiation, ringtone and vibration affects anxiety-like behaviour and oxidative stress biomarkers in albino wistar rats.

Clear policies on mobile phones vital.

Personal exposure to mobile phone frequencies and well-being in adults: a cross-sectional study based on dosimetry.

Self-report of physical symptoms associated with using mobile phones and other electrical devices.

Mobile phones as mediators of health behavior change in cardiovascular disease in developing countries.

Experimental and numerical assessment of low-frequency current distributions from UMTS and GSM mobile phones.

Are men talking their reproductive health away?

The effect of mobile phone to audiologic system.

The effects of 884 MHz GSM wireless communication signals on headache and other symptoms: an experimental provocation study.

Associations between problematic mobile phone use and psychological parameters in young adults.

Effects on auditory function of chronic exposure to electromagnetic fields from mobile phones.

Mobile phones are good for you, p0.36! Observations on Keetley, Wood, Spong and Stough (2006).

Cordless telephone use: implications for mobile phone research.

Mobile phone exposure and spatial memory.

Prevalence of subjective poor health symptoms associated with exposure to electromagnetic fields among university students.

Mobile phone headache: a double blind, sham-controlled provocation study.

Human short-term exposure to electromagnetic fields emitted by mobile phones decreases computer-assisted visual reaction time.

Evaluation of the effects of mobile phones on the neural tube development of chick embryos.

Mobile telephone use is associated with changes in cognitive function in young adolescents.

Mobile phone use, blood lead levels, and attention deficit hyperactivity symptoms in children: a longitudinal study.

Is mobile phone radiation genotoxic? An analysis of micronucleus frequency in exfoliated buccal cells.

Mobile Phone, Computer, and Internet Use Among Older Homeless Adults: Results from the HOPE HOME Cohort Study.

Mobile phones carry the personal microbiome of their owners.

Mobile phones and sex work in South India: the emerging role of mobile phones in condom use by female sex workers in two Indian states.

Spatial memory performance of Wistar rats exposed to mobile phone.

The effect of the duration of exposure to the electromagnetic field emitted by mobile phones on human attention.

Interference of GSM mobile phones with communication between Cardiac Rhythm Management devices and programmers: A combined in vivo and in vitro study.

Guidance for exposure design of human studies addressing health risk evaluations of mobile phones.

Exposure to non-ionizing electromagnetic fields emitted from mobile phones induced DNA damage in human ear canal hair follicle cells.

Effects of mobile phone radiofrequency on the structure and function of the normal human hemoglobin.

Impact of head morphology on local brain specific absorption rate from exposure to mobile phone radiation.

Can the Accuracy of Home Blood Glucose Monitors be affected by the Received Signal Strength of 900 MHz GSM Mobile Phones?

Are some people sensitive to mobile phone signals? Within participants double blind randomised provocation study.

Women's mobile phone use in birth suite: A West Australian perspective.

Effect of mobile telephones on sperm quality: a systematic review and meta-analysis.

Social behavioral testing and brain magnetic resonance imaging in chicks exposed to mobile phone radiation during development.

[Effects of electromagnetic fields emitted by cellular phone on auditory and vestibular labyrinth].

Mobile phone hygiene: potential risks posed by use in the clinics of an Indian dental school.

Impact of Adolescents' Screen Time and Nocturnal Mobile Phone-Related Awakenings on Sleep and General Health Symptoms: A Prospective Cohort Study.

The association between use of mobile phones after lights out and sleep disturbances among Japanese adolescents: a nationwide cross-sectional survey.

The influence of direct mobile phone radiation on sperm quality.

The effect of mobile phone on the number of Purkinje cells: a stereological study.

Some ocular symptoms experienced by users of mobile phones.

Radio frequency exposure in mobile phone users: implications for exposure assessment in epidemiological studies.

Mobile phone use facilitates memory in male, but not female, subjects.

Use of mobile phones and changes in cognitive function in adolescents.

Assessment of the radio-frequency electromagnetic fields induced in the human body from mobile phones used with hands-free kits.

An epidemiological review of mobile telephones and cancer.

Interaction of mobile phones with superficial passive metallic implants.

The effect of electromagnetic field emitted by a mobile phone on the inhibitory control of saccades.

Effects of GSM 900 MHz on middle cerebral artery blood flow assessed by transcranial Doppler sonography.

How to encourage children to use mobile phones safely.

Effects of electromagnetic fields from mobile phones on depression and anxiety after titanium mesh cranioplasty among patients with traumatic brain injury.

Individual differences in the effects of mobile phone exposure on human sleep: rethinking the problem.

[Correlation of health literacy and mobile phone use dependence with psychopathological symptoms in middle school students].

Acute effects of using a mobile phone on CNS functions.

Effect of electromagnetic fields emitted by cellular phones on the latency of evoked electrodermal activity.

Evaluation of the effect of using mobile phones on male fertility.

Effect of prenatal exposure to mobile phone on pyramidal cell numbers in the mouse hippocampus: a stereological study.

Mobile phone use on a young person's unit.

Mobile phones: are children at higher risk?

Effects of mobile phone radiation on reproduction and development in Drosophila melanogaster.

Quantitative changes in testicular structure and function in rat exposed to mobile phone radiation.

The effects of the duration of mobile phone use on heart rate variability parameters in healthy subjects.

Specific absorption rate variation in a brain phantom due to exposure by a 3G mobile phone: problems in dosimetry.

[Acute ear trauma caused by failure of mobile phone/cellular phone].

Assessment of auditory evoked potential in long-term mobile phone users.

Is exposure to personal music players a confounder in adolescent mobile phone use and hearing health studies?

Interference to medical equipment from mobile phones.

Identifying seasonal mobility profiles from anonymized and aggregated mobile phone data. Application in food security.

Effects of mobile phone electromagnetic fields: critical evaluation of behavioral and neurophysiological studies.

Estimation of RF energy absorbed in the brain from mobile phones in the Interphone Study.

[Electromagnetic fields in the vicinity of DECT cordless telephones and mobile phones].

Thermal effects of mobile phones on facial nerves and surrounding soft tissue.

Intraoperative observation of changes in cochlear nerve action potentials during exposure to electromagnetic fields generated by mobile phones.

An old issue and a new look: electromagnetic hypersensitivity caused by radiations emitted by GSM mobile phones.

Can mobile phone emissions affect auditory functions of cochlea or brain stem?

Influence on the mechanisms of generation of distortion product otoacoustic emissions of mobile phone exposure.

Does acute exposure to the electromagnetic field emitted by a mobile phone influence visual evoked potentials? A pilot study.

The estimation of 3D SAR distributions in the human head from mobile phone compliance testing data for epidemiological studies.

The acute auditory effects of exposure for 60 minutes to mobile`s electromagnetic field.

Long-term digital mobile phone use and cognitive decline in the elderly.

Mobile Phone: A Possible Vector of Bacterial Transmission in Hospital Setting.

Electrophysiological Assessment of the Impact of Mobile Phone Radiation on Cognition in Persons With Epilepsy.

Mobile phone affects cerebral blood flow in humans.

Association between mobile phone use and semen quality: a systemic review and meta-analysis.

Effects of Mobile Phones on Children's and Adolescents' Health: A Commentary.

[Activity of vestibular organ in people using mobile phones professionally].

Analysis of three-dimensional SAR distributions emitted by mobile phones in an epidemiological perspective.

Perception of the electromagnetic field emitted by a mobile phone.

[Effects of electromagnetic fields generated by mobile phones on the nervous system].

Effects of high frequency electromagnetic field (EMF) emitted by mobile phones on the human motor cortex.

[Cellular phones and cancer: current status].

Local vasodilator response to mobile phones.

Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication--a literature review published between 2000 and 2004.

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Diverse radiofrequency sensitivity and radiofrequency effects of mobile or cordless phone near fields exposure in Drosophila melanogaster.

Biophysical evaluation of radiofrequency electromagnetic field effects on male reproductive pattern.

The semen quality of the mobile phone users.

Tinnitus and mobile phone use.

Association of excessive mobile phone use during pregnancy with birth weight: an adjunct study in Kumamoto of Japan Environment and Children's Study.

Is problematic mobile phone use explained by chronotype and personality?

Effects of mobile phone emissions on human brain activity and sleep variables.

Effect of Bluetooth headset and mobile phone electromagnetic fields on the human auditory nerve.

Cranial and postcranial skeletal variations induced in mouse embryos by mobile phone radiation.

Effects of exposure to a mobile phone on testicular function and structure in adult rabbit.

"MXing it up": how African adolescents may affect social change through mobile phone use.

Mobile-phone-based home exercise training program decreases systemic inflammation in COPD: a pilot study.

Effects of short-term radiation emitted by WCDMA mobile phones on teenagers and adults.

Diseases of modern living: neurological changes associated with mobile phones and radiofrequency radiation in humans.

GSM mobile phone radiation suppresses brain glucose metabolism.

The electromagnetic interference of mobile phones on the function of a gamma-camera.

Effects of mobile phone exposure on biochemical parameters of cord blood: A preliminary study.

[An analysis of the pathogenetic significance of irradiations from mobile phones].

Self-reported symptoms associated with exposure to electromagnetic fields: a questionnaire study.

The mobile phone decreases fructose but not citrate in rabbit semen: a longitudinal study.

Mobile phone use and subjective symptoms. Comparison of symptoms experienced by users of analogue and digital mobile phones.

Acute effects of the electromagnetic waves emitted by mobile phones on attention in emergency physicians.

Multiple assessment methods of prenatal exposure to radio frequency radiation from telecommunication in the Mothers and Children's Environmental Health (MOCEH) study.

Mobile phone use and possible cancer risk: Current perspectives in India.

Symptomatic complex partial status epilepticus manifesting as utilization behavior of a mobile phone.

Genotoxic and carcinogenic effects of non-ionizing electromagnetic fields.

"Mate! I'm running 10 min late": An investigation into the self-regulation of mobile phone tasks while driving.

Dose related shifts in the developmental progress of chick embryos exposed to mobile phone induced electromagnetic fields.

Growing concern over the safety of using mobile phones and male fertility.

Is there any exposure from a mobile phone in stand-by mode?

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MEMO--a mobile phone depression prevention intervention for adolescents: development process and postprogram findings on acceptability from a randomized controlled trial.

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Neurodevelopment for the first three years following prenatal mobile phone use, radio frequency radiation and lead exposure.

Electromagnetic safety of children using wireless phones: a literature review.

Histological and histochemical study of the protective role of rosemary extract against harmful effect of cell phone electromagnetic radiation on the parotid glands.

Mobile phone use for contacting emergency services in life-threatening circumstances.

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Fetal and neonatal responses following maternal exposure to mobile phones.

Is the effect of mobile phone radiofrequency waves on human skin perfusion non-thermal?

Systematic review and meta-analysis of psychomotor effects of mobile phone electromagnetic fields.

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Age-dependent tissue-specific exposure of cell phone users.

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Neuropsychological sequelae of digital mobile phone exposure in humans.

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Genotoxicity evaluation of electromagnetic fields generated by 835-MHz mobile phone frequency band.

A survey study on some neurological symptoms and sensations experienced by long term users of mobile phones.

Does evening exposure to mobile phone radiation affect subsequent melatonin production?

Nocebo as headache trigger: evidence from a sham-controlled provocation study with RF fields.

Mobile phones and elderly people: a noisy communication.

Recall of past use of mobile phone handsets.

Mobile phone use and willingness to pay for SMS for diabetes in Bangladesh.

Electromagnetic absorption in the head of adults and children due to mobile phone operation close to the head.

Preliminary evaluation of nanoscale biogenic magnetite-based ferromagnetic transduction mechanisms for mobile phone bioeffects.

[Mobile phones, web chat, and sex among Norwegian adolescents].

Comparison of FDTD-calculated specific absorption rate in adults and children when using a mobile phone at 900 and 1800 MHz.

Effects of the Effect of Ultra High Frequency Mobile Phone Radiation on Human Health.

Dosimetric comparison of the specific anthropomorphic mannequin (SAM) to 14 anatomical head models using a novel definition for the mobile phone positioning.

Can electromagnetic fields emitted by mobile phones stimulate the vestibular organ?

Analysis on the effect of the distances and inclination angles between human head and mobile phone on SAR.

The use of a 'phantom scalp' to assess the possible direct pickup of mobile phone handset emissions by electroencephalogram electrode leads.

[Change settings for visual analyzer of child users of mobile communication: longitudinal study].

Determinants of mobile phone output power in a multinational study: implications for exposure assessment.

Effects of electromagnetic radiation of mobile phones on the central nervous system.

Noncommunicable Disease Risk Factors and Mobile Phones: A Proposed Research Agenda.

Effect of mobile phone usage time on total antioxidant capacity of saliva and salivary immunoglobulin a.

Mobile cell-phones (M-phones) in telemicroscopy: increasing connectivity of isolated laboratories.

A new problem in inflammatory bladder diseases: use of mobile phones!

Effects of mobile phone exposure on metabolomics in the male and female reproductive systems.

The pattern of the electromagnetic field emitted by mobile phones in motor vehicle driving simulators.

Derangement of chick embryo retinal differentiation caused by radiofrequency electromagnetic fields.

Mobile-phone dispatch of laypersons for CPR in out-of-hospital cardiac arrest.

Numerical assessment of induced ELF currents in the human head due to the battery current of a digital mobile phone.

Moving the Agenda on Noncommunicable Diseases: Policy Implications of Mobile Phone Surveys in Low and Middle-Income Countries.

Effect of mobile phone use on salivary concentrations of protein, amylase, lipase, immunoglobulin A, lysozyme, lactoferrin, peroxidase and C-reactive protein of the parotid gland.

Wi-Fi technology--an uncontrolled global experiment on the health of mankind.

Phantom vibration and phantom ringing among mobile phone users: A systematic review of literature.

Structural and kinetic effects of mobile phone microwaves on acetylcholinesterase activity.

Tinnitus and cell phones: the role of electromagnetic radiofrequency radiation.

[Psychophysiological indicators for children using mobile phones. Communication 2. Results of four-year monitoring].

Who reaps the benefits, who bears the risks? Comparative optimism, comparative utility, and regulatory preferences for mobile phone technology.

Electromagnetic Fields of Mobile Phone Jammer Exposure on Blood Factors in Rats.

Effect of handheld mobile phone use on parotid gland salivary flow rate and volume.

[Mobile phone use as a risk factor for affection of the central nerve system--secondary publication].

ELF exposure from mobile and cordless phones for the epidemiological MOBI-Kids study.

The Effect of Electromagnetic Radiation due to Mobile Phone Use on Thyroid Function in Medical Students Studying in a Medical College in South India.

Long-term mobile phone use and the risk of vestibular schwannoma: a Danish nationwide cohort study.

Cellular phones for reducing battlefield stress: rationale and a preliminary research.

Assessment of SAR and thermal changes near a cochlear implant system for mobile phone type exposures.

Evaluation of the mobile phone electromagnetic radiation on serum iron parameters in rats.

Asymmetries in hip mineralization in mobile cellular phone users.

Mobile Phone Use Behaviors and Postures on Public Transportation Systems.

Radiofrequency Electromagnetic Radiation and Memory Performance: Sources of Uncertainty in Epidemiological Cohort Studies.

The effects of multivitamin supplementation on mood and general well-being in healthy young adults. A laboratory and at-home mobile phone assessment.

[Monitor of ECG signal and heart rate using a mobile phone with Bluetooth communication protocol].

Safe use of mobile phones in hospitals.

Exposure to mobile phone radiation opens new horizons in Alzheimer's disease treatment.

[Mobile telephones: a 'new risk'].

Predicting the biological effects of mobile phone radiation absorbed energy linked to the MRI-obtained structure.

Metal-framed spectacles and implants and specific absorption rate among adults and children using mobile phones at 900/1800/2100 MHz.

Mobile telephones: a comparison of radiated power between 3G VoIP calls and 3G VoCS calls.

A framework for spatial interaction analysis based on large-scale mobile phone data.

Self-reported depression and anxiety symptoms and usage of computers and mobile phones among working-age Finns.

Motivating men who have sex with men to get tested for HIV through the internet and mobile phones: a qualitative study.

The influence of handheld mobile phones on human parotid gland secretion.

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Development of a problematic mobile phone use scale for Turkish adolescents.

Radiofrequency fields, transthyretin, and Alzheimer's disease.

Mobile phone mast effects on common frog (Rana temporaria) tadpoles: the city turned into a laboratory.

Analysis of RF exposure in the head tissues of children and adults.

SARs for pocket-mounted mobile telephones at 835 and 1900 MHz.

Intravital Computer Morphometry on Protozoa: A Method for Monitoring of the Morphofunctional Disorders in Cells Exposed in the Cell Phone Communication Electromagnetic Field.

Estimation of the SAR in the human head and body due to radiofrequency radiation exposure from handheld mobile phones with hands-free accessories.

Ants can be used as bio-indicators to reveal biological effects of electromagnetic waves from some wireless apparatus.

Regulating hearing aid compatibility of cell phones: results from a national survey.

Laughter counteracts enhancement of plasma neurotrophin levels and allergic skin wheal responses by mobile phone-mediated stress.

Recently published papers: take your predictions with a drop of saline... and breathe deeply before turning on your phone.

Interactions of problematic mobile phone use and psychopathological symptoms with unintentional injuries: a school-based sample of Chinese adolescents.

Critical comments on DNA breakage by mobile-phone electromagnetic fields [Diem et al., Mutat. Res. 583 (2005) 178-183].

Mobile phone-delivered reminders and incentives to improve childhood immunisation coverage and timeliness in Kenya (M-SIMU): a cluster randomised controlled trial.

Enhancement of allergic skin wheal responses in patients with atopic eczema/dermatitis syndrome by playing video games or by a frequently ringing mobile phone.

Pilot study of mobile phone technology in allergic rhinitis in European countries: the MASK-rhinitis study.

A closed-loop process to recover Li and Co compounds and to resynthesize LiCoO2 from spent mobile phone batteries.

--Leaf Cluster 1 (36)

Theme - Effects of mobile phones on brain and neural function

Titles

Mobile phone use for 5 minutes can cause significant memory impairment in humans.

Association between mobile phone use and inattention in 7102 Chinese adolescents: a population-based cross-sectional study.

Clinical features of headache associated with mobile phone use: a cross-sectional study in university students.

Predictors of mobile telephone use and exposure analysis in Australian adolescents.

Acute Effect of Electromagnetic Waves Emitted from Mobile Phone on Visual Evoked Potential in Adult Male : A Preliminary Study.

Analysis of the mobile phone effect on the heart rate variability by using the largest Lyapunov exponent.

Mobile Phone Use and The Risk of Headache: A Systematic Review and Meta-analysis of Cross-sectional Studies.

Acute mobile phone effects on pre-attentive operation.

Effect Of Electromagnetic Waves Emitted From Mobile Phone On Brain Stem Auditory Evoked Potential In Adult Males.

Acute effects of radiofrequency electromagnetic field emitted by mobile phone on brain function.

Psychophysiological tests and provocation of subjects with mobile phone related symptoms.

The cardiac effects of a mobile phone positioned closest to the heart.

Electromagnetic field of mobile phones affects visual event related potential in patients with narcolepsy.

Acute mobile phone operation affects neural function in humans.

Use of mobile and cordless phones and cognition in Australian primary school children: a prospective cohort study.

Effect of mobile phone radiation on heart rate variability.

[Effects of radiation emitted from mobile phones on short- term heart rate variability parameters].

Mobile phone use and health symptoms in children.

The sensitivity of human event-related potentials and reaction time to mobile phone emitted electromagnetic fields.

Factors associated with mental health among high school students in Iran: Does mobile phone overuse associate with poor mental health?

Mobile phone effects on children's event-related oscillatory EEG during an auditory memory task.

Examining the effects of electromagnetic fields emitted by GSM mobile phones on human event-related potentials and performance during an auditory task.

Estimating transmitted power density from mobile phone: an epidemiological pilot study with a software modified phone.

"Nomophobia": impact of cell phone use interfering with symptoms and emotions of individuals with panic disorder compared with a control group.

Mobile phone use and exposures in children.

The effect of mobile phone electromagnetic fields on the alpha rhythm of human electroencephalogram.

Use of mobile and cordless phones and change in cognitive function: a prospective cohort analysis of Australian primary school children.

Neurophysiological effects of mobile phone electromagnetic fields on humans: a comprehensive review.

[Effect of mobile phone electromagnetic emission on characteristics of cerebral blood circulation and neurohumoral regulations in humans].

Effects of 2G and 3G mobile phones on human alpha rhythms: Resting EEG in adolescents, young adults, and the elderly.

Acute mobile phones exposure affects frontal cortex hemodynamics as evidenced by functional near-infrared spectroscopy.

Effects of exposure to a mobile phone on sexual behavior in adult male rabbit: an observational study.

Effects of concurrent caffeine and mobile phone exposure on local target probability processing in the human brain.

Some ocular symptoms and sensations experienced by long term users of mobile phones.

Nasal colonization and bacterial contamination of mobile phones carried by medical staff in the operating room.

Headache and sferics.

--Leaf Cluster 25 (68)

Theme - Effects of cell phone radiation on cognitive function and hearing

Titles

Effects of GSM cellular phones on human hearing: the European project "GUARD".

Effects of weak mobile phone - electromagnetic fields (GSM, UMTS) on event related potentials and cognitive functions.

Mobile phone emission modulates event-related desynchronization of alpha rhythms and cognitive-motor performance in healthy humans.

Effects of pulsed and continuous wave 902 MHz mobile phone exposure on brain oscillatory activity during cognitive processing.

Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task.

Effects of weak mobile phone - electromagnetic fields (GSM, UMTS) on well-being and resting EEG.

Assessment of potential effects of the electromagnetic fields of mobile phones on hearing.

Effects of electromagnetic field emitted by cellular phones on the EEG during an auditory memory task: a double blind replication study.

Human brain wave activity during exposure to radiofrequency field emissions from mobile phones.

Effects of electromagnetic fields emitted by cellular phones on the electroencephalogram during a visual working memory task.

Effects of radiofrequency radiation emitted by cellular telephones on the cognitive functions of humans.

Cognitive effects of radiation emitted by cellular phones: the influence of exposure side and time.

Mobile phone emission modulates interhemispheric functional coupling of EEG alpha rhythms.

Effects of mobile phone exposure on time frequency fine structure of transiently evoked otoacoustic emissions.

[Effects of electromagnetic field from cellular phones on selected central nervous system functions: a literature review].

Effects of UMTS cellular phones on human hearing: results of the European project EMFnEAR.

Is the brain influenced by a phone call? An EEG study of resting wakefulness.

Effect of acute exposure to radiofrequency electromagnetic fields emitted by a mobile phone (GSM 900 MHz) on electrodermal responsiveness in healthy human.

Human brain activity during exposure to radiofrequency fields emitted by cellular phones.

Effects of exposure to electromagnetic fields emitted by GSM 900 and WCDMA mobile phones on cognitive function in young male subjects.

Preattentive auditory information processing under exposure to the 902 MHz GSM mobile phone electromagnetic field: a mismatch negativity (MMN) study.

Pulsed and continuous wave mobile phone exposure over left versus right hemisphere: effects on human cognitive function.

Mobile phone emission increases inter-hemispheric functional coupling of electroencephalographic alpha rhythms in epileptic patients.

Electromagnetic fields produced by GSM cellular phones and heart rate variability.

Effect of a 902 MHz electromagnetic field emitted by mobile phones on human cognitive function: A replication study.

The effects of mobile-phone electromagnetic fields on brain electrical activity: a critical analysis of the literature.

Hypersensitivity symptoms associated with exposure to cellular telephones: no causal link.

Nonlinear heart rate variability measures under electromagnetic fields produced by GSM cellular phones.

Mobile phone emission modulates inter-hemispheric functional coupling of EEG alpha rhythms in elderly compared to young subjects.

A study of the effects of cellular telephone microwave radiation on the auditory system in healthy men.

Cognitive effects of cellular phones: a possible role of non-radiofrequency radiation factors.

Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG.

Mobile phones exposure induces changes of contingent negative variation in humans.

Investigation of potential effects of cellular phones on human auditory function by means of distortion product otoacoustic emissions.

Effects of microwaves emitted by cellular phones on human slow brain potentials.

Effects of radiofrequency electromagnetic fields on the human nervous system.

Variations in electroencephalography with mobile phone usage in medical students.

Effects of 2G and 3G mobile phones on performance and electrophysiology in adolescents, young adults and older adults.

The excretion of 6-hydroxymelatonin sulfate in healthy young men exposed to electromagnetic fields emitted by cellular phone -- an experimental study.

Gender related differences on the EEG during a simulated mobile phone signal.

Cellular Phone Irradiation of the Head Affects Heart Rate Variability Depending on Inspiration/Expiration Ratio.

Effects of RF exposure of teenagers and adults by CDMA cellular phones.

Effects of 900 MHz electromagnetic fields exposure on cochlear cells' functionality in rats: evaluation of distortion product otoacoustic emissions.

Effects of the acute exposure to the electromagnetic field of mobile phones on human auditory brainstem responses.

Do mobile phones pose a potential risk to autonomic modulation of the heart?

Effects of intensive and moderate cellular phone use on hearing function.

A meta-analysis for neurobehavioural effects due to electromagnetic field exposure emitted by GSM mobile phones.

Mobile phone emissions modulate brain excitability in patients with focal epilepsy.

Mobile phone emissions and human brain excitability.

Effects of pulsed electromagnetic fields on cognitive processes - a pilot study on pulsed field interference with cognitive regeneration.

Scalp localization of human auditory cortical activity modified by GSM electromagnetic fields.

Effect of 902 MHz mobile phone transmission on cognitive function in children.

Physiological effects of RF exposure on hypersensitive people by a cell phone.

Effects of mobile phone signals over BOLD response while performing a cognitive task.

Effect of electromagnetic field emitted by cellular phones on fetal heart rate patterns.

Effects of W-CDMA 1950 MHz EMF emitted by mobile phones on regional cerebral blood flow in humans.

Does acute exposure to mobile phones affect human attention?

Effects of GSM signals during exposure to event related potentials (ERPs).

The effect of GSM and TETRA mobile handset signals on blood pressure, catechol levels and heart rate variability.

Thermal effects of mobile phone RF fields on children: a provocation study.

Controversies on electromagnetic field exposure and the nervous systems of children.

The influence of the call with a mobile phone on heart rate variability parameters in healthy volunteers.

[A study on the biological effects of exposure mobile-phone frequency EMF].

Effects of Bluetooth device electromagnetic field on hearing: pilot study.

Evaluation in humans of the effects of radiocellular telephones on the circadian patterns of melatonin secretion, a chronobiological rhythm marker.

Comparison of earphone radiation recorded from hearing impaired subjects and a resistor network simulator.

[The influence of hypogeomagnetic field on bioelectric activity of the brain in epilepsy].

Non-ionizing radiofrequency electromagnetic waves traversing the head can be used to detect cerebrovascular autoregulation responses.

--Leaf Cluster 14 (93)

Theme - Myriad adverse health effects from cellphones

Titles

Cell phone use and acoustic neuroma: the need for standardized questionnaires and access to industry data.

Cell-phone use and self-reported hypertension: national health interview survey 2008.

Cell phones: modern man's nemesis?

Impact of cell phone use on men's semen parameters.

A preliminary examination of cell phone use and helping behavior.

Cell phone use and behavioural problems in young children.

An analysis of the impact of cell phone use on depressive symptoms among Japanese elders.

Cell phones and brain tumors: a review including the long-term epidemiologic data.

Prenatal and postnatal exposure to cell phone use and behavioral problems in children.

Maternal cell phone use during pregnancy and child behavioral problems in five birth cohorts.

Maternal cell phone use in early pregnancy and child's language, communication and motor skills at 3 and 5 years: the Norwegian mother and child cohort study (MoBa).

Cell phones and tumor: still in no man's land.

Cell phones and male infertility: a review of recent innovations in technology and consequences.

Prenatal exposure to cell phone use and neurodevelopment at 14 months.

The effects of cell phone use on peripheral vision.

Factors associated with cell phone use in adolescents in the community of Madrid (Spain).

The Impact of Using Cell Phones After Light-Out on Sleep Quality, Headache, Tiredness, and Distractibility Among Students of a University in North of Iran.

Prospective study of pregnancy outcomes after parental cell phone exposure: the Norwegian Mother and Child Cohort Study.

Prenatal and Postnatal Cell Phone Exposures and Headaches in Children.

Cell-Phone Addiction: A Review.

Ambulatory cell phone injuries in the United States: an emerging national concern.

[Cell Phones and Risk of brain and acoustic nerve tumours: the French INTERPHONE case-control study].

Cell phone usage and erectile function.

Real-world cell phone radiofrequency electromagnetic field exposures.

Cell phone exposures and hearing loss in children in the Danish National Birth Cohort.

Cell Phone Information Seeking Explains Blood Pressure in African American Women.

Habits of cell phone usage and sperm quality - does it warrant attention?

Maternal cell phone use during pregnancy and child cognition at age 5years in 3 birth cohorts.

Cell phones and male infertility: dissecting the relationship.

Augmentative and alternative communication and cell phone use: one off-the-shelf solution and some policy considerations.

Cell phones and cancer: what is the evidence for a connection?

Cancer risks related to low-level RF/MW exposures, including cell phones.

Effect of cell-phone radiofrequency on angiogenesis and cell invasion in human head and neck cancer cells.

Is health literacy related to health behaviors and cell phone usage patterns among the text4baby target population?

Cell phone use and risk of thyroid cancer: a population-based case-control study in Connecticut.

Cell phones and children: follow the precautionary road.

Association between number of cell phone contracts and brain tumor incidence in nineteen U.S. States.

Maternal cell phone and cordless phone use during pregnancy and behaviour problems in 5-year-old children.

The effect of cell phone use on postural balance and mobility in older compared to young adults.

Cell phones change the way we walk.

Effects of cell phone use on semen parameters: Results from the MARHCS cohort study in Chongqing, China.

Multidrug-Resistant Bacteria Associated with Cell Phones of Healthcare Professionals in Selected Hospitals in Saudi Arabia.

Effect of cell phone exposure on physiologic and hematologic parameters of male medical students of Bijapur (Karnataka) with reference to serum lipid profile.

General health of students of medical sciences and its relation to sleep quality, cell phone overuse, social networks and internet addiction.

The role of anxiety in the perception of technological hazards - a cross-sectional study on cell phones and masts.

Effect of Cell Phone Radiations on Orofacial Structures: A Systematic Review.

The association of sleep and late-night cell phone use among adolescents.

Cell Phone Use and Child and Adolescent Reading Proficiency.

Reach for your cell phone at your own risk: The cognitive costs of media choice for breaks.

Effects of cell phone radiofrequency signal exposure on brain glucose metabolism.

A forecasting method to reduce estimation bias in self-reported cell phone data.

The incidence rate and mortality of malignant brain tumors after 10 years of intensive cell phone use in Taiwan.

Abnormal responses of electronic pocket dosimeters caused by high frequency electromagnetic fields emitted from digital cellular telephones.

[Cell phones: health risks and prevention].

Does cell phone use increase the chances of parotid gland tumor development? A systematic review and meta-analysis.

Prevalence of problematic cell phone use in an adult population in Spain as assessed by the Mobile Phone Problem Use Scale (MPPUS).

Use of mobile phone during pregnancy and the risk of spontaneous abortion.

Cell phones: the psychosocial risks.

Prenatal cell phone use and developmental milestone delays among infants.

Associations of Maternal Cell-Phone Use During Pregnancy With Pregnancy Duration and Fetal Growth in 4 Birth Cohorts.

Effect of cell phone magnetic fields on adjustable cerebrospinal fluid shunt valves.

Combined effects of varicocele and cell phones on semen and hormonal parameters.

Chatting in the face of the eyewitness: The impact of extraneous cell-phone conversation on memory for a perpetrator.

Risks to Health and Well-Being From Radio-Frequency Radiation Emitted by Cell Phones and Other Wireless Devices.

Effect of Mobile Phone Radiofrequency Electromagnetic Fields on.

Absorption of wireless radiation in the child versus adult brain and eye from cell phone conversation or virtual reality.

Is there a relationship between cell phone use and semen quality?

Cell phone-generated radio frequency electromagnetic field effects on the locomotor behaviors of the fishes Poecilia reticulata and Danio rerio.

Not So Smart: Cell Phone Use Hurts Our Patients and Profession.

Cell phone etiquette in the clinical arena: A professionalism imperative for healthcare.

Mobile Phones: Potential Sources of Nickel and Cobalt Exposure for Metal Allergic Patients.

[Risk perception of the general public of cell phone towers and cancer: trend and associated factors, 2005-2010].

Electromagnetic field and brain development.

Impacts of silver-coated antimicrobial screen covers on the cell-phone microbiome of resident physicians.

The effects of cell phone conversations on the attention and memory of bystanders.

Effects of cell-phone and text-message distractions on true and false recognition.

Vestibular schwannoma and cell-phones. Results, limits and perspectives of clinical studies.

The psychometric properties of cellular phone dependency questionnaire in students of Isfahan: A pilot study.

Multidrug-resistant bacteria isolated from cell phones in five intensive care units: Exploratory dispersion analysis.

[The health problems which can brougth by 3G cell phones to our country].

Exposure limits: the underestimation of absorbed cell phone radiation, especially in children.

Do people understand IARC's 2B categorization of RF fields from cell phones?

Allergic Contact Dermatitis to a Cell Phone.

Impact of pinna compression on the RF absorption in the heads of adult and juvenile cell phone users.

Do cell phones, iPods/MP3 players, siblings and friends matter? Predictors of child body mass in a U.S. Southern Border City Middle School.

The role of cellular phone usage by parents in the increase in ASD occurrence: A hypothetical framework.

Psychophysiological patterns during cell phone text messaging: a preliminary study.

From sweeteners to cell phones-Cancer myths and beliefs among journalism undergraduates.

Can Fish and Cell Phones Teach Us about Our Health?

Cell Phone Counseling Improves Retention of Mothers With HIV Infection in Care and Infant HIV Testing in Kisumu, Kenya: A Randomized Controlled Study.

Association between problematic cellular phone use and suicide: the moderating effect of family function and depression.

Symptoms of problematic cellular phone use, functional impairment and its association with depression among adolescents in Southern Taiwan.

Adolescent in-school cellphone habits: a census of rules, survey of their effectiveness, and fertility implications.

--Leaf Cluster 7 (44)

Theme - Risks from cell phone use, especially brain tumors

Titles

Validation of self-reported cellular phone use.

Cellular phone use and brain tumor: a meta-analysis.

[Symptoms reported by mobile cellular telephone users].

Risk of pituitary tumors in cellular phone users: a case-control study.

Cellular and cordless telephone use and the association with brain tumors in different age groups.

Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany).

Use of cellular telephones and the risk for brain tumours: A case-control study.

Further aspects on cellular and cordless telephones and brain tumours.

Cellular phone use and risk of benign and malignant parotid gland tumors--a nationwide case-control study.

Cellular-telephone use and brain tumors.

[In vitro and in vivo study of electromagnetic compatibility of cellular phones and pacemakers].

Cellular and cordless telephones and the risk for brain tumours.

Cellular phones and their hazards: the current evidence.

Use of cellular telephones and brain tumour risk in urban and rural areas.

Case-control study on the use of cellular and cordless phones and the risk for malignant brain tumours.

Characteristics of excessive cellular phone use in Korean adolescents.

Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma.

Cellular telephone use and risk of intratemporal facial nerve tumor.

[Study of the influence of cellular phones and personal computers on schoolchildren's health: hygienic aspects].

Cellular telephone use and time trends for brain, head and neck tumours.

[Experimental data on radiofrequency].

Assessment of radiofrequency exposure from cellular telephone daily use in an epidemiological study: German Validation study of the international case-control study of cancers of the brain--INTERPHONE-Study.

Use of cellular and cordless telephones and risk of testicular cancer.

[Cellular phones and public health].

Estimation of relative exposure levels for cellular phone users using a neural network.

Cellular phone and cellular phone accessory dermatitis due to nickel allergy: report of five cases.

[Health risks of mobile phones].

Brain cancer incidence trends in relation to cellular telephone use in the United States.

Cellular phones and risk of brain tumors.

Correlation between cellular phone use and epithelial parotid gland malignancies.

[On the evaluation of the influence of cellular phones on their users].

Mobile Phone Use and the Risk of Parotid Gland Tumors: A Retrospective Case-Control Study.

Behavioral support to parents through a cellular-phone website that provides the degree of urgency for medical attention of a child.

Risk perception and public concerns of electromagnetic waves from cellular phones in Korea.

Frequent cellular phone use modifies hypothalamic-pituitary-adrenal axis response to a cellular phone call after mental stress in healthy children and adolescents: A pilot study.

The relationship between adolescents' well-being and their wireless phone use: a cross-sectional study.

New Zealand adolescents' cellphone and cordless phone user-habits: are they at increased risk of brain tumours already? A cross-sectional study.

Prevalence of headache among handheld cellular telephone users in Singapore: a community study.

[The electromagnetic fields of cellular phones and the health of children and of teenagers (the situation requiring to take an urgent measure)].

Patterns of cellular phone use among young people in 12 countries: Implications for RF exposure.

Use of wireless telephones and serum S100B levels: a descriptive cross-sectional study among healthy Swedish adults aged 18-65 years.

Risks for central nervous system diseases among mobile phone subscribers: a Danish retrospective cohort study.

The effect of feedback on attitudes toward cellular phone use while driving: a comparison between novice and experienced drivers.

Radio frequency electromagnetic fields: cancer, mutagenesis, and genotoxicity.

--Leaf Cluster 8 (106)

Theme - Risk of brain tumors/acoustic neuromas from mobile phone use

Titles

Mobile phone use and risk of glioma in 5 North European countries.

Mobile phone use and brain tumours in the CERENAT case-control study.

Use of mobile phones in Norway and risk of intracranial tumours.

Long-term mobile phone use and brain tumor risk.

Long-term use of cellular phones and brain tumours: increased risk associated with use for > or =10 years.

Mobile phone use and risk of glioma in adults: case-control study.

A case-case study of mobile phone use and acoustic neuroma risk in Japan.

Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries.

Mobile phone use and risk of acoustic neuroma: results of the Interphone case-control study in five North European countries.

Mobile phones, cordless phones and the risk for brain tumours.

Mobile phone use and the risk of acoustic neuroma.

Case-control study of the association between malignant brain tumours diagnosed between 2007 and 2009 and mobile and cordless phone use.

Mobile phone use and glioma risk: A systematic review and meta-analysis.

Mobile phone use and risk of brain neoplasms and other cancers: prospective study.

Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones.

Long-term mobile phone use and acoustic neuroma risk.

Mobile phone use and risk of brain tumours: a systematic review of association between study quality, source of funding, and research outcomes.

Mobile phone use and incidence of brain tumour histological types, grading or anatomical location: a population-based ecological study.

Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects.

Mobile phone use and acoustic neuroma risk in Japan.

Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: a case-control study.

Meta-analysis of long-term mobile phone use and the association with brain tumours.

The anatomical distribution of cerebral gliomas in mobile phone users.

Meningioma patients diagnosed 2007-2009 and the association with use of mobile and cordless phones: a case-control study.

Epidemiologic evidence on mobile phones and tumor risk: a review.

Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study.

The Intracranial Distribution of Gliomas in Relation to Exposure From Mobile Phones: Analyses From the INTERPHONE Study.

Childhood brain tumour risk and its association with wireless phones: a commentary.

Mobile phone use and risk for intracranial tumors.

Meningioma and mobile phone use--a collaborative case-control study in five North European countries.

Mobile phone use and glioma risk: comparison of epidemiological study results with incidence trends in the United States.

The controversy about a possible relationship between mobile phone use and cancer.

Mobile phones and brain tumours: a review of epidemiological research.

Meta-analysis of association between mobile phone use and glioma risk.

Mobile phones and head tumours. The discrepancies in cause-effect relationships in the epidemiological studies - how do they arise?

Mobile phone use and brain tumors in children and adolescents: a multicenter case-control study.

Mobile phone use and risk of intracranial tumors: a consistency analysis.

Pituitary tumor risk in relation to mobile phone use: A case-control study.

[Long-term use of mobile phone and its association with glioma: a systematic review and meta-analysis].

Mobile phones, brain tumors, and the interphone study: where are we now?

[Motivation and significance of IARC classification for mobile phone].

Mobile telephones and cancer--a review of epidemiological evidence.

The INTERPHONE study: design, epidemiological methods, and description of the study population.

Evaluation of Mobile Phone and Cordless Phone Use and Glioma Risk Using the Bradford Hill Viewpoints from 1965 on Association or Causation.

Pooled analysis of Swedish case-control studies during 1997-2003 and 2007-2009 on meningioma risk associated with the use of mobile and cordless phones.

Meta-analysis of mobile phone use and intracranial tumors.

[Association between radiation from mobile phones and tumour risk in adults].

Probabilistic Multiple-Bias Modeling Applied to the Canadian Data From the Interphone Study of Mobile Phone Use and Risk of Glioma, Meningioma, Acoustic Neuroma, and Parotid Gland Tumors.

Childhood brain tumours and use of mobile phones: comparison of a case-control study with incidence data.

Medical exposure to ionising radiation and the risk of brain tumours: Interphone study group, Germany.

Recall bias in the assessment of exposure to mobile phones.

Lost in laterality: interpreting "preferred side of the head during mobile phone use and risk of brain tumour" associations.

Wireless Phone Use and Risk of Adult Glioma: Evidence from a Meta-Analysis.

Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones.

Mobile phone use and risk for intracranial tumors and salivary gland tumors - A meta-analysis.

Use of mobile phones and risk of brain tumours: update of Danish cohort study.

Mobile phone use and risk of tumors: a meta-analysis.

[Mobile phones and head tumours: it is time to read and highlight data in a proper way].

Cellular telephones and risk for brain tumors: a population-based, incident case-control study.

Changes in brain glioma incidence and laterality correlates with use of mobile phones--a nationwide population based study in Israel.

Survival of glioma patients in relation to mobile phone use in Denmark, Finland and Sweden.

Mobile phone use and the risk for malignant brain tumors: a case-control study on deceased cases and controls.

A case-control study of risk of leukaemia in relation to mobile phone use.

Validation of self-reported start year of mobile phone use in a Swedish case-control study on radiofrequency fields and acoustic neuroma risk.

Environmental risk factors for sporadic acoustic neuroma (Interphone Study Group, Germany).

Use of mobile and cordless phones and survival of patients with glioma.

Impact of random and systematic recall errors and selection bias in case--control studies on mobile phone use and brain tumors in adolescents (CEFALO study).

Electromagnetic fields and brain tumours: a commentary.

[Radio and microwave frequency radiation and health--an analysis of the literature].

Long-term and frequent cellular phone use and risk of acoustic neuroma.

Validation of short term recall of mobile phone use for the Interphone study.

Systematic review of wireless phone use and brain cancer and other head tumors.

Mobile phone use and incidence of glioma in the Nordic countries 1979-2008: consistency check.

Selection bias due to differential participation in a case-control study of mobile phone use and brain tumors.

Decreased survival of glioma patients with astrocytoma grade IV (glioblastoma multiforme) associated with long-term use of mobile and cordless phones.

Quantifying the impact of selection bias caused by nonparticipation in a case-control study of mobile phone use.

Analyses of temporal and spatial patterns of glioblastoma multiforme and other brain cancer subtypes in relation to mobile phones using synthetic counterfactuals.

Estimating associations of mobile phone use and brain tumours taking into account laterality: a comparison and theoretical evaluation of applied methods.

Mobile phone use and risk of parotid gland tumor.

Epidemiology of brain tumors.

Review of four publications on the Danish cohort study on mobile phone subscribers and risk of brain tumors.

Evaluation of carcinogenic effects of electromagnetic fields (EMF).

Use of wireless phones and the risk of salivary gland tumours: a case-control study.

Epidemiology and etiology of gliomas.

Epidemiology of Intracranial Gliomas.

The effects of recall errors and of selection bias in epidemiologic studies of mobile phone use and cancer risk.

Time trend in incidence of malignant neoplasms of the central nervous system in relation to mobile phone use among young people in Japan.

A three-dimensional point process model for the spatial distribution of disease occurrence in relation to an exposure source.

[Risk of major lymphoma subtypes and use of mobile phones].

Simulation of the incidence of malignant brain tumors in birth cohorts that started using mobile phones when they first became popular in Japan.

Risks of carcinogenesis from electromagnetic radiation of mobile telephony devices.

Current state of our knowledge on brain tumor epidemiology.

Mobile phones, cordless phones and rates of brain tumors in different age groups in the Swedish National Inpatient Register and the Swedish Cancer Register during 1998-2015.

Has the incidence of brain cancer risen in Australia since the introduction of mobile phones 29 years ago?

Inferring the 1985-2014 impact of mobile phone use on selected brain cancer subtypes using Bayesian structural time series and synthetic controls.

Location of gliomas in relation to mobile telephone use: a case-case and case-specular analysis.

Mobile phone use and risk of uveal melanoma: results of the risk factors for uveal melanoma case-control study.

[The probability of developing brain tumours among users of cellular telephones (scientific information to the decision of the International Agency for Research on Cancer (IARC) announced on May 31, 2011)].

[Risk of neoplastic diseases in conditions of exposure to radio- and microwave fields--epidemiologic investigations].

Effects of alternative styles of risk information on EMF risk perception.

Medical students' risk perceptions on decreased attention, physical and social risks in using mobile phones and the factors related with their risk perceptions.

The possible role of radiofrequency radiation in the development of uveal melanoma.

Mobile phones and multiple sclerosis--a nationwide cohort study in Denmark.

Mobile phone use and the risk of skin cancer: a nationwide cohort study in Denmark.

Use of wireless phones and serum beta-trace protein in randomly recruited persons aged 18-65 years: a cross-sectional study.

Exposure to wireless phone emissions and serum beta-trace protein.

Fourth Level Cluster 89 (869)
Theme - Human health risks from electromagnetic radiation, including adverse effects on implanted electronic devices, and possible protections

--Leaf Cluster 0 (63)

Theme - Electromagnetic interference with cardiac pacemakers

Titles

The effects of mobile phones on pacemaker function.

[The effect of cell phones on pacemaker function].

Electromagnetic interference with implantable cardiac pacemakers by video capsule.

Influence of digital and analogue cellular telephones on implanted pacemakers.

[Pacemaker dysfunction during use of a mobile telephone].

Life after pacemaker implantation: management of common problems and environmental interactions.

[Cardiac pacemakers designed for magnetic resonance environment].

Interference with cardiac pacemakers by cellular telephones.

Pacemakers and magnetic resonance imaging: Current status and survey in Switzerland.

Influence of D-net (European GSM-Standard) cellular phones on pacemaker function in 50 patients with permanent pacemakers.

[Pacemaker dysfunction in the clinical practice].

Interference in pacemakers.

Interactions between pacemakers and security systems.

Electromagnetic interference with pacemakers caused by portable media players.

Electromagnetic compatibility of electronic implants--review of the literature.

Do European GSM mobile cellular phones pose a potential risk to pacemaker patients?

Electromagnetic interference in pacemakers in single-engine fixed-wing aircraft: a European perspective.

Pacemaker inhibition and asystole in a pacemaker dependent patient.

[Cardiac pacemaker dysfunction secondary to outside interference: a review].

Pacemakers: some of the risks and complications you are not warned about.

Is there a risk for interaction between mobile phones and single lead VDD pacemakers?

SAR evaluations of mobile phone close to a pacemaker implanted in human body.

[Is there any risk interaction between electromagnetic field generated by mobile phones and artificial pacemakers].

Electromagnetic interference with cardiac pacemakers and implantable cardioverter-defibrillators from low-frequency electromagnetic fields in vivo.

Induction ovens and electromagnetic interference: what is the risk for patients with implanted pacemakers?

Magnetic resonance imaging for patients with permanent pacemakers: initial clinical experience.

Magnetic interference of cardiac pacemakers from a surgical magnetic drape.

Electromagnetic compatibility study of the in-vitro interaction of wireless phones with cardiac pacemakers.

Do media players cause interference with pacemakers?

Electromagnetic interference of pacemakers by mobile phones.

Interference by cellular phones with permanent implanted pacemakers: an update.

Electrocardiographic "pacemaker pseudo-spikes" and radio frequency interference.

Pacemaker interference.

Reliability of electromagnetic filters of cardiac pacemakers tested by cellular telephone ringing.

Pacemaker interference by 60-Hz contact currents.

The effect of 50 Hz external electrical interference on implanted cardiac pacemakers.

Concerns about sources of electromagnetic interference in patients with pacemakers.

Selective interference with pacemaker activity by electrical dental devices.

Hospital pager systems may cause interference with pacemaker telemetry.

[Environment and permanent cardiac pacing].

Interference with cardiac pacing.

Electromagnetic interference of analog cellular telephones with pacemakers.

The effect of power frequency high intensity electric fields on implanted cardiac pacemakers.

The effect of radar on cardiac pacemakers.

Pacing in high field cardiac magnetic resonance imaging:.

Do induction cooktops interfere with cardiac pacemakers?

[Interference between cardiac pacemaker and electromagnetic anti-theft devices in stores].

[Effect of external electrical interference on pacemakers].

Effects of an increased air gap on the in vitro interaction of wireless phones with cardiac pacemakers.

Radiofrequency Scanning for Retained Surgical Items Can Cause Electromagnetic Interference and Pacing Inhibition if an Asynchronous Pacing Mode Is Not Applied.

Interference between mobile phones and pacemakers: a look inside.

[Electromagnetic interference of electrical dental equipment with cardiac pacemakers].

Electromagnetic interference of an external temporary pacemaker during maxillofacial and neck surgery.

Effect of electronic apex locators on cardiac pacemaker function.

Mode of operation induced by rapid external chest wall stimulation in patients with normally functioning QRS-inhibited (VVI) pacemakers.

Disturbances in the function of cardiac pacemaker caused by short wave and microwave diathermies and pulsed high frequency current.

[Cardiac Pacemakers, implantable defibrillators and IRM].

The safety of digital mobile cellular telephones with minute ventilation rate adaptive pacemakers.

Electromagnetic interference of implantable unipolar cardiac pacemakers by an induction oven.

[Health Council Report 'Radiofrequency electromagnetic fields (300 Hz-300 GHz). The Health Council of the Netherlands].

Characteristics of telemetry interference with pacemakers caused by digital media players.

Influence of mobile magnetic resonance imaging on implanted pacemakers.

[Compatibility of active implants in the professional environment].

--Leaf Cluster 16 (103)

Theme - Electromagnetic interference on implanted cardiac devices

Titles

Incidence of electromagnetic interference in implantable cardioverter defibrillators.

Effects of electromagnetic interference on implanted cardiac devices and their management.

Electromagnetic interference in cardiac rhythm management devices.

Surgical management of the patient with an implanted cardiac device: implications of electromagnetic interference.

Electromagnetic interference and implanted cardiac devices: the nonmedical environment (part I).

[The influence of non-ionizing electromagnetic fields on implantable cardiac medical devices].

Electromagnetic interference and implanted cardiac devices: the medical environment (part II).

Potential interference of small neodymium magnets with cardiac pacemakers and implantable cardioverter-defibrillators.

Implanted devices and electromagnetic interference: case presentations and review.

Are patients with cardiac implants protected against electromagnetic interference in daily life and occupational environment?

Safety of the colonoscope magnetic imaging device (ScopeGuide) in patients with implantable cardiac devices.

Intermittent, erratic behaviour of an implantable cardioverter defibrillator secondary to a hidden magnetic source of interference.

Shock whilst gardening--implantable defibrillators & lawn mowers.

An update on mobile phones interference with medical devices.

Characterization of electromagnetic interference of medical devices in the hospital due to cell phones.

Mobile phone interference with medical equipment and its clinical relevance: a systematic review.

Electromagnetic interference in implantable cardioverter defibrillators: present but rare.

Electromagnetic interference with implantable cardioverter-defibrillators at power frequency: an in vivo study.

Cellular phone interference with external cardiopulmonary monitoring devices.

Do airport metal detectors interfere with implantable pacemakers or cardioverter-defibrillators?

Electromagnetic interference of dental equipment with implantable cardioverter defibrillators.

Treatment of patients with cardiac pacemakers and implantable cardioverter-defibrillators during radiotherapy.

AANA Journal Course: update for nurse anesthetists. Arrhythmia management devices and electromagnetic interference.

In-vivo testing of digital cellular telephones in patients with implantable cardioverter-defibrillators.

[Magnets, pacemaker and defibrillator: fatal attraction?].

Induction ovens and electromagnetic interference: what is the risk for patients with implantable cardioverter defibrillators?

Electromagnetic Interference (EMI) and arrhythmic events in ICD patients undergoing gastrointestinal procedures.

Radiofrequency interference with medical devices. A technical information statement.

How do mobile phones affect electromedical devices?

Cell phones and electromagnetic interference revisited.

Patient safety and electromagnetic protection: a review.

[Return to work of a pacemaker bearing worker: the relationship between health problems and electromagnetic interferences].

Report of the American Medical Association (AMA) Council on Scientific Affairs and AMA recommendations to medical professional staff on the use of wireless radio-frequency equipment in hospitals.

Implantable cardioverter defibrillators and cellular telephones: is there any interference?

Wireless technologies and patient safety in hospitals.

[Magnetic resonance imaging in patients with pacemakers and implantable cardioverter-defibrillators: a systematic review].

[Medical implantable devices and electromagnetic compatibility].

Electromagnetic interference in critical care.

In vitro tests reveal sample radiofrequency identification readers inducing clinically significant electromagnetic interference to implantable pacemakers and implantable cardioverter-defibrillators.

[Do mobile telephones have adverse effects on the functions of implantable cardioverter defibrillators?].

A follow-up study of electromagnetic interference of cellular phones on electronic medical equipment in the emergency department.

Electromagnetic interference can cause hospital devices to malfunction, McGill group warns.

Electromagnetic interference from radio frequency identification inducing potentially hazardous incidents in critical care medical equipment.

Electromagnetic interference of endodontic equipments with cardiovascular implantable electronic device.

Is magnetic resonance safe in implanted cardiac devices patients?

Safety Considerations in Magnetic Resonance Imaging of Patients With Implanted Medical Devices.

Cardiac devices and electromagnetic interference revisited: new radiofrequency technologies and implications for dermatologic surgery.

State of the science: pacemaker and defibrillator interference from wireless communication devices.

Measurements of electromagnetic fields radiated from communication equipment and of environmental electromagnetic noise: impact on the use of communication equipment within the hospital.

Dosimetry of electromagnetic field exposure of an active armlet and its electromagnetic interference to the cardiac pacemakers using adult, child and infant models.

Implanted medical devices in workers exposed to radio-frequency radiation.

[Radiotherapy in patients with a pacemaker or an implantable cardioverter defibrillator].

Electromagnetic interference of communication devices on ECG machines.

Interference by new-generation mobile phones on critical care medical equipment.

[Use of mobile phones in hospitals do not jeopardise the safety of the patients].

Electromagnetic interference between external defibrillator and cardiac resynchronization therapy-pacemaker (CRT-P) devices.

Interference of electrical dental equipment with implantable cardioverter-defibrillators.

Electromagnetic interference of cardiac rhythmic monitoring devices to radio frequency identification: analytical analysis and mitigation methodology.

A practical procedure to prevent electromagnetic interference with electronic medical equipment.

Clinically significant magnetic interference of implanted cardiac devices by portable headphones.

Electromagnetic interference from GSM and TETRA phones with life-support medical devices.

Electromagnetic interference to infusion pumps. Update2008 from GSM mobile phones.

Electronic article surveillance systems and interactions with implantable cardiac devices: risk of adverse interactions in public and commercial spaces.

Electromagnetic immunity of infusion pumps to GSM mobile phones: a systematic review.

Avoidance behaviors in patients with implantable cardioverter defibrillators.

Electromagnetic interference of implantable cardiac devices from a shoulder massage machine.

Deaths associated with implantable cardioverter defibrillator failure and deactivation reported in the United States Food and Drug Administration Manufacturer and User Facility Device Experience Database.

Cochlear implants: in vitro investigation of electromagnetic interference at MR imaging--compatibility and safety aspects.

Electromagnetic interference with electronic medical equipment induced by automatic conveyance systems.

Possible electromagnetic interference with electronic medical equipment by radio waves coming from outside the hospital.

Interactions between electronic article surveillance systems and implantable cardioverter-defibrillators.

Risk of cellular phone interference with an implantable loop recorder.

Interference of electrocardiographic recordings by a mobile telephone.

Electromagnetic interference with infusion pumps from GSM mobile phones.

An implanted spherical head model exposed to electromagnetic fields at a mobile communication frequency.

Biomedical concerns in wireless communications.

In vitro study of the electromagnetic interaction between wireless phones and an implantable neural stimulator.

Safety aspects of radiofrequency power deposition in magnetic resonance.

Electromagnetic interference of bone-anchored hearing aids by cellular phones revisited.

Fatal collision? Are wireless headsets a risk in treating patients?

Ventricular fibrillation induced by radiofrequency energy delivery for premature ventricular contractions arising from the right ventricular outflow tract: is implantablecardioverterdefibrillator indicated?

Interaction of radio frequency electromagnetic fields and passive metallic implants--a brief review.

Use of mobile phones in ICU--why not ban?

Clinical testing of cellular phone ringing interference with automated external defibrillators.

Electromagnetic immunity of implantable pacemakers exposed to wi-fi devices.

Effect of digital cellular phones on tachyarrhythmia analysis of automated external defibrillators.

Interference with the operation of medical devices resulting from the use of radio frequency identification technology.

The impact of dental devices on neurostimulators.

Detection of refrigerator-associated 60 Hz alternating current as ventricular fibrillation by an implantable defibrillator.

Solutions to electromagnetic interference problems between cochlear implants and GSM phones.

[Electromagnetic fields of mobile telephone systems--thresholds, effects and risks for cochlear implant patients and healthy people].

[Interference testing in certification of medical equipment].

Assessment of the exposure to WLAN frequencies of a head model with a cochlear implant.

Mobile phones to improve the practice of neurology.

Is electromagnetic interference still a risk?

Cellular phone interference with the operation of mechanical ventilators.

[Electromagnetic fields in hospitals: wireless-LAN as a risk factor?].

[Influence of electromagnetic waves on portable electronic instruments in medicine].

Electromagnetic energy radiated from mobile phone alters electrocardiographic records of patients with ischemic heart disease.

Nullification of electromagnetic radiation: 50 Hz artifact during electroencephalogram recording.

Development of a silicon retinal implant: cortical evoked potentials following focal stimulation of the rabbit retina with light and electricity.

[Influence of the radiofrequency current on the left ventricular systolic function].

Smart phone: a popular device supports amylase activity assay in fisheries research.

--Leaf Cluster 5 (120)

Theme - Health risks from mobile phone base stations

Titles

Epidemiological evidence for a health risk from mobile phone base stations.

Mobile phone base stations and adverse health effects: phase 1 of a population-based, cross-sectional study in Germany.

Perception of mobile phone and base station risks.

Effect of mobile phone station on micronucleus frequency and chromosomal aberrations in human blood cells.

Public exposure to radio waves near GSM microcell and picocell base stations.

Mobile phone base stations and well-being--A meta-analysis.

Mobile phone base stations and adverse health effects: phase 2 of a cross-sectional study with measured radio frequency electromagnetic fields.

Assessment of exposure to mobile telecommunication electromagnetic fields.

Assessment of radiofrequency/microwave radiation emitted by the antennas of rooftop-mounted mobile phone base stations.

Electromagnetic field pattern in the environment of GSM base stations.

[Electormagnetic field of the mobile phone base station: case study].

Determination of exposure due to mobile phone base stations in an epidemiological study.

Variographic analysis of public exposure to electromagnetic radiation due to cellular base stations.

[Subjective symptoms reported by people living in the vicinity of cellular phone base stations: review].

Neurobehavioral effects among inhabitants around mobile phone base stations.

[Assessment of electromagnetic fields intensity emitted by cellular phone base stations in surrounding flats--a preliminary study].

Effects of short-term W-CDMA mobile phone base station exposure on women with or without mobile phone related symptoms.

Statistical analysis of electromagnetic radiation measurements in the vicinity of indoor microcell GSM/UMTS base stations in Serbia.

Estimates of Environmental Exposure to Radiofrequency Electromagnetic Fields and Risk of Lymphoma Subtypes.

[Level of microwave radiation from mobile phone base stations built in residential districts].

Do mobile phone base stations affect sleep of residents? Results from an experimental double-blind sham-controlled field study.

Effect of electromagnetic radiations from mobile phone base stations on general health and salivary function.

Use of portable exposure meters for comparing mobile phone base station radiation in different types of areas in the cities of Basel and Amsterdam.

[Investigation on the health of people living near mobile telephone relay stations: I/Incidence according to distance and sex].

Statistical analysis of electromagnetic radiation measurements in the vicinity of GSM/UMTS base station antenna masts.

Impact of radiofrequency radiation on DNA damage and antioxidants in peripheral blood lymphocytes of humans residing in the vicinity of mobile phone base stations.

Analysis of the effect of mobile phone base station antenna loading on localized SAR and its consequences for measurements.

Public safety assessment of electromagnetic radiation exposure from mobile base stations.

Survey of RF exposure levels from mobile telephone base stations in Australia.

Subjective complaints of people living near mobile phone base stations in Poland.

Time averaged transmitter power and exposure to electromagnetic fields from mobile phone base stations.

[Symptoms experienced by people in vicinity of base stations: II/ Incidences of age, duration of exposure, location of subjects in relation to the antennas and other electromagnetic factors].

[Danger of cellular telephones and their relay stations].

Mobile phone base stations and early childhood cancers: case-control study.

Exposure to non-ionizing electromagnetic radiation from mobile telephony and the association with psychiatric symptoms.

Feasibility of future epidemiological studies on possible health effects of mobile phone base stations.

Electromagnetic fields from mobile phone base station - variability analysis.

Assessment of RF radiation levels in the vicinity of 60 GSM mobile phone base stations in Iran.

Biological responses of mobile phone frequency exposure.

Radio frequency electromagnetic field compliance assessment of multi-band and MIMO equipped radio base stations.

A possible effect of electromagnetic radiation from mobile phone base stations on the number of breeding house sparrows (Passer domesticus).

Statistical analysis of electromagnetic radiation measurements in the vicinity of GSM/UMTS base station installed on buildings in Serbia.

A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.

Does short-term exposure to mobile phone base station signals increase symptoms in individuals who report sensitivity to electromagnetic fields? A double-blind randomized provocation study.

Modeled and Perceived Exposure to Radiofrequency Electromagnetic Fields From Mobile-Phone Base Stations and the Development of Symptoms Over Time in a General Population Cohort.

Determination of safety distance limits for a human near a cellular base station antenna, adopting the IEEE standard or ICNIRP guidelines.

Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations.

Occupational exposure to base stations-compliance with EU directive 2004/40/EC.

Measurement and analysis of radiofrequency radiations from some mobile phone base stations in Ghana.

UMTS base station-like exposure, well-being, and cognitive performance.

Outdoor and indoor sources of residential radiofrequency electromagnetic fields, personal cell phone and cordless phone use, and cognitive function in 5-6 years old children.

[Increased occurrence of nuclear cataract in the calf after erection of a mobile phone base station].

Impact of a small cell on the RF-EMF exposure in a train.

A large-scale measurement, analysis and modelling of electromagnetic radiation levels in the vicinity of GSM/UMTS base stations in an urban area.

Determinants of exposure to electromagnetic fields from mobile phones.

Improving the efficiency of measurement procedures for assessing human exposure in the vicinity of mobile phone (GSM/DCS/UMTS) base stations.

Population exposure to electromagnetic fields generated by radio base stations: evaluation of the urban background by using provisional model and instrumental measurements.

On the safety assessment of human exposure in the proximity of cellular communications base-station antennas at 900, 1800 and 2170 MHz.

What input data are needed to accurately model electromagnetic fields from mobile phone base stations?

Methods of evaluating human exposure to electromagnetic fields radiated from operating base stations in Korea.

Non-specific physical symptoms in relation to actual and perceived proximity to mobile phone base stations and powerlines.

[Mobile communication: radiobiology problems and evaluation of danger].

Temporal and spatial variability of personal exposure to radio frequency electromagnetic fields.

The precautionary principle in the context of mobile phone and base station radiofrequency exposures.

Adolescents' risk perceptions on mobile phones and their base stations, their trust to authorities and incivility in using mobile phones: a cross-sectional survey on 2240 high school students in Izmir, Turkey.

Systematic review on the health effects of exposure to radiofrequency electromagnetic fields from mobile phone base stations.

Radiofrequency electromagnetic fields emitted from base stations of DECT cordless phones and the risk of glioma and meningioma (Interphone Study Group, Germany).

Mobile telecommunications and health: report of an investigation into an alleged cancer cluster in Sandwell, West Midlands.

Study of variations of radiofrequency power density from mobile phone base stations with distance.

Health risks from the use of mobile phones.

Mobile phones, mobile phone base stations and cancer: a review.

Animal carcinogenicity studies on radiofrequency fields related to mobile phones and base stations.

Association of Exposure to Radio-Frequency Electromagnetic Field Radiation (RF-EMFR) Generated by Mobile Phone Base Stations with Glycated Hemoglobin (HbA1c) and Risk of Type 2 Diabetes Mellitus.

Assessment of the temporal trend of the exposure of people to electromagnetic fields produced by base stations for mobile telephones.

Risk and benefit perceptions of mobile phone and base station technology in Bangladesh.

Clinically defined non-specific symptoms in the vicinity of mobile phone base stations: A retrospective before-after study.

Specific absorption rate and electric field measurements in the near field of six mobile phone base station antennas.

Output power levels from mobile phones in different geographical areas; implications for exposure assessment.

Influence of mobile phone traffic on base station exposure of the general public.

Residential exposure to RF-EMF from mobile phone base stations: Model predictions versus personal and home measurements.

Physics and biology of mobile telephony.

Longitudinal associations between risk appraisal of base stations for mobile phones, radio or television and non-specific symptoms.

Exposure assessment of mobile phone base station radiation in an outdoor environment using sequential surrogate modeling.

Exposure of farm workers to electromagnetic radiation from cellular network radio base stations situated on rural agricultural land.

Mobile phones. precautionary options.

Aggregated data from two double-blind base station provocation studies comparing individuals with idiopathic environmental intolerance with attribution to electromagnetic fields and controls.

GSM base stations: short-term effects on well-being.

Sensitivity to electricity--temporal changes in Austria.

How does long term exposure to base stations and mobile phones affect human hormone profiles?

Effects of exposure to GSM mobile phone base station signals on salivary cortisol, alpha-amylase, and immunoglobulin A.

Determinants and stability over time of perception of health risks related to mobile phone base stations.

[Cellular radio systems. Problems faced in assessing exposure to electromagnetic fields].

Effect of radiofrequency radiation on reproductive health.

Exposure assessment in front of a multi-band base station antenna.

Protect children from EMF.

Symptoms of ill health ascribed to electromagnetic field exposure--a questionnaire survey.

Spatial electromagnetic field intensity modelling of global system for mobile communication base stations in the Istanbul Technical University Ayazaga campus area.

[Protection against electromagnetic fields emitted by mobile phone facilities in Poland and the European Union countries].

Prevalence of nuclear cataract in Swiss veal calves and its possible association with mobile telephone antenna base stations.

[GSM fields and health: an updated literature review].

Knowledge and perceptions of the health effects of environmental hazards in the general population in Italy.

Dynamics of the public concern and risk communication program implementation.

Microwaves in the cold war: the Moscow embassy study and its interpretation. Review of a retrospective cohort study.

Health effects of living near mobile phone base transceiver station (BTS) antennae: a report from Isfahan, Iran.

Replication of heart rate variability provocation study with 2.4-GHz cordless phone confirms original findings.

[Reports on the impact of objects emitting electromagnetic fields on the environment: issues concerning their better understanding by non-specialists in telecommunication].

Cognitive and physiological responses in humans exposed to a TETRA base station signal in relation to perceived electromagnetic hypersensitivity.

Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais state, Brazil.

Are wireless phones safe? A review of the issue.

A novel method to assess human population exposure induced by a wireless cellular network.

[Metrology of pulse modulated electromagnetic fields with diode-type meters].

Radiofrequency radiation injures trees around mobile phone base stations.

Comparison of temporal realistic telecommunication base station exposure with worst-case estimation in two countries.

[Cellular telephones and their relay stations: a health risk?].

[Evaluation of the levels of radiofrequency electromagnetic fields in the territory of the city of Bari in outside and inside environments].

A geographical model of radio-frequency power density around mobile phone masts.

[Ecological aspects of electromagnetic radiation emitted by mobile stations of communication means].

Assessment of nuclear abnormalities in exfoliated cells from the oral epithelium of mobile phone users.

Occupational exposure to radiofrequency fields in antenna towers.

Joint minimization of uplink and downlink whole-body exposure dose in indoor wireless networks.

--Leaf Cluster 19 (84)

Theme - Electromagnetic hypersensitivity

Titles

Does electromagnetic hypersensitivity originate from nocebo responses? Indications from a qualitative study.

Symptoms, personality traits, and stress in people with mobile phone-related symptoms and electromagnetic hypersensitivity.

Electromagnetic field induced biological effects in humans.

Development and evaluation of the electromagnetic hypersensitivity questionnaire.

Hypothesis on how to measure electromagnetic hypersensitivity.

[Subjective non-specific symptoms related with electromagnetic fields: description of 2 cases].

Characteristics of perceived electromagnetic hypersensitivity in the general population.

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Cognitive and neurobiological alterations in electromagnetic hypersensitive patients: results of a case-control study.

Becoming electro-hypersensitive: A replication study.

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Electromagnetic hypersensitivity: fact or fiction?

IEI-EMF provocation case studies: A novel approach to testing sensitive individuals.

A systematic review of treatments for electromagnetic hypersensitivity.

[Hypersensitivity syndrome].

Electromagnetic hypersensitivity: a systematic review of provocation studies.

Symptom attribution and risk perception in individuals with idiopathic environmental intolerance to electromagnetic fields and in the general population.

Polluted places or polluted minds? An experimental sham-exposure study on background psychological factors of symptom formation in 'Idiophatic Environmental Intolerance attributed to electromagnetic fields'.

Is There a Connection Between Electrosensitivity and Electrosensibility? A Replication Study.

Idiopathic environmental intolerance attributed to electromagnetic fields (IEI-EMF): a systematic review of identifying criteria.

Electromagnetic hypersensitivity (EHS) in occupational and primary health care: A nation-wide survey among general practitioners, occupational physicians and hygienists in the Netherlands.

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Blood laboratory findings in patients suffering from self-perceived electromagnetic hypersensitivity (EHS).

Representative survey on idiopathic environmental intolerance attributed to electromagnetic fields in Taiwan and comparison with the international literature.

Can explicit suggestions about the harmfulness of EMF exposure exacerbate a nocebo response in healthy controls?

Idiopathic environmental intolerance attributed to electromagnetic fields: a content analysis of British newspaper reports.

A cognitive-behavioral treatment of patients suffering from "electric hypersensitivity". Subjective effects and reactions in a double-blind provocation study.

Development and evaluation of an electromagnetic hypersensitivity questionnaire for Japanese people.

Increasing levels of saliva alpha amylase in electrohypersensitive (EHS) patients.

Are media reports able to cause somatic symptoms attributed to WiFi radiation? An experimental test of the negative expectation hypothesis.

Electrical hypersensitivity in humans--fact or fiction?

Electromagnetic hypersensitivity: evidence for a novel neurological syndrome.

Effect of short exposure to radiofrequency electromagnetic fields on saliva biomarkers: a study on the electrohypersensitive individuals.

[Electromagnetic fields hypersensitivity].

Heavy metal exposure in patients suffering from electromagnetic hypersensitivity.

[Controversies around electromagnetic fields and electromagnetic hypersensitivity. The construction of "low noise" public problems].

Coping and self-image in patients with visual display terminal-related skin symptoms and perceived hypersensitivity to electricity.

Neurophysiological effects of flickering light in patients with perceived electrical hypersensitivity.

Effects of personalised exposure on self-rated electromagnetic hypersensitivity and sensibility - A double-blind randomised controlled trial.

Psychologic aspects of patients with symptoms presumed to be caused by electricity or visual display units.

Odontologic survey of referred patients with symptoms allegedly caused by electricity or visual display units.

Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder.

[Idiopathic environmental intolerance: 2 disabling entities to recognize].

Neurophysiological study of patients with perceived 'electrical hypersensitivity'.

Description of persons with symptoms presumed to be caused by electricity or visual display units--oral aspects.

Provocation with stress and electricity of patients with "sensitivity to electricity".

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Association of tinnitus and electromagnetic hypersensitivity: hints for a shared pathophysiology?

The microwave syndrome or electro-hypersensitivity: historical background.

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Altered cortical excitability in subjectively electrosensitive patients: results of a pilot study.

An assessment of the autonomic nervous system in the electrohypersensitive population: a heart rate variability and skin conductance study.

Provocation of the electromagnetic distress syndrome.

Skin problems from visual display units. Provocation of skin symptoms under experimental conditions.

Cognitive behavioural therapy for patients with electric sensitivity - a multidisciplinary approach in a controlled study.

Self-reporting of symptom development from exposure to radiofrequency fields of wireless smart meters in victoria, australia: a case series.

Improvement of gastroesophageal reflux symptoms after radiofrequency energy: a randomized, sham-controlled trial.

Does "electromagnetic pollution" cause illness? An inquiry among Austrian general practitioners.

"Struggle to obtain redress": Women's experiences of living with symptoms attributed to dental restorative materials and/or electromagnetic fields.

Environmental illness: fatigue and cholinesterase activity in patients reporting hypersensitivity to electricity.

The views of primary care physicians on health risks from electromagnetic fields.

Review of extensive workups of 34 patients overexposed to radiofrequency radiation.

Providing cell phone numbers and e-mail addresses to patients: The patient's perspective, a cross sectional study.

[Effects of millimetric electromagnetic waves on regional blood flow and effectiveness of multimodal therapy of patients with pulmonary tuberculosis].

Environmental illness: evaluation of salivary flow, symptoms, diseases, medications, and psychological factors.

30-MINUTES-TUMT. Use of the visual analogue scale to investigate patients' pain perception, different cocktail options and tolerability during 30 minutes' treatment.

Non-ionizing radiation exposure causing ill-health and alopecia areata.

Low-frequency pulsed electromagnetic field therapy in fibromyalgia: a randomized, double-blind, sham-controlled clinical study.

Electrohypersensitivity: a functional impairment due to an inaccessible environment.

Accidental exposure to electromagnetic fields from the radar of a naval ship: a descriptive study.

[Mechanism of biotropic effects of regional electromagnetic fields in patients with left ventricular ischemic dysfunction].

A comparision of percutaneous radiofrequency trigeminal neurolysis and microvascular decompression of the trigeminal nerve for the treatment of tic douloureux.

Medical aspects of radiofrequency radiation overexposure.

Prospective, randomized, single-blind, sham treatment-controlled study of the safety and efficacy of an electromagnetic field device for the treatment of chronic low back pain: a pilot study.

Atrial fibrillation therapies: lest we forget surgery.

Non-resection approaches for colorectal liver metastases.

[Indices of thrombocyte conductance and permeability in microwave fields in ischemic and hemorrhagic stroke patients].

Health care utilisation and attitudes towards health care in subjects reporting environmental annoyance from electricity and chemicals.

A primer of magnetic stimulation as a tool for neuropsychology.

--Leaf Cluster 43 (202)

Theme - Health risks from low-frequency electromagnetic fields

Titles

Health risks of electromagnetic fields. Part I: Evaluation and assessment of electric and magnetic fields.

The Bernal Lecture 2004 Are low-frequency electromagnetic fields a health hazard?

Electric and magnetic fields (EMF): what do we know about the health effects?

EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses.

[Non-thermal bioeffects of static and extremely low frequency electromagnetic fields].

Effects of extremely low frequency electromagnetic fields on health.

Exposure assessment for power frequency electric and magnetic fields (EMF) and its application to epidemiologic studies.

The question of health effects from exposure to electromagnetic fields.

[Influence of low frequency electromagnetic fields on the nervous system].

Biological responses to electromagnetic fields.

Teratogen update: electromagnetic fields.

Intrauterine effects of electromagnetic fields--(low frequency, mid-frequency RF, and microwave): review of epidemiologic studies.

The effects of electromagnetic fields from power lines on avian reproductive biology and physiology: a review.

The effects of extremely low-frequency magnetic fields on melatonin and cortisol, two marker rhythms of the circadian system.

The sensitivity of children to electromagnetic fields.

Electromagnetic fields and cancer: the cost of doing nothing.

Biological effects from electromagnetic field exposure and public exposure standards.

[Low frequency electromagnetic fields in the working environment--exposure and health effects. Elevated risk of cancer, reproductive hazards or other unwanted health effects?].

Designing EMF experiments: what is required to characterize "exposure"?

Electromagnetic fields and public health.

[Current state of knowledge on health and electromagnetic fields].

Health risks associated with residential exposure to extremely low frequency electromagnetic radiation.

Electromagnetic fields and health outcomes.

[Biological mechanisms and health effects of emf in view of requirements of reports on the impact of various installations on the environment].

[Exposure to low-frequency electromagnetic fields and pregnancy outcome: a review of the literature with particular attention to exposure to video terminals].

Fielding a current idea: exploring the public health impact of electromagnetic radiation.

A literature review: the cardiovascular effects of exposure to extremely low frequency electromagnetic fields.

Exposure to low-frequency electromagnetic fields--a health hazard?

Human disease resulting from exposure to electromagnetic fields.

Electromagnetic radiation.

Future needs of occupational epidemiology of extremely low frequency electric and magnetic fields: review and recommendations.

Comparative health risk assessment of electromagnetic fields.

[Effects of electromagnetic fields on health].

WHO health risk assessment process for static fields.

A review of cancer induction by extremely low frequency electromagnetic fields. Is there a plausible mechanism?

Reproductive and teratologic effects of electromagnetic fields.

Developing policy in the face of scientific uncertainty: interpreting 0.3 microT or 0.4 microT cutpoints from EMF epidemiologic studies.

[Electromagnetic fields--effects on health].

Biologic effects and health consequences of low and high (radio) frequency electromagnetic fields.

[Electromagnetic pollution (electrosmog)--potential hazards of our electromagnetic future].

Risk governance for mobile phones, power lines, and other EMF technologies.

[Bioeffects of electromagnetic fields--safety limits of each frequency band, especially less than radio one].

Setting prudent public health policy for electromagnetic field exposures.

Effects of extremely low frequency electromagnetic field on the health of workers in automotive industry.

Electromagnetic fields: low dose exposure, current update.

Effects of extremely low frequency electromagnetic fields on distortion product otoacoustic emissions in rabbits.

Exposure to extremely-low-frequency electromagnetic fields and radiofrequency radiation: cardiovascular effects in humans.

Effects of electromagnetic field exposure on the heart: a systematic review.

Current Understanding of the Health Effects of Electromagnetic Fields.

Personal digital assistant (PDA) cell phone units produce elevated extremely-low frequency electromagnetic field emissions.

Biological effects of low frequency electromagnetic fields.

Exposure to extremely low frequency electromagnetic fields during pregnancy and the risk of spontaneous abortion: a case-control study.

[Neurotic disturbances, depression and anxiety disorders in the population living in the vicinity of overhead high-voltage transmission line 400 kV. Epidemiological pilot study].

The effect of extremely low frequency electromagnetic fields on pregnancy and fetal growth, and development.

The Effects of Electromagnetic Field on the Endocrine System in Children and Adolescents.

EMF and current cancer concepts.

Attitudes about electric and magnetic fields: do scientists and other risk experts perceive risk similarly?

[Effects of extremely low frequency electromagnetic radiation on cardiovascular system of workers].

Perception of health risks of electromagnetic fields by MRI radiographers and airport security officers compared to the general Dutch working population: a cross sectional analysis.

[Electromagnetic poles and reproduction].

[Biophysical mechanisms of electromagnetic fields interaction and health effects].

Resveratrol may reverse the effects of long-term occupational exposure to electromagnetic fields on workers of a power plant.

ELF noise fields: a review.

[The health risks of exposure to electromagnetic fields in work environments].

Scientific panel on electromagnetic field health risks: consensus points, recommendations, and rationales.

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Study of extremely low frequency electromagnetic fields in infant incubators.

Intensity-time dependence dosing criterion in the EMF exposure guidelines in Russia.

[Electromagnetic fields: their biological effects and regulation].

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Combined effects of electromagnetic fields on immune and nervous responses.

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Effects of noise and electromagnetic fields on reproductive outcomes.

Health and safety implications of exposure to electromagnetic fields in the frequency range 300 Hz to 10 MHz.

The role of electromagnetic fields in neurological disorders.

Biophysical estimation of the environmental importance of electromagnetic fields.

[Biological effects of electromagnetic fields].

Effects of electromagnetic fields exposure on plasma hormonal and inflammatory pathway biomarkers in male workers of a power plant.

The epidemiology of exposure to electromagnetic fields: an overview of the recent literature.

Microwave electromagnetic field regulates gene expression in T-lymphoblastoid leukemia CCRF-CEM cell line exposed to 900 MHz.

Effects of 60 Hz electromagnetic field exposure on APP695 transcription levels in differentiating human neuroblastoma cells.

Ambiguous evidence and institutional interpretation: an alternative view of electric and magnetic fields.

[Combined biological effect of electromagnetic fields and chemical substances (toxic)].

EMFs: cutting through the controversy.

The effect of chronic exposure to extremely low-frequency electromagnetic fields on sleep quality, stress, depression and anxiety.

[Norms and standards for radiofrequency electromagnetic fields in Latin America: guidelines for exposure limits and measurement protocols].

Health risk assessment of electromagnetic fields: a conflict between the precautionary principle and environmental medicine methodology.

[Constant low-frequency electrical and electromagnetic fields (biological action and hygienic evaluation)].

[Electrical field exposure and human health. Risk assessment and problems relative to bureaucratic procedures and to the role of instituitional organizations in control and prevention].

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Electromagnetic fields enhance chemically-induced hyperploidy in mammalian oocytes.

Electromagnetic field exposure assessment in Europe radiofrequency fields (10 MHz-6 GHz).

Exposure of the critically ill patient to extremely low-frequency electromagnetic fields in the intensive care environment.

Biological effects of electromagnetic fields on vertebrates. A review.

Electromagnetic effects on people.

Time-dependent hematological changes in workers exposed to electromagnetic fields.

Characterisation of exposure to non-ionising electromagnetic fields in the Spanish INMA birth cohort: study protocol.

Electromagnetic fields in neonatal incubators: the reasons for an alert.

[Electromagnetic fields and people's health].

Electromagnetic fields: mechanism, cell signaling, other bioprocesses, toxicity, radicals, antioxidants and beneficial effects.

Project NEMESIS: perception of a 50 Hz electric and magnetic field at low intensities (laboratory experiment).

Non-ionising electromagnetic environments on manned spacecraft.

Health hazards and electromagnetic fields.

The design, construction and calibration of a carefully controlled source for exposure of mammalian cells to extremely low-frequency electromagnetic fields.

[HEALTH STATUS OF ELECTROTECHNICAL PERSONNEL EXPOSED TO THE COMBINED IMPACT OF ELECTROMAGNETIC FIELDS OF 50 HZ AND CHEMICALS].

EMF recommendations specific for children?

[Electromagnetic fields emitted in radio- and microwave- frequency range: equipment and methods for the environment protection and survey measurements].

Health-Economics Analyses Applied to ELF Electric and Magnetic Fields.

Alterations in human EEG activity caused by extremely low frequency electromagnetic fields.

Adverse human reproductive outcomes and electromagnetic fields: a brief summary of the epidemiologic literature.

Nonionizing electromagnetic fields and cancer: a review.

Recommended minimal requirements and development guidelines for exposure setups of bio-experiments addressing the health risk concern of wireless communications.

Genetic damage in mammalian somatic cells exposed to extremely low frequency electro-magnetic fields: a meta-analysis of data from 87 publications (1990-2007).

Health effects of low-level electromagnetic fields: phantom or not-so-phantom risk?

"Dirty electricity": what, where, and should we care?

Basic problems of diversely reported biological effects of radio frequency fields.

Investigation of the spinal cord as a natural receptor antenna for incident electromagnetic waves and possible impact on the central nervous system.

Assessment of electromagnetic field levels from surrounding high-tension overhead power lines for proposed land use.

Effects of electromagnetic fields on photophasic circulating melatonin levels in American kestrels.

How dangerous are mobile phones, transmission masts, and electricity pylons?

Understanding the effects of electromagnetic field emissions from Marine Renewable Energy Devices (MREDs) on the commercially important edible crab, Cancer pagurus (L.).

Actual and perceived exposure to electromagnetic fields and non-specific physical symptoms: an epidemiological study based on self-reported data and electronic medical records.

Is newborn melatonin production influenced by magnetic fields produced by incubators?

Does exposure to environmental radiofrequency electromagnetic fields cause cognitive and behavioral effects in 10-year-old boys?

Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on nonchemical factors.

Public health hazards from electricity-producing plants.

[Saccharomyces cerevisiae as a model organism for studying the carcinogenicity of non-ionizing electromagnetic fields and radiation].

How to approach complex mixtures: lessons from the epidemiology of electromagnetic fields.

[The role of chemical and physical factors in cancer development].

[Possible outer hair cells hazards from occupational exposure to very low frequency electric and magnetic fields: a pilot study].

Effects of electromagnetic fields on the reproductive success of American kestrels.

A 50-Hz electromagnetic field impairs sleep.

A structured literature review for risk assessment: EMF and human health risk.

Is MRI imaging in pediatric age totally safe? A critical reprisal.

Health effects of microwave exposures: a review of the recent (1995-1998) literature.

Epidemiological studies of human exposures to radiofrequency radiation. A critical review.

Human adverse reproductive outcomes and electromagnetic field exposures: review of epidemiologic studies.

The infant incubator in the neonatal intensive care unit: unresolved issues and future developments.

Biologic effects of low-level electromagnetic fields: current issues and controversies.

[Non-thermal electromagnetic fields and estimation of the convulsive syndrome probable development].

[The precautionary principle: scientific evidence and decision processes].

Study of self-reported hypersensitivity to electromagnetic fields in California.

Occupational EMF exposure from radar at X and Ku frequency band and plasma catecholamine levels.

Clustering of excess health concerns for electromagnetic fields among health personnel: A quantitative and qualitative approach.

Biological effects of environmental electromagnetic fields: molecular mechanisms.

[Biological effects of exposure to electromagnetic fields: introduction].

Effects of low-level radio-frequency (3kHz to 300GHz) energy on human cardiovascular, reproductive, immune, and other systems: a review of the recent literature.

Human performance and physiology: a statistical power analysis of ELF electromagnetic field research.

Psychological studies in nonionizing electromagnetic energy research.

Potential emotional and cognitive disorders associated with exposure to EMFs. A review.

Electric power plant emissions and public health.

Electromagnetic fields produced by incubators influence heart rate variability in newborns.

Study of human neurovegetative and hematologic effects of environmental low-frequency (50-Hz) electromagnetic fields produced by transformers.

[The influence of occupational environment and professional factors on the risk of cardiovascular disease].

Synergistic health effects between chemical pollutants and electromagnetic fields.

Effect of short-term 50 Hz electromagnetic field exposure on the behavior of rats.

Estimating air pollution and health loss embodied in electricity transfers: An inter-provincial analysis in China.

Alternative functional relationships between ELF field exposure and possible health effects: report on an expert workshop.

[Impact of electromagnetic fields on a computer user].

Mechanisms of electromagnetic interaction with cellular systems.

Opinion on potential health effects of exposure to electromagnetic fields.

Electromagnetic hypersensitivity: biological effects of dirty electricity with emphasis on diabetes and multiple sclerosis.

The "Moscow signal" epidemiological study, 40 years on.

Effects of EMF emissions from undersea electric cables on coral reef fish.

Exposure to electric power generator noise among small scale business operators in selected communities in Ibadan, Nigeria.

Northern cardiometeopathies.

EMF-cancer link: the ferritin hypothesis.

Effect of occupational EMF exposure from radar at two different frequency bands on plasma melatonin and serotonin levels.

Dirty electricity, chronic stress, neurotransmitters and disease.

[The perceptibility of a microwave field under experimental conditions].

Women growing older with environmental sensitivities: A grounded theory model of meeting one's needs.

A perspective on environmental health in the USSR: research and practice.

[Clinical variants of the disease caused by exposure to radio-frequency electromagnetic fields].

Work environment and cardiovascular diseases. A short review of the literature.

Noise, impulse noise, and other physical factors: combined effects on hearing.

[Characteristics of electromagnetic situation in Far North regions].

Fifty Hertz electromagnetic field exposure stimulates secretion of beta-amyloid peptide in cultured human neuroglioma.

[Ethical values in the regulation of the exposure to electromagnetic fields].

[Ecological significance of electromagnetic fields: the 20th century--century of electricity, the 21st--century of magnetism].

[Video display terminals: their electromagnetic safety].

Male proportion in offspring of parents exposed to strong static and extremely low-frequency electromagnetic fields in Norway.

Video display terminals: risk of electromagnetic radiation.

[The evaluation of the exposure of seamstresses to electromagnetic fields, emitted by sewing machines].

Scientometric study of the effects of exposure to non-ionizing electromagnetic fields on fertility: A contribution to understanding the reasons of partial failure.

[Influences of solar and geomagnetic activity on health status of people with various nosological forms of diseases].

Earthing: health implications of reconnecting the human body to the Earth's surface electrons.

Prevalence of annoyance attributed to electrical equipment and smells in a Swedish population, and relationship with subjective health and daily functioning.

Fetal loss associated with two seasonal sources of electromagnetic field exposure.

Use of kappa statistic in determining validity of quality filtering for meta-analysis: a case study of the health effects of electromagnetic radiation.

[Health risks from the use of NMR tomography and in vivo NMR spectroscopy].

Geomagnetics and society interact in weekly and broader multiseptans underlying health and environmental integrity.

[Contribution of physical factors to the complex anthropogenic load in an industrial town].

Evidence that dirty electricity is causing the worldwide epidemics of obesity and diabetes.

Possible effects of electric blankets and heated waterbeds on fetal development.

Environmental variables and the risk of disease.

[The use of geographic information technologies in the sanitary control of an environmental electromagnetic field].

[Personal computer: physical factors, effect on the user].

A low cost, re-usable electricity-free infant warmer: evaluation of safety, effectiveness and feasibiliy.

Iatrogenic environmental hazards in the neonatal intensive care unit.

Space weather and human deaths distribution: 25 years' observation (Lithuania, 1989-2013).

Acute myocardial infarction (AMI) (n-11026) on days of zero geomagnetic activity (GMA) and the following week: differences at months of maximal and minimal solar activity (SA) in solar cycles 23 and 24.

Current strategies in the management of atrial fibrillation.

--Leaf Cluster 33 (91)

Theme - Health risks to workers in different occupations

Titles

[Levels of occupational exposure to extremely low frequency magnetic fields among workers in different jobs].

[Occupational exposure to 50 Hz magnetic fields in workers employed in various jobs].

[Exposure to electromagnetic fields with frequencies of 50 Hz and changes in the circulatory system in workers at electrical power stations].

Absenteeism and mortality of workers exposed to electromagnetic fields in the French Electricity Company.

[Evaluation of selected parameters of circulatory system function in various occupational groups exposed to high frequency electromagnetic fields. II. Electrocardiographic changes].

[Fitness of workers with particular sensitivity to non-ionizing radiation].

Health of workers exposed to electric fields.

[Health protection of workers occupationally exposed to effects of electromagnetic fields in Poland and in the European Union member states].

[Occupational exposure to electromagnetic fields of extremely low frequency (with particular regard to power plants) and the health status of workers, based on a literature review].

A biomonitoring study of genotoxic risk to workers of transformers and distribution line stations.

[Health effects of occupational exposure to electromagnetic fields in view of studies performed in Poland and abroad].

[Evaluation of the genotoxicity of the extremely low frequency-magnetic fields (ELF-MF) in workers exposed for professional reasons].

[Evaluation of selected parameters of circulatory system function in various occupational groups of workers exposed to high frequency electromagnetic fields].

[Health status of railway workers using magnetic powder flaw detectors].

[Health effects of occupational exposure to static magnetic fields used in magnetic resonance imaging: a review].

Health problems among workers of iron welding machines: an effect of electromagnetic fields.

[Health and work ability of workers of the electricity sector in Sao Paulo].

[Hygienic assessment of working conditions and functional resistance in electric power station workers].

Should the threshold limit value for power frequency (60 Hz) magnetic fields be changed? Perceptions among scientists and other risk experts.

Prevalence of depression among electrical workers.

[Health status of the workers exposed to strong, constant magnetic fields].

[Observations of changes in neurobehavioral functions in workers exposed to high-frequency radiation].

Occupational exposure to electromagnetic fields of uninterruptible power supply industry workers.

Neurovegetative disturbances in workers exposed to 50 Hz electromagnetic fields.

[Possible consequence on measures for the protection of electromagnetic fields exposed workers].

Extremely low frequency-magnetic fields (ELF-EMF) occupational exposure and natural killer activity in peripheral blood lymphocytes.

Evaluation of chromosomal alteration in electrical workers occupationally exposed to low frequency of electro magnetic field (EMFs) in Coimbatore population, India.

Injuries among electric power industry workers, 1995-2013.

Health status of personnel occupationally exposed to radiowaves.

[Effect of exposure to extremely low-frequency electromagnetic fields on liver function of workers].

[Evaluation of selected functional circulation parameters of workers from various occupational groups exposed to electromagnetic fields of high frequency. III. 24-h monitoring of arterial blood pressure (ABP)].

Depression in high voltage power line workers.

[A methodological approach to studying the values of 50-Hz electromagnetic fields that influence the workers of power enterprises].

Guidance note: risk management of workers with medical electronic devices and metallic implants in electromagnetic fields.

ECG changes in factory workers exposed to 27.2 MHz radiofrequency radiation.

Low-back pain among electric power supply workers and their attitude toward its prevention and the treatment.

Exposure to high-frequency transient electromagnetic fields.

[Exposure to VHF and UHF electromagnetic fields among workers employed in radio and TV broadcast centers. I. Assessment of exposure].

Heart rate variability (HRV) analysis in radio and TV broadcasting stations workers.

[Functional status of workers engaged in connecting high-voltage electric power lines].

[Offshore substation workers' exposure to harmful factors - Actions minimizing risk of hazards].

[Medical and biologic research of electromagnetic fields in radiofrequencies range. Results and prospects].

[Health surveillance guidelines after the European directive on electromagnetic fields].

Monitoring of people and workers exposure to the electric, magnetic and electromagnetic fields in an Italian National Cancer Institute.

[Risk of electromagnetic fields in electric power stations and substations of a petrochemical plant].

An analysis of fatal and non-fatal injuries and injury severity factors among electric power industry workers.

[Hygienic optimization of the use of chemical protective means on railway transport].

[High-frequency electromagnetic field exposure on reproductive and endocrine functions of female workers].

Assessment of levels of occupational exposure to workers in radiofrequency fields of two television stations in Accra, Ghana.

[Evaluation of vital activity of workers with obliterating diseases of lower extremities servicing electric transmission lines].

[Occupational health evaluation of electromagnetic fields in electric trains and subway technologic areas].

[Reports on electromagnetic field strength measurements issued for occupational health and safety needs in the opinion of radio communication station users].

Health problems among operators of plastic welding machines and exposure to radiofrequency electromagnetic fields.

[Various psychological parameters in subjects occupationally exposed to radiofrequencies].

Ocular medical surveillance on microwave and laser workers.

Evaluation of non ionizing radiation around the dielectric heaters and sealers: a case report.

The psychosocial work environment and skin symptoms among visual display terminal workers: a case referent study.

The strategy of targetted health surveillance. II. Genetically determined susceptibility to chemical substances and other issues related to health surveillance.

[Screen dermatitis and visual display units].

[Occupational risks in grocery stores].

Health Effects of Electromagnetic Fields on Reproductive-Age Female Operators of Plastic Welding Machines in Fuzhou, China.

Reproductive hazards among workers at high voltage substations.

[Evaluation of various psychologic parameters in a group of workers occupationally exposed to radiofrequency].

Occupational influences on male fertility and sexuality. I.

[Radiation safety at atomic electric power stations].

[Effect of wide-band modulated electromagnetic fields on the workers of high-frequency telephone exchanges].

[Effect of ultra high frequency electromagnetic waves and lead on the workers' health; phytotherapy of the disorders].

Biosomatic effects of the electromagnetic fields on view of the physiotherapy personnel health.

[On prevention of electromagnetic rays effects in workers exposed to extreme climate conditions].

Microwave sickness: a reappraisal.

Building an index of activity of inhabitants from their activity on the residential electrical power line.

Radiofrequency electromagnetic leakage fields from plastic welding machines. Measurements and reducing measures.

Rate of change of frequency under line contingencies in high voltage electric power networks with uncertainties.

Risk-management and risk-analysis-based decision tools for attacks on electric power.

Exposure from occupational versus other sources.

Occupational exposure of herbicide applicators to herbicides used along electric power transmission line right-of-way.

[Clinical monitoring in areas of exposure to radiofrequency electromagnetic fields].

Electromagnetic noise superimposed on the electric power supply to electronic medical equipment.

Cardiovascular risk in operators under radiofrequency electromagnetic radiation.

Occupational exposure to physical agents: the new Italian database for risk assessment and control.

Erratic electricity supply (Dumsor) and anxiety disorders among university students in Ghana: a cross sectional study.

[A survey on diabetes mellitus in the staff of electric power system in Baotou city].

Correction: The effects of electric power lines on the breeding ecology of greater sage-grouse.

[Risk of electromagnetic fields in control board and switchboard rooms at petrochemical plants].

Biomonitoring of 20 trace elements in blood and urine of occupationally exposed workers by sector field inductively coupled plasma mass spectrometry.

Effects of atmospheric electricity on some substrates of disordered social behavior.

Electricity prices in Italy: Data registered during photovoltaic activity interval.

[Dermatitis in VDT operators: a review of the literature].

Black sky: Exposing electricity as the Achilles' heel of resilience.

[Danger of electricity in the bathtub].

The role of microwave radiometry in carotid artery disease. Diagnostic and clinical prospective.

--Leaf Cluster 36 (84)

Theme - Precautionary measures to reduce potential EMF health risks

Titles

Workgroup report: base stations and wireless networks-radiofrequency (RF) exposures and health consequences.

Recent advances in research on radiofrequency fields and health: 2004-2007.

Recent advances in research on radiofrequency fields and health: 2001-2003.

International and national expert group evaluations: biological/health effects of radiofrequency fields.

Low-level exposure to radiofrequency electromagnetic fields: health effects and research needs.

Public responses to precautionary information from the Department of Health (UK) about possible health risks from mobile phones.

Health risks of electromagnetic fields. Part II: Evaluation and assessment of radio frequency radiation.

The precautionary principle and risk perception: experimental studies in the EMF area.

[Autoimmune processes after long-term low-level exposure to electromagnetic fields (the results of an experiment). Part 1. Mobile communications and changes in electromagnetic conditions for the population. Needs for additional substantiation of the existing hygienic standards].

[In the consumers' interest: precautionary principles for protection against electromagnetic fields].

Exposure Knowledge and Perception of Wireless Communication Technologies.

Epidemiology of health effects of radiofrequency exposure.

World Health Organization, radiofrequency radiation and health - a hard nut to crack (Review).

Public perception of risk concerning celltowers and mobile phones.

Risks perception of electromagnetic fields in Taiwan: the influence of psychopathology and the degree of sensitivity to electromagnetic fields.

The prevalence of symptoms attributed to electromagnetic field exposure: a cross-sectional representative survey in Switzerland.

The development of human exposure standards for radio-frequency fields.

Recent advances in research on radiofrequency fields and health.

Searching for the perfect wave: the effect of radiofrequency electromagnetic fields on cells.

[Mutagenic, carcinogenic and teratogenic effects induced by radiofrequency electromagnetic field of mobile phone].

Cell phones and health concerns: impact of knowledge and voluntary precautionary recommendations.

An international prospective cohort study of mobile phone users and health (Cosmos): design considerations and enrolment.

Risk of brain tumors from wireless phone use.

Does precautionary information about electromagnetic fields trigger nocebo responses? An experimental risk communication study.

Vehicle-mounted high-power microwave systems and health risk communication in a deployed environment.

Mobile phone health risk policy in Germany: the role of the federal government and the Federal Office for Radiation Protection.

Electromagnetic fields (EMF): do they play a role in children's environmental health (CEH)?

Improving Precautionary Communication in the EMF Field? Effects of Making Messages Consistent and Explaining the Effectiveness of Precautions.

Discourse and policy making on consumer protection in the areas of mobile telecommunication and tanning.

Source of funding and results of studies of health effects of mobile phone use: systematic review of experimental studies.

Radiofrequency exposure from wireless LANs utilizing Wi-Fi technology.

Near-field radiofrequency electromagnetic exposure assessment.

Wi-Fi and health: review of current status of research.

[Mobile communication and health of population: estimation of danger, social and ethical problems].

Improved classification of evidence for EMF health risks.

German wide cross sectional survey on health impacts of electromagnetic fields in the view of general practitioners.

Thermal and non-thermal health effects of low intensity non-ionizing radiation: An international perspective.

Cell phone radiation: Evidence from ELF and RF studies supporting more inclusive risk identification and assessment.

Assessment of cellular telephone and other radio frequency exposure for epidemiologic research.

Potential health risks due to telecommunications radiofrequency radiation exposures in Lagos State Nigeria.

[Electromagnetic fields: damage to health due to the nocebo effect].

Radiofrequency exposure in the French general population: band, time, location and activity variability.

Public health and the radio frequency radiation emitted by cellphone technology, smart meters and WiFi.

[Fundamentally new electromagnetic pollution and the lack of adequate regulatory framework--on the risk assessment (analysis of modern domestic and foreign data)].

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Appendix 5 – Potential Impact of Wireless Radiation Exposure on the Opioid Crisis and Coronavirus Pandemic

A5-A. Potential Impact on Opioid Crisis

The previous findings of wireless radiation adverse effects reported in Chapter 2 of this monograph are based on ***hard evidence*** and have been ***validated*** in numerous studies. The present section on the link of wireless radiation to the opioid crisis is based on ***hard evidence*** as well, but the link of wireless radiation to the opioid crisis is ***not as far along in the validation process***. It should be viewed as a hypothesis at this point, and serve as a basis for discussion and further research.

The opioid crisis (drug dependence and overdosing) has become of increasing concern since the 1990s (coincidentally, when mobile networking technology was being introduced on a larger scale). This appendix addresses potential relationships between wireless radiation and increasing dependence on drugs.

"Over the past two decades, the United States has experienced a growing crisis of substance abuse and addiction that is illustrated most starkly by the rise in deaths from drug overdoses. Since 2000, the annual number of drug overdose deaths has quadrupled from 17,500 to 70,000 in 2017…..Most of these deaths involved opioids, including heroin, prescription painkillers, and synthetic opioids such as fentanyl." [Planalp, Hest, Lahr, 2019].

According to the US Department of Health and Human Services [HHS, 2019], 47,600 people died from overdosing on opioids in 2018, 10.3 million people misused prescription opioids in 2018, and 2 million people had an opioid misuse disorder in 2018. While there can be myriad contributing factors to such a widespread disorder, wireless radiation exposure (which increased dramatically over the same period that drug overdose deaths increased dramatically) may be a significant contributing factor. The reasons follow.

An analogy to climate change would be helpful in framing the perspective. The contribution of fossil fuel combustion to anthropogenic (man-made) climate change is ***conceptually*** similar to the contribution of wireless radiation to opioid overuse. The climate change analogy will be presented initially, since it crystallizes the nature of the causative effect. It will then be followed by the analogous details of the wireless radiation link.

The main contributing factor to anthropogenic climate change is the combustion of fossil fuels. The combustion process produces two major products relevant to climate change: carbon dioxide (CO2) and fossil sulphates/nitrates [Dutton, 2019].

CO2 from fossil fuel combustion percolates to the upper atmosphere and remains there for decades. It is transparent to the high frequency solar radiation and is partially absorbent of the lower frequency radiation returning from the Earth, thereby trapping some of the incoming solar energy in the atmosphere (and especially the ocean). Decades are required for the Earth's

global mean surface temperature to come into equilibrium with the levels of CO2 in the atmosphere.

The fossil sulphates and nitrates rise in the atmosphere, form small particles called aerosols, remain there for very short periods of time (days or weeks), then precipitate to Earth. They increase the effective albedo of the atmosphere (the albedo is a measure of the reflectiveness of the Earth's atmosphere to the incoming solar radiation), and this partial mirroring effect reduces the level of solar flux reaching the Earth's surface.

Thus, from the perspective of climate change, there is 1) an apparent *positive short-term* effect from the aerosol shielding of the solar radiation, and 2) a *negative long-term* effect from the energy trapping of the CO2. ***The positive short-term effect is masking the harmful effects of the negative long-term effect!***

What is the analogy of the climate change phenomena described above to the impact of wireless radiation on the opioid crisis? Consider the endogenous opioid system. This innate pain-relieving system "consists of widely scattered neurons that produce three opioids: beta-endorphin, the met- and leu-enkephalins, and the dynorphins. These opioids act as neurotransmitters and neuromodulators at three major classes of receptors, termed mu, delta, and kappa, and produce analgesia" and other effects [Holden et al, 2005].

It has been shown many times that one impact of wireless radiation (at myriad frequencies) is release of endogenous opioids [e.g., Radzievsky et al, 2008; Lai et al, 1983]. This release of endogenous opioids can enable analgesic effects by itself [Wu et al, 2012], or can enhance the analgesic effects of exogenous analgesics [Emilie et al, 2012; Thomas et al, 1979]. This has been demonstrated at pulsed millimeter-wave frequencies [Miryutova et al, 2001; Radzievsky et al, 2008; Hura et al, 2011], WiFi frequencies [Maillefer and Quock, 1992], mobile phone frequencies [Bodera et al, 2019], radiofrequencies [Foley-Nolan, 1992], and extremely low frequencies [Ozdemir et al, 2017; Demirkazik et al, 2019]. Additionally, as has been demonstrated by the results of the current monograph, wireless radiation at all the above frequencies has resulted in serious mid-term and especially long-term adverse health effects.

Therefore, analogous to the climate change example, wireless radiation exposure, especially at cell phone, WiFi, and millimeter-wave pulsed and modulated frequencies, generates ***1) analgesic and pleasurable short-term effects and 2) serious adverse mid- and long-term effects***. There would be some exceptions for the short-term, such as electrohypersensitivity (EHS) sufferers, who are immediately affected adversely by wireless radiation exposure.

For most people, the enhanced analgesic short-term effects of the wireless radiation would in effect mask the long-term damage from this radiation, analogous to the short-term positive effects from the aerosols masking the long-term negative effects from the CO2 in the climate change example.

Consider the following cases. In the first case, a person with ordinary pains and aches starts using a cell phone or WiFi system. There will be an almost immediate feeling of less pain and pleasurable sensations, similar to that experienced after a modest period of exercise (another stimulant of endogenous opioids) [Boecker et al, 2008]. This feeling can last for a short to intermediate length of time, after which another bout of stimulation is required to release further endogenous opioids. The cell phone/WiFi user will get conditioned to associating the immediate positive feelings with the wireless radiation-emitting devices. As time proceeds, the latent longer-term adverse effects of the wireless radiation will result in various levels of increasing discomfort and unpleasant symptoms, if not outright diseases. The immediate analgesic effects from the wireless devices will become even more important, but may be insufficient to overcome the increased levels of discomfort due to prolonged wireless radiation exposure. The individual will then be forced to use 1) exogenous opioids and narcotics and 2) wireless radiation devices to help attenuate the increasing feelings of discomfort, leading to possible addiction.

In the second case, a person with serious pain-producing disease or injury starts using a cell phone or WiFi system. This person has already been prescribed pain-killers of various types. Research has shown that wireless radiation of selected frequency characteristics in the parameter ranges discussed above not only exhibits an enhanced analgesic effect in its own right, but can enhance further the analgesic effects of exogenous analgesics [Emilie et al, 2012; Thomas et al, 1979]. Again, this person will become conditioned to the short-term analgesic and analgesic-enhancing effects of the wireless radiation devices. And, again, the increasing levels of discomfort eventually produced by prolonged wireless radiation exposure (augmented in many cases by adverse long-term effects of prolonged analgesics (https://www.painedu.org/pain-medications-long-term/) will increase the need for 1) further wireless radiation exposure and 2) additional exogenous analgesics. This positive feedback mechanism will lead to ***two forms of addiction***: exogenous pain-killers and wireless radiation.

Finally, consider the following. Alcohol has been shown to have analgesic effects [Thompson et al, 2017]. In Lai's experiments involving microwave irradiation and consumption of a mixture containing ethanol, microwave irradiation enhanced consumption of ethanol by about 25% [Lai et al, 1984]. As Lai pointed out, microwave irradiation may stress the rats, and consumption of ethanol may serve to reduce stress. So, the microwave irradiation triggers the release of endogenous opioids, producing a calming/analgesic effect, and at the same time increases stress or other adverse symptoms, driving the rodents to seek analgesia from an external source.

The above examples focus on positive short-term analgesic effects from wireless radiation followed by negative long-term addictive effects. There is no reason to believe this short-term long-term dichotomy is limited to analgesic effects. Wireless radiation ***short-term performance enhancements*** of many types accompanied by ***long-term detrimental effects*** cannot be ruled out (witness such effects for anabolic steroids on the performance of athletes in myriad sports [Vorona and Niesshlag, 2018], where short-term athletic performance is enhanced, with long-term adverse health consequences).

While the non-wireless radiation determinants of the opioid crisis should not be downplayed, a credible component of the opioid crisis may be the downward spiral of the self-re-enforcing positive feedback mechanisms generated by the wireless radiation. While there are obviously cultural influences, peer-presssure influences, over-prescribing of medications, etc, the pain and discomfort induced by the wireless radiation exposure may directly impact increased use of wireless radiation devices.

There is some overlap between the opioid crisis and the increased suicide crisis in the USA relative to wireless radiation exposure [Cheatle, 2011, 2016; Racine et al, 2017]. There are the same reasons existing for an increase in discomfort due to wireless radiation exposure, and the increase in suicide-related opioid abuse from wireless radiation, but the suicide crisis will not be addressed further in the current monograph.

A5-B. Potential Impact on Coronavirus Pandemic

The previous findings of wireless radiation adverse effects reported in Chapter 2 of this monograph are based on ***hard evidence*** and have been ***validated*** in numerous studies. The present section linking wireless radiation to exacerbation of the coronavirus pandemic is based on ***hard evidence*** as well, but the link of wireless radiation to exacerbation of the coronavirus pandemic is ***not as far along in the validation process***. It should be viewed as a hypothesis at this point, and serve as a basis for discussion and further research.

The fundamental hypothesis in this section is that wireless radiation weakens the immune system, and a weakened immune system increases the chances that exposure to the coronavirus (or any virus) will translate into symptoms/disease.

Almost a decade ago, I published a paper on potential treatments for SARS [Kostoff, 2011], the China-based pandemic of 2002-2003 that was associated with another coronavirus. As in the present China-based coronavirus pandemic, the SARS zoonotic virus/disease was thought to originate with infected bats. These bats then infected other species, which were then sold in open-air markets, and eventually infected their buyers.

Approximately 8,000 people globally presented with SARS symptoms, and approximately 10% of those who presented died. However, those who succumbed were not a random ten percent. Most had many co-morbidities, and it appeared their immune systems could not handle yet another insult.

The SARS pandemic was not ended with drugs or vaccines. None of these measures worked. Instead, quarantine and good hygiene contributed most to ending the pandemic.

After the pandemic ended, a number of physicians (especially in Asia) reviewed the records of all patients they had examined for various health issues (or standard annual physical examinations) during the pandemic, and concentrated especially on the blood test results. There were many cases where the coronavirus antibodies had shown up in the blood tests, ***but the patient had exhibited none of the SARS symptoms***.

In other words, the patient's adaptive immune system was sufficiently strong to operate properly and neutralize the coronavirus to which the patient had been exposed!

To me, that was the key finding, and contributed to the approach I have taken for developing protocols to reverse chronic diseases [e.g., Kostoff, Porter, and Buchtel, 2018].

There are on the order of 300,000 viruses, many/most of which have zoonotic potential. To develop vaccines for all of these viruses (before an epidemic or pandemic strikes) is unreasonable (based on present technology) because of the sheer numbers involved. To develop vaccines for any specific virus ***during*** an epidemic or pandemic (which was the mainstream approach taken for the coronavirus during the SARS pandemic of 2002-2003) is completely unrealistic, because of the lead times required for vaccine development, efficacy testing, ***credible*** mid-and long-term safety testing, and implementation.

Those who succumbed during the SARS pandemic had 1) myriad co-morbidities and 2) weakened immune systems unable to neutralize the SARS coronavirus. Having a strong immune system that allowed a smooth transition from innate immune system operation to adaptive immune system operation ***was the one intrinsic defense that worked!*** The SARS experience showed that the best and most realistic approach for defense against any potential viral attack is reversing immune-degrading lifestyles well before any pandemic or epidemic outbreaks. In that case, the immune system would be sufficiently strong to be able to handle viral exposure on its own without the emergence of serious symptoms, as was the case with those exposed to the SARS coronavirus (with coronavirus antibodies in their serum) who exhibited no (or minimal) symptoms.

This gets to the link between wireless radiation exposure and the latest coronavirus pandemic. Wireless radiation adversely affects the immune system (see boxed references at end of this section). To the degree that non-ionizing radiation exposure (superimposed on the myriad toxic stimuli to which many people are exposed by choice or imposition) degrades the operation of the innate and adaptive immune systems, it would increase the likelihood that the immune system could not counteract the exposure to the coronavirus (or any other virus) as nature intended. Thus, ***wireless radiation would contribute to the exacerbation of adverse effects from coronavirus exposure***. The bottom line is that exposures to essentially ALL the exogenous immune-damaging toxic stimuli (including, but not limited to, wireless radiation) need to be removed before resistance to viruses of any type can be improved substantially.

ADVERSE IMPACT OF WIRELESS RADIATION ON IMMUNE SYSTEM-REFERENCES

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Appendix 6 – Funding Source Bias on Research Outcomes

Upton Sinclair, noted muckracker and one-time candidate for Governor of California, once stated: "It is difficult to get a man to understand something, when his salary depends upon his not understanding it!" (https://www.brainyquote.com/quotes/upton_sinclair_138285). In a nutshell, this crystallizes the central problem of integrity and credibility of the biomedical literature, especially for topics of commercial, military, and political sensitivity.

There have been many studies addressing how researcher and institutional conflicts-of-interest relate to their published findings. The following article titles reflect only the tip of the iceberg of biased outcomes related to funding sources. Since research manipulations to achieve a predetermined agenda tend not to be advertised (e.g., see section 3.2.2 of Kostoff [2016]), what eventually sees the light of day is truly the very small tip of a very large iceberg. In these sensitive topical areas, ***bias may in fact be closer to the norm than to the exception***!

Titles of Sample Records

(to obtain Abstracts, insert titles into Pubmed, or similar search engines, if available)

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Appendix 7 – Adverse Effects of Wireless Radiation Related to Implants and Appendages

A7-A. Overview

Adverse impacts of wireless radiation on myriad medical implants and appendages don't get much discussion in the literature, especially passive medical implants (A7-B2), non-medical implant analogues (A7-B3), and metallic appendages (defined below), and especially with regard to radiofrequency radiation. The FCC has raised concerns about the interactions of RF radiation with passive implants. Paragraph 230 of [FCC, 2013] states: "Electrically conductive objects in or on the body may interact with sources of RF energy in ways that are not easily predicted. Examples of conductive objects in the body include implanted metallic objects. Examples of conductive objects on the body include eyeglasses, jewelry, metallic accessories, etc."

A number of articles in the database addressed non-organic implants, which are foreign bodies inserted into humans and animals for medical purposes. Non-organic implants addressed in the present database are typically not rejected by the immune system like organic foreign substances (although some adjuvants such as metal could induce autoimmune responses [Loyo et al, 2013]). Non-rejection does not imply safety, especially from exposure to wireless radiation.

There were two major types of implants covered by the database articles showing adverse effects: active implants that produced electrical signals mainly for controlling heart irregularities (e.g., pacemakers, defibrillators) and hearing deficiencies (e.g., cochlear implants), and passive metallic implants for structural support (e.g., dental implants, bone pins, plates, etc). The active implants also have a passive component, since their structural components are imbedded in, and interactive with, the incoming RF. Additionally, there are articles addressing adverse effects from wireless radiation in the vicinity of **metallic** appendages (e.g., eyeglasses, jewelry, etc).

The external EMF from microwaves (and other sources) could 1) impact the electrical operation of the active implants adversely, 2) increase the Specific Absorption Rate (SAR) values of tissue in the vicinity of the passive implants substantially, and 3) increase the flow rate and acidity of saliva. While the EMF effects on the cochlear implants could adversely affect auditory capability, EMF effects on the heart-related implants could potentially be life-threatening. The increased SAR values around the passive metal implants could result in increased tissue temperatures, and could adversely impact integration and longevity of the passive metallic implants. In the mouth, the combination of 1) increased tissue temperatures in proximity to the implant, and 2) increased saliva flow rate and acidity, could lead to 3) increased leaching of heavy metals from metallic orthodontic structures. This also raises the question: what other adverse health effects from the exposure of both the active and passive implants to increasing levels of wireless radiation have not been identified or addressed or reported?

While Table A7-1 shows that substantial research has been done on exogenous electrical interference with cardiac pacemakers and defibrillators, the impacts of automotive-based electrical sources on these active implants have not been promulgated widely. Appendix 8 addresses the larger issue of automotive-based wireless radiation at myriad frequencies, including adverse impacts on these active implants.

A7-B. Specific Impacts from Passive Implants

A7-B1. Overview

This sub-section of Appendix 7 examines two types of passive implants: passive metallic medical implants (dental implants, orthodontic structures, nails, plates, etc) and passive micro/nano implant analogues. The former types of implants are well-known, and the latter are much less well-known, especially in their interactions with radiofrequency radiation. The latter include exogenous nanoparticles that could also enhance absorption of RF radiation. Section A7-B2 focuses on the passive metallic medical implants, and section A7-B3 focuses on passive micro/nano implant analogues.

A7-B2. Impacts from Passive Metallic Medical Implants

The potential interference from external electromagnetic fields on implanted devices that emit electromagnetic signals is somewhat obvious, and has been the subject of extensive study. Some relevant documentation will be presented later. Less studied is the impact from external electromagnetic fields on passive metallic medical implants and appendages. What are the technical issues surrounding these EMF-implant interactions?

A good summary of these interactions is contained in Virtanen et al [2006]. The following excerpts are most relevant, and critical issues are highlighted.

"When a conductive object like an implant lies close to the source of the EM field, it affects the shape of the radiated field and thus the SAR distribution. Within a perfectly conducting object, the E field disappears; and outside the implant E field, ***lines bend perpendicular to the surface of the implant***. If the surface area is small, a ***dense EM flux may arise near the implant***. In lossy tissues, this leads to ***higher power absorption near the implant*** compared to the same tissue volume with no implant present. Correspondingly, tissue volumes with lower power absorption also occur as the implant redistributes the SAR pattern. This phenomenon may especially occur with implants that are thin…..or have sharp edges or tips…..Furthermore, the ***conductive implant may couple with a radiating source***, for example, an antenna. The coupling can be either conductive, magnetic, or both…..As a consequence, a ***current oscillating on an antenna induces a current on the implant***, too. Furthermore, the ***induced surface current produces a secondary EM field, which contributes to the power absorption, that is, SAR, in tissues around the implant…..Hence the implant acts as a weak radiating antenna…..or a re-radiator…..***in tissues. At high frequencies, the induced current flows in a thin surface layer of the implant, that is, at the implant–tissue interface, which may even slightly warm up due to ohmic losses…..However, this warming is marginal compared to warming of surrounding tissues.

The size of an implant is essential when evaluating its effect on the SAR distribution…..If the implant is very small compared to the wavelength, it does not have a strong effect on the SAR distribution…..Generally intermediate size implants, with dimensions close to the wave length, and ***especially those with resonance dimensions***, may cause strong EM fields around themselves, and thus ***enhanced SAR may occur around such an implant***. Large implants

again may cause a ***major change in the SAR distribution***, since they may ***scatter or reflect the field…..***

In addition to other size-related effects, the size affects the magnitude of induced currents…..A special case of this is the ***implant with a resonance dimension…..***The length, which apparently causes the highest SAR enhancement is about lT/3, where lT is the wavelength in the media…..or lT/2…..An implant with such a dimension may even ***cause enhancement of SAR1g or SAR10g by a factor of 2–3…..*** The shape of an implant is an important factor…..Pin- or rod-shaped implants ***may act as antennas and re-radiate the external field***…..Rings and other loop structures may act as induction loops and thus ***carry high induced currents***…..A gap in the loop or rod would ***induce high SAR in the gap***…..Sharp corners and tips in the implant may cause ***concentration of the EM flux around them***.

One essential factor in the interaction is the orientation of an implant with respect to the external source, that is, polarization of the field in the far field…..Especially for pin- or rod-like structures, the orientation parallel to the electric field or antenna is the worst case…..In this orientation, the ***implant may act like an antenna*** as described earlier. For arbitrary-shaped implants, the mutual inductance of an antenna and an implant depends on their orientation with respect to each other and other geometrical factors. The higher the mutual inductance, the stronger is the interaction…..

Since dielectric properties of tissues vary, the ***tissues that surround an implant have a great impact on the SAR distribution…..***If tissues have high conductivity and relative permittivity, they are very lossy and ***SAR around the implant can be high***…..

However, in bone, the ***SAR enhancement due to an implant is assumingly higher because of lower base level***. In general, the larger the relative difference between the dielectric properties, especially conductivity, of the two media, the ***greater is the bending of the EM field when it enters the more lossy media***. Hence ***larger averaged SAR values can be expected in small volumes on the boundary***. Furthermore, the dielectric properties affect the wavelength of EM field. In certain (plane wave) cases, the distance between the implant and the skin surface may match the wavelength in tissue so that constructive interference occurs in the surface layers…..This may ***cause elevated SAR in the surface***…..Similar phenomena may also occur in other layered structures…..

As a consequence of the described effects, the maximum SAR may occur at a different location with and without the implant in the EM field…..Usually the SAR will be at maximum either on the surface of the body, that is, in the skin, or in the tissue with the highest water content. However, due to the interaction of implant and RF field, the ***location of the highest SAR may be shifted to the proximity of the implant***. This is an important fact for RF exposure evaluation."

Key takeaways from this summary are that resonance between the incoming EMF waves and the implants can contribute to increased SAR levels, and these increased SAR levels can occur in the bone or soft tissue adjacent to the implant(s).

Implants, both active and passive, are cornerstones of modern medical treatment, and are big business. Many of the implant-related articles read for this monograph attempted to downplay the significance of the EMF effects on passive implants. While some acknowledged that substantial increases in SAR are possible, especially near the implant(s), some/many concluded that while SAR was indeed increased, the values averaged over the appropriate volume (as allowed by the FCC) were small, and well under the FCC exposure limits.

This is misleading. Stating that exposure levels well below those allowed by the FCC (which bear no relation to safety) are somehow 'safe' is disingenuous! There can be very high SAR levels in volumes smaller than those allowed by the FCC, and this could have a dual impact. The bonding between the implant and surrounding media could be impacted adversely, and the myriad other adverse effects associated with SAR levels of that magnitude could be operable. Many of the articles identified the presence of 'hot spots", where the SARs were very high, but the effect was numerically attenuated by averaging over a somewhat larger area.

Additionally, most of these measurements/computations were for single stressors only (the incoming EMF radiation). Adding the real-life combinations of other toxic stimuli would tend to exacerbate the effect, perhaps substantially. Finally, the measurements and computations tended to end with a demonstration of the increase in SAR level. That's equivalent to performing a chest x-ray on someone who smoked his first cigarette, and then writing a paper that smoking had little effect on the lungs and other structures! What are the long-term effects of the incoming wireless radiation on the passive implants and their supportive tissue/bone, both in terms of structural integrity and increased incidence of non-communicable disease impacts? The question about long-term effects and combined toxic stimuli applies to the operation of the active implants as well.

A recent paper addressing adverse effects of RF impacts on osseointegration (dental implant integration with underlying bone) illustrates the issues raised above [Kavyashree et al, 2019]. "Forty-eight implants were placed in tibia and femur bone of rabbits, and after 90 days the rabbits were sacrificed and bone surrounding the implant was retrieved…..Significantly less bone-to-implant contact and bone area surrounding implant threads were found in the test groups compared to the control group. There was a significant difference in regular bone formation…..among the three groups…..Implants exposed to cell phone radiation showed more inflammatory reaction when compared to the nonexposed implants, thus indicating that ***cellular phone overuse could affect the maturation of bone and thus delay osseointegration***."

If other toxic stimuli were co-exposed along with EMF radiation, and if longer-term data were taken, more severe impacts could be expected. Similar effects could be expected for other types of implants!

Table A7-1 contains an implant taxonomy. The format is category heading followed by a few selected references. The active implant categories cover cardiac, cochlear, and other devices, and the passive implant categories cover imbedded implants and appendages.

A sub-category of passive implants called Metal Release was created. This category reflects adverse effects of wireless radiation that are almost unknown to the general public. The

focus is on increased corrosive abilities of saliva due to wireless radiation exposure, and the subsequent release/leaching of metal from myriad orthodonic structures in the mouth. Many of these metals are heavy metals, such as nickel and chromium, which can be extremely dangerous when released into the body. Most of the references in Table A7-1 in this Metal Release category deal with nickel release from orthodonic appliances.

In these circumstances, the mobile phone radiation stimulates the parotid glands, causing them to produce more saliva. Not only is the flow of saliva increased, but its properties are altered, including reduction of pH. Additionally, as the larger passive implant category has shown, there will be some preferential heating at the saliva-orthodonic appliance interface. The net effect will be to increase corrosion of the metal appliance, resulting in release of nickel. ***Given that children are major customers for many of these dental appliances as well as increasingly major users of cell phones, WiFi, etc, these children will be adversely impacted by the wireless radiation through myriad pathways!***

Table A7-1 – Implant Taxonomy

CATEGORY/SAMPLE REFERENCES
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A7-B3. Impacts from Passive Macro/Nano Implant Analogues

The FCC regulations for RF exposure limits are based on an ***average exposure*** over a six-minute period for occupational applications, and thirty-minute period for the general public. For near-field exposures, the guidelines can be summarized as follows: "Whole-Body SAR is averaged over the entire body; Partial-body SAR is averaged over any 1 g of tissue in the shape of a cube; SAR for hands, wrists, feet and ankles is averaged over any 10 g of tissue in the shape of a cube.

SAR limits are not applicable above 6.0 GHz; MPE limits for field strength and power density should be applied. Categorical exclusion of routine MPE evaluation for mobile transmitters does not apply to portable devices operating above 6.0 GHz" (https://transition.fcc.gov/oet/ea/presentations/files/oct05/RF_Exposure_Concepts_Support_KC.pdf).

Averaging processes attenuate the extremes. In particular, within the thirty-minute averaging window used for general population exposure averaging (above), there could be many examples of RF power fluxes exceeding the FCC guidelines, perhaps substantially. Given that the FCC Guidelines are based on thermal limits not being exceeded, this means that (within the thirty-minute averaging window) temperatures (and related thermal stresses) could reach peak values capable of inflicting serious damage. Neufeld and Kuster [2018] examined RF heating of skin in the >10 GHz region, and concluded: "Transient exposure with high PAR [peak-to-average ratio] can lead to large temperature oscillations, with peak temperature increases in the skin reaching tens of degrees, thus exceeding tissue damage thresholds after short exposure durations." The computations were made "at an intensity resulting in a temperature increase of 1 K at ***continuous exposure***".

Would implant analogues, such as imbedded nanoparticles in the heated tissue, change the characteristics of tissue heating from the pulsed RF described above? Section 2E contains the statement "At the millimeter carrier wavelengths characteristic of high performance 5G, one can expect resonance phenomena with small-scale human structures [Betzalel, 2018], as well as resonances with insects/insect components [Thielens et al, 2018, 2020]." Can this enhanced heating of tissue due to high-frequency pulsed RF be extended to nanoparticle-imbedded tissue?

These implant analogues could include e.g. tattoos using nanoparticle materials, nanoparticles from cosmetics and air pollution, possibly nanoparticles from forced-air occupational situations, etc. These analogues could be metallic or non-metallic. Whether they would be heated by RF, and how that would vary by particle characteristics, RF characteristics, and surrounding tissue properties, is an open question. Collins et al [2014] conclude, for gold nanoparticles: "The chief conclusion is that in some cases gold nanoparticles immersed in RF fields heat, and in other cases they do not."

There is a substantial literature on RF heating of nanoparticles within tissue, motivated by applications to hyperthermia treatment of diseased tissue, especially cancer [e.g., Huang et al, 2008; Gupta et al, 2010; Hanson et al, 2011; Cardinal et al, 2008; Xu et al, 2008; Tamarov et al, 2016; Ocampo-Garcia et al, 2015; San et al, 2013; Pantano et al, 2017; Nordebo et al, 2017; Nguyen et al, 2016; Mironava et al, 2017; Mackeyev et al, 2017; Liu et al, 2015; Letfullin et al,

2015; Kim et al, 2013; Corr et al, 2012; Collins et al, 2014; Chaurasia et al, 2016; Amini et al, 2018; Glazer et al, 2010; Dennis and Ivkov, 2013; Sassaroli et al, 2012; Moran et al, 2009; Gannon et al, 2008]. The references/bibliography at the end of this section contain more examples of RF (and other pulsed) heating of nanoparticles, including some non-tissue-related heating.

There is not uniform consensus on heating mechanisms. While myriad specific approaches are used for RF heating of nanoparticle-imbedded tissue for cancer therapy, Glazer and Curley [2011] provide a reasonable summary of the technical issues involved. They state: "RF field therapy is a non-invasive method to expose cancers to nonionizing radiation that is relatively nontoxic in and of itself….. nanoparticle-mediated RF field hyperthermia induces heating on the scale of approximately 100 μm. Fortunately, noninvasive RF fields easily penetrate human tissues and pass through the entire body with minimal perturbations until the RF fields interact with metal….. The size of the field can be scaled from small, animal-sized devices….. up to very large volumes that could theoretically treat small (local tumor) or large regions of the human body. Samples (cells, animals or, theoretically, patients) are placed in an RF field created by a parallel plate capacitor ….. This establishes a directional electromagnetic field that passes through tissues and organs without significant absorbance. Metal, however, absorbs RF energy and quickly releases heat to the surrounding region. Nanoparticles in general, and metal nanoparticles specifically, are utilized because of this general principle, as well as their unique qualities that absorb even more energy (and release even more heat), due to their very small size and quantum characteristics.

Recent advancements in nanotechnology have resulted in multiple types of nanoparticles that can be targeted with antibodies, peptides/proteins and sugar residues to cancer cells….. Thermal therapy is induced with either focused laser irradiation, manipulation of magnetic fields or RF field exposure….. While these nanoparticles may be more selective than specific, animal studies have demonstrated promising results without major toxicity….. nanoparticles induce intracellular hyper-thermia. Unique physicochemical properties of metallic and non-metallic nanoparticles result in different heating rates for various types of non-ionizing radiation exposure….. We have found that solid gold nanoparticles less than 50 nm in diameter are safely taken up by macrophages in the liver and spleen without major toxicity….. large nanoparticles with a large aspect ratio (i.e., rods or tubes) have been associated with fibrosis and cellular injury….. Nanoparticle heating in RF fields is a very complex phenomenon. The end result is that RF fields induce nanoparticle heating rates of 1–3°C/s in various solutions and at various power levels…..Most RF field devices are based on shortwave RF fields (13.56 MHz) as licensing agencies permit this frequency for 'medical use'….. The power of these devices is typically from 100–800 W. The energy transfer efficiency from the field generator to actual field strength varies amongst the devices; determining the exact field strength is problematic….. The RF electrical field strength in general, however, is approximately 5–15 kV/m…..

In a nanoparticle concentration and field strength-dependent manner, nanoparticles in aqueous solutions can reach boiling temperatures in 2–3 min. Interestingly, deionized water negligibly heats in RF fields, while antibody solutions (e.g., with ions or proteins) typically heat around

five- to ten-times less than nanoparticle solutions….. In the RF field, SWNTs heat in the range of 2–6°C/s, slighter faster than gold…..

There remains some controversy regarding the mechanism in which nanoparticles heat in an RF field. Our group has demonstrated that gold nanoparticles heat primarily via Joule heating….. This work has demonstrated that gold nanoparticles behave as 'mini-resistors', where free electrons on the surface have limitations to their movement. In this way, friction is created at the individual nanoparticle level, which release heat into the surrounding aqueous solution. Furthermore, based on these findings, one would predict less heating for larger nanoparticles as well as much less for aggregates where there are far fewer limitations placed on the movement of electrons."

Extrapolation from the cancer (and similar) therapy use of nanoparticles for hyperthermia destruction of diseased tissue to heating of nanoparticles imbedded in near-surface tissue by communications-level RF powers is not straight-forward. The therapeutic situation involves high-power RF targeting nanoparticles with desired pre-selected properties at specific locations to achieve high temperatures, whereas the communications situation involves low-power RF interacting with nanoparticles of unknown properties at unknown locations constrained to low temperature increases. Additionally, the RF therapy situation has typically involved RF frequencies in the MHz range, whereas the RF communications scenario (especially for 5G) would involve frequencies in the GHz range (high-band 5G would be in the 10s of GHz).

It is unclear how the effects on tissue surrounding these micro/nanoscale implant analogues would relate to those of the macro-implants of the previous section. More detailed computations are required to identify specific temperature excursions and related thermal stresses for specific nanoscale implant anologues.

As mentioned previously, there may also be electromagnetic and thermal resonances with insects and other small living creatures and substances. For insects, there could be resonances based on overall body dimensions [e.g., Thielens, 2018, 2020], and/or resonances based on specific appendage dimensions. Insects are a critical part of the ecological chain, and severe functional damage may occur even if only critical organs or appendages are damaged. If antennae are heated, or experience even moderate cyclic thermal stresses, that may be sufficient to disable the insect, and eliminate their functional contribution to the ecological chain.

Additionally, for insects, heating at different spatial macroscales may be sufficient to cause damage, as well as microscale heating. More detailed insect heating computations at the microscale, and at the macroscale (covering the spectrum of full body resonance to critical appendage resonance) are required before declarations of safety become credible.

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Appendix 8 – Adverse Effects of Automotive-Based Wireless Radiation

A8-A. Overview

The modern automobile is a powerful source of wireless radiation at myriad frequencies, and is subject to external wireless radiation at myriad frequencies as well. The trend has not been to reduce these sources, but rather to add equipment both to the vehicle and to the external environment that will increase the wireless radiation flux associated with the vehicle substantially. The numbers and types of sources are not well-known, even among those experts and laymen concerned about adverse effects from wireless radiation. This appendix will address only a few of those sources.

Some/much of the appendix is based on recent personal experience. Over a year ago, I began looking for a new car. My previous vehicle had almost none of the wireless add-ons that are promoted extensively by the automotive industry, and I had hoped to replace it with a similar wireless-free vehicle.

I spent perhaps six months part-time test-driving vehicles, taking some EMF measurements in selected vehicles, and researching myriad vehicles on the Web. This appendix uses some of my findings as a starting point to identify the full scope of the radiation flux exiting and entering the vehicle.

A8-B. Specific Automotive Wireless Radiation Sources

During this automotive evaluation process, I received a very informative response from Dr. Theodore Metsis. It is summarized at the following link (http://www.radiationdangers.com/automotive-radiation/automotive-radiation/). Of particular interest is the diagram at the beginning of the article, showing radars and wireless sensors in modern cars. I would recommend the reader study that diagram in detail, to better appreciate how ubiquitous are these sources of wireless radiation. Not all the wireless radiation enters the cabin, since some/much is outward-directed, but some/much of it will enter the cabins of other cars on the road.

However, that diagram tells only part of the story. Assume there is a car pool commuting to work from the suburbs of a major city. It is not uncommon (in today's world) for a one-way trip to take from one-two hours, or more. Even in a regular car, or mid-size SUV, there might be four or so passengers. They may be using cell phones, WiFi, or both, thereby adding to the radiation from the automotive-based sensors/transmitters. For example, in an experiment comparing cell phone-Bluetooth use inside and outside a car, Dhami [2015] concluded "The increase in radiation power density with the use of Bluetooth was observed to be 313% higher as compared to phone alone when measured outside the car…..The power density was observed to have increased by 393% when cell-phone and Bluetooth were used inside the car with windows rolled up as compared to using no phone/Bluetooth."

There will be cell towers lining the sides of a major highway, thereby increasing the radiation to the occupants substantially. Depending on conditions, there may be substantial air pollution to which the occupants are exposed. Additionally, the prolonged sitting is very dangerous, and is a contributing factor to many serious diseases [Kostoff, 2015]. If the vehicle is new, there may be substantial out-gassing of toxic chemicals from the interior materials (https://www.ecocenter.org/newsletter/2012-02/dangers-lurk-behind-new-car-smell). Combined exposure to the wireless radiation, air pollution and other toxic substances, coupled with prolonged sitting and continual impacts from the car's motions, produces a synergistic effect that exacerbates adverse impacts from any of the constituent components substantially.

A8-B1. Automotive ELF

About a decade ago, the Israeli Ministry of Environmental Protection undertook an evaluation of the safety of hybrid vehicles with respect to emissions of non-ionizing radiation. The following excerpt summarizes their findings, and the context (https://www.thetruthaboutcars.com/2010/03/israel-preps-worlds-first-hybrid-car-radiation-scale/):

"Not exactly flower power, the radiation in question is cast by the electromagnetic field made by alternating current (AC) flowing from the batteries in the back to the engine up front. The medical implications of this non ionizing radiation, similar to radiation from cellphone antennas, are not yet clear.

The Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) recommends a limit of 1,000 mG (milligauss) for a 24-hour exposure period. While other guidelines pose similar limits, the International Agency for Research on Cancer (IARC) deemed extended exposure to electromagnetic fields stronger than 2 mG to be a "possible cause" for cancer. ***Israel's Ministry of Health recommends a maximum of 4 mG***.

The ministry's foray into this topic is a culmination of a public outcry resulting from publications in the media regarding possible dangers from radiation in hybrid cars. Last year, Israeli automotive website Walla! Cars conducted a series of tests on the previous generation Toyota Prius, Honda Insight and Honda Civic Hybrid, and recorded radiation figures of ***up to 100 mG during acceleration***. Measurements also peaked when the batteries were either full (and in use) or empty (and being charged from the engine), while ***normal driving at constant speeds yielded 14 to 30 mG*** on the Prius, depending on the area of the cabin."

Over the past couple of decades (bracketing the Israeli study), a number of researchers have conducted studies measuring EMF emissions in conventional gasoline/diesel-powered cars, hybrids, and electric vehicles. Some of these studies are listed in the first part of Table A8-1. The results are all over the map. One reason is that "there are alternating magnetic fields produced by its engine, control systems, air conditioning, sound, video, communications, etc. In vehicles with tyres, one has to add the magnetic field produced by the magnetized metal of the wheels" [Paniagua et al, 2017]. Additionally, the results vary by location, vehicle speed, and braking/acceleration.

The findings of [Paniagua et al, 2017] are instructive, and provide a good summary of magnetic fields found in fossil-fuel powered cars. "Other works, however, detect magnetic fields inside cars with values that are comparable to ours. Thus, for example, Milham et al…..found a range of 0.2 to 2.0μT, Stankowski et al…..0.1 to 9.5μT, and Halgamuge et al…..0.3 to 3.5μT. These studies used instrumentation that measures magnetic fields with frequencies ***above 5 Hz***.

As in our study, Stankowski et al…..found magnetic fields that were higher in the rear seats than in the front seats, and higher at floor level than at the seat and head levels. The reason for the differences between the two sets of studies lies in the frequency ranges used. The rotating wheels produce spectral peaks that coincide with the rotation frequency, typically 6–12 Hz…..and are ***not detectable when using instrumentation whose lowest frequency threshold is 30–40 Hz***. This instrumentation detects the magnetic fields generated by the motor and the electrical systems of the vehicle, but not those generated by the effect of wheel rotation. One can therefore conclude that the magnetic fields from the rotating wheels represent a very important part of the total magnetic field inside the vehicles." The maximum exposures reported in the study by [Panigua et al, 2017] were about twenty milligauss (1 μT=10 milligauss).

In a 2014 article on hybrids [Karabetsos et al, 2014], Figure 8 (cruising at 80-120 kilometers/hour) shows exposures in the right-rear seat reaching over 20% of 1998 ICNIRP limits for the general public. The article does not provide actual magnetic field numbers for these exposures, nor does it provide the frequencies at which these magnetic fields were measured. The 1998 ICNIRP limits are a function of frequency.

The article states: "it was observed that the major components of the magnetic flux density appeared at frequencies lower than 100 Hz." In this frequency spectrum, the ICNIRP limits range from 400,000 milligauss at 1 Hz to 500 milligauss at 100 Hz. So, the actual measurement of ~20% of ICNIRP limit could range from 80,000 milligauss to 100 milligauss, depending on the frequency(s) at which the measurements were made. For reference, the ICNIRP limits for power frequency (60 Hz) are 830 milligauss (for the general public), far above the levels shown as dangerous in the Israeli reference above, and other references in the biomedical literature.

In Vasilev et al, [2015], magnetic field measurements were made in myriad electric vehicles and hybrids. The two major sources of magnetic fields were traction currents and wheels. The findings for each were as follows: "Therefore, if the traction current has variations up to ±300 A, the magnetic field could also have variations of up to ±300 μT." The upper limit translates to ***3,000 milligauss***! "The permanent magnetization of steel belted tires is a well known source of in-vehicle magnetic fields…..Our measurements show that this phenomenon is responsible for a magnetic field inside the car of up to 2 μT at the wheel frequency fw (which ranges from 0 to 20 Hz for speeds ranging from 0 to 130 km/h)." This upper limit translates to twenty milligauss, and is similar to the upper limit above reported by Panigua et al, [2017].

References to other studies are shown in the first section of Table A8-1. The hybrids and EVs are associated with larger magnetic fields (especially at acceleration and braking), due mainly to large electrical power transfers for all operations. My own measurements in hybrids

showed magnetic fields around the driver could range up to 15-30 milligauss, depending on the vehicle. However, the meter I used had a lower limit of 40 Hz, so I could not measure the powerful magnetic fields shown by the above studies to occur at the extremely low frequencies. However, in my view, chronic exposure even to the 15-30 milligauss I measured is something to be avoided at all costs, much less the larger fields at the lower frequencies!

In many of the references shown in Table A8-1 (and beyond), the authors don't present actual magnetic field numbers, but rather magnetic fields ***normalized to ICNIRP*** recommended exposure limits. They usually conclude that, because the ratios are less than one, therefore, the vehicles are safe. This is fallacious and disingenuous, since the ICNIRP recommended limits have nothing to do with safety (based on exposures shown to cause harm in the biomedical literature) and everything to do with providing cover for the wireless radiation promoters.

Additionally, many of the guidelines tend to be based on single stressor experiments. Vehicle cabins expose their occupants to many types of toxic stimuli (described initially in this appendix), and the synergies will reduce the levels of EMF exposure at which damage occurs, sometimes dramatically.

Table A8-1 – Appendix 8 References

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A8-B2. Automotive Radar

Radar has become ubiquitous on modern automotive vehicles. Many of the new 'safety' systems use radar in their operation, and in-cabin radars have been proposed to further enhance 'safety'. How safe are these radar add-ons, and what are their potential radiation levels?

The FCC used to have a requirement that when cars were stopped, such as in a traffic jam or at a traffic light, any onboard radars would have to reduce power to minimize longer-term exposure to humans. In 2009, Toyota applied to relax these rules, for reasons described in the linked document below. Naturally, the FCC complied with the request, as per the following 2012 directive addressing vehicle radar systems:
https://www.federalregister.gov/documents/2012/08/13/2012-19732/operation-of-radar-systems-in-the-76-77-ghz-band.

The FCC promulgated the following emission limits:

"In lieu of separate emission limits for in-motion and not-in-motion, the Commission proposed to increase the average power density limit to 88 μW/cm2 at 3 meters (average EIRP of 50 dBm) and to decrease the peak power density limit to 279 μW/cm2 at 3 meters (peak EIRP of 55 dBm) for vehicular radar systems regardless of the direction of illumination."

Converting units, the average power density limit would be 88*10^4 microwatts/square meter, or 880,000 microwatts/square meter, ***at three meters***. So, in slow moving traffic on a superhighway, if there was six meters separation between the bumper of the car behind and the driver of the car ahead (a conservative estimate in bumper-to-bumper traffic), there could be as much as ***220,000 microwatts/square meter radiating the front vehicle***, if the radars were operating at the allowable limit. Some bands would be absorbed by the glass at these frequencies, and other bands would penetrate the glass. There could also be side radar coming from cars other than the rear car.

However, pedestrians or highway workers would not have whatever protections from wireless radiation are afforded by vehicle structures. For example, a person walking on a crosswalk in front of stopped traffic could even be closer to the bumper than three meters, and could be exposed directly full body to a ***million microwatts/square meter***, or more, if the radars were operating at the allowable limit! Children walking close to a vehicle would be even more vulnerable, since they are closer to the horizontal plane of bumper antennas. And, these numbers carry the assumption of being radiated from one car only. If there are multiple cars, with some emissions spreading to the side, then the cumulative exposures could be well above the FCC exposure limits at selected points. Walking on streets in high traffic areas may become a dangerous pastime, and few people realize it!

Are there sources of radar potentially entering the vehicle cabin other than those from the 'safety' sensors? The answer is yes (at least in the future and maybe in the present), as shown in the following article: https://www.eetimes.com/document.asp?doc_id=1333330.

The author of the above article states:

"For example, the digital processing capability inside the mmWave sensor can filter out noise, said Wasson, allowing TI's radar chips to detect very small movements, even the breathing that indicates the presence of a person or animal inside a vehicle.

Wasson noted that "child occupancy detection" is likely to become a feature in the Euro NCAP roadmap. This, he believes, will open the door for TI's radars in body, chassis, and in-cabin applications. As tier ones and OEMs look for the right sensing technology to enable such detection possibilities, Wasson noted that radars are much better-positioned.

Radar, for example can "see" through a blanket to determine whether a child is underneath. TI's radar chips can even distinguish between a person and a static object like a duffel bag, explained Wasson, because their on-chip digital signal processing can detect a heartbeat."

The aim seems to be to deliberately flood the cabin with radar RFR, for various detection purposes. The article makes no mention about potential power levels.

Another potential in-cabin source would be radar aimed at the driver continuously, to insure alertness and awareness. For example, consider the following statement:

"Sudipto Bose, director of marketing for automotive radar at Texas Instruments, points out that in-cabin radar offers a number of benefits. It can alert parents if they've left children in a car, and it can be used for gesture controls, which let drivers control navigation, phone and stereo with hand motions. This proximity radar could also identify if a driver's attention is not focused out the windshield.....If automakers take Texas Instruments up on its new radar sensors, a production vehicle with radar-based gesture control would still be two to five years away (https://www.cnet.com/roadshow/news/volkswagen-invests-100-million-to-develop-solid-state-battery-tech/)."

There appears to be a developing market focused on occupancy sensing using radar. For example, "So radar is no longer the preserve of complex and costly, high-end markets, it's ready for 100% reliable infant presence detection in cars. Whether our children's movements are major, minor or finer, they'll be detected and the driver can be automatically reminded: 'Don't forget, you have someone very precious in the back seat of your car.' (https://www.xethru.com/blog/posts/hot-car-deaths-radar-tech-can-help-save-lives)".

As another example: "Startup Caaresys imagines its radar-based system monitoring the respiration and heart rates of everyone in the car, with a particular focus on sensing a child that might be hidden from view in the back and potentially left behind in the car (https://www.cnet.com/roadshow/news/in-car-monitoring-surveillance-technology-privacy/)."

Novelic (https://www.novelic.com/applications-and-system-solutions/) offers radar-based sensors for car interiors, including: Seat Occupancy Sensor; Vital Signs Detection Sensor; Passenger Detection Sensor; "Baby left in a car" Sensor; Driver and Passengers Fatigue Sensor; Driver and Passengers Emotion Sensor. Other non-car applications include: Baby crib monitoring; Assistive Living for Elderly People; Visual Impairments Sensor; Emotion Sensor.

Azcom (http://www.services.azcomtech.com/index.php/services/mmwave-sensors/automobile-in-cabin-monitoring/) promotes continuous in-cabin monitoring as follows: "In case of autonomous vehicles, it will also be critical to continuously monitor vehicle occupancy without creating privacy concerns. Radar sensors, combined with special ad hoc algorithms, are well-suited for such applications."

There are even nascent efforts to make some of these systems mandatory. For example, "U.S. lawmakers and European safety regulators are considering rules that could mandate "child presence detection" systems aimed at avoiding hot-car deaths of unattended children. That has suppliers scrambling to develop new systems for automakers, according to interviews with several high-tech suppliers at a recent industry conference here….."The moment you have regulation, things are going to move fast," Melamed told Automotive News. "The timelines are very, very close."" (https://www.autonews.com/regulation-safety/safety-idea-gets-mandate)

Also, it's not clear how the FCC exposure limits (which are already six orders of magnitude higher than exposures shown in the biomedical literature to cause damage) would apply to limit in-cabin radiation levels. Would they apply to each source, or to the total radiation? The former seems more likely, since it is unclear how they would enforce the latter. If that is the case, cabin occupants could be exposed theoretically to radiation levels in excess of the FCC present limits.

In sum, the occupants of a hybrid vehicle with a full load of passengers will be subject to:

*ELF-EMF from the tires and other sources unique to hybrids

*RFR from the passengers' cell phones

*RFR from Bluetooth

*RFR from the WiFi 'hot spot' and the devices communicating with the hot spot

*RFR from the myriad cell towers that dot the sides of most highways

*RFR from the radar sensors of other cars

*RFR from on-board radar sensors to detect motions and driver alertness within the cabin

Almost all these radiation sources will also be operable in a gasoline-powered car, and there will be some radiation reflections within the cabin because of the surrounding metal.

Our studies on combinations of toxic stimuli including non-ionizing radiation [Kostoff et al, 2018; Kostoff and Lau, 2013, 2017] showed that adverse health effects are exacerbated when non-ionizing radiations of different characteristics are combined. What would be the effects of the above complex combination that goes well beyond EMF constituents?

The middle part of Table A8-1 contains references to other studies showing deliberate radar impingement on vehicle occupants.

A8-B3. Automotive Wireless Networks

The past decade has seen an increasing number of wireless networks that are fully or partially intra-vehicular. The final segment of Table A8-1 presents only a small part of the studies that have been done to expand these intra-vehicular wireless networks. The remainder of this appendix addresses two of these many intra-vehicular networks, and it is based on my personal experience with these two networks.

A8-B3a. Keyless Access Network

As mentioned at the beginning of this appendix, about six months ago I bought a new car. One of the features in the particular model trim is the ability to open the doors and allow engine ignition with a key fob in proximity. This capability is not unique to the model I bought. Far from it! My search process showed that the push-button start capability is rapidly becoming ubiquitous in new cars. The strenuous process of turning a key in a lock is thereby bypassed.

According to the Owner's Manual, my new car has a 130+ KHz continuously operating wireless network that allows 1) the doors to be opened and closed, and 2) the ignition to be started by push-button, when the key fob is within proximity of the car. The Owner's Manual also states that the radio waves from the network could potentially interfere with an implanted pacemaker or defibrillator, and accompanies this statement with a Warning icon.

Luckily, according to the Owner's Manual, this wireless system/function can be disabled. The disabling is allowed not because of any manufacturer-stated concern for the adverse effects of wireless radiation on normal humans. It is allowed because it could potentially interfere with the operation of pacemakers and other similar devices.

The Owner's Manual provides two approaches to disabling the keyless access capability, thereby converting the key fob to effectively a key with some remote-control functions (like the TV remote-control). Disabling this capability is not a simple process, as I discovered. I was not able to do it myself, even though two alternative methods were provided in the Owner's Manual.

The dealer from whom I bought the vehicle said the disabling was not possible, and the second dealer I visited required two technicians to experiment before they could finally disable it.

Here's the critical point of this narrative. The technicians (and other service personnel) of the second dealer told me ***no one had ever requested this disabling before***! From the first dealer's feedback, I'm sure they had never received such a request either.

This means that the customers with implanted electrical devices who purchase these vehicles are (for the most part) not disabling the 130+ KHz wireless network. My guess is they don't even know about this network. None of the salespeople I had at any of the dealerships who offered test drives in cars with keyless access function (and in my case there were probably half a dozen different brands I drove that had this capability) asked whether I had an implantable electrical device (I don't) nor mentioned the presence of the 130+ KHz network, or any other frequency applicable to their model/brand. I doubt whether any of them knew!

Here's the bottom line. If people with implantable devices are not motivated to eliminate these wireless networks, where there exists a rather obvious potential danger to health, how will healthy (or relatively healthy) people become motivated to avoid wireless systems/radiation?

Whatever dangers the 130+ KHz network (or different frequency networks of other brands performing the same function) would pose in isolation, I suspect the adverse effects would be amplified substantially in combination with the other toxic stimuli sources I mentioned in previous sections. While one could make arguments about some applications of wireless radiation being useful/justified in extreme emergencies, installing a potentially harmful wireless network to eliminate inserting a key into a lock is technology gone mad!

A8-B3b. Tire Pressure Monitoring System

Another intra-vehicular source of wireless radiation entering the automobile cabin is the tire pressure monitoring system (TPMS). TPMS has been mandated in the USA by the TREAD Act, and has been installed on all cars for the past decade. (https://en.wikipedia.org/wiki/Tire-pressure_monitoring_system). It is mandated and used in many other countries as well.

There are myriad types of these sensors. Most Direct TPMS deploy tire pressure sensors on each wheel of a vehicle. As tire pressure data is collected for each tire, it is sent to one or more TPMS receivers, using RF (radio frequency) technology. The majority of Direct TPMS installations transmit their data via UHF (Ultra High Frequency) radio. TPMS data is typically transmitted in one of two frequency ranges, which depends on the geographical location of the TPMS: about 433MHz in Europe, and at 315MHz in most other parts of the world. https://tpms247.com/blogs/tpms-faq/73376901-tpms-frequencies-315-mhz-433mhz.

I didn’t find any articles addressing adverse health effects from the TPMS. That doesn’t mean they don’t exist. There may be myriad reasons why I didn’t find adverse health effects.

A8-B4. Other

The latter part of Table A8-1 alludes to many other types of networks being studied, as well as optimizing some already implemented. Most of these are not mentioned in my new car Owner's Manual, since I assume they don't affect pacemakers and defibrillators (at least according to whomever has responsibility for monitoring such systems). The only way to fully understand the levels of wireless radiation to which vehicle occupants are being subjected is through detailed measurements of the wireless radiation environment.

This would include full spectrum monitoring (from 1 Hz for ELF to >100 GHz for millimeter-wave communications and detection). Testing would be done under at least four conditions:

- no passengers and in an EMF quiet zone, with all on-board electronics operating;
- with passengers using myriad wireless devices, in EMF quiet zone, and with all on-board electronics operating;
- no passengers and in a typical urban business high EMF antenna concentration zone, and with all on-board electronics operating;
- with passengers using myriad wireless devices, in high EMF high antenna concentration zone, and with all on-board electronics operating.

Those results should begin to provide some idea of the complex and potentially dangerous wireless radiation environment that many commuters face.

ABOUT THE AUTHOR

Ronald Neil Kostoff received a Ph. D. in Aerospace and Mechanical Sciences from Princeton University in 1967. He has worked for Bell Laboratories, Department of Energy, Office of Naval Research, and MITRE Corp. He invented the Wake Shield for producing high vacuum in low orbit, and used in manned space missions for research and development. He has published over 200 peer-reviewed articles, served as Guest Editor of four journal Special Issues since 1994, obtained two text mining system patents, and presently is a Research Affiliate at Georgia Institute of Technology.

He has published on numerous medical topics in the peer-reviewed literature, including:

- potential treatments for
    - Multiple Sclerosis,
    - Parkinson's Disease,
    - Raynaud's Phenomenon,
    - Cataracts,
    - SARS,
    - Vitreous Restoration,
    - Peripheral Neuropathy/Peripheral Arterial Disease
    - Alzheimer's Disease, and
    - Chronic Kidney Disease;
- potential causes of Chronic Kidney Disease;
- potential causes of Alzheimer's Disease;
- potential causes of Peripheral Neuropathy/Peripheral Arterial Disease
- potential impacts of Electromagnetic Fields on health; and
- synergistic effects of toxic stimuli combinations.

His recent publications in toxicology have shown that regulatory exposure limits to toxic stimuli are, on average, orders of magnitude too high compared to exposures shown to cause damage in the biomedical literature, and are not protecting the public from harmful substances.

He is listed in:

- Who's Who in America, 60th Edition (2006),
- Who's Who in Science and Engineering, 9th Edition (2006), and
- 2000 Outstanding Intellectuals of the 21st Century, 4th Edition, (2006).

Sent. N.

Cron. N.

R. Gen. N. **361/08**

R E P U B B L I C A I T A L I A N A

I N N O M E D E L P O P O L O I T A L I A N O

La Corte d'Appello di Brescia, Sezione Lavoro, composta dai Sigg.:

Dott. Angelo TROPEANO Presidente

Dott. Antonella NUOVO Consigliere rel.

Dott. Anna Luisa TERZI Consigliere

ha pronunciato la seguente

S E N T E N Z A

nella causa civile promossa in grado d'appello con ricorso depositato in Cancelleria il giorno 08/07/2008 iscritta al n. 361/08 R.G. Sezione Lavoro **e posta in discussione all'udienza collegiale del 10/12/2009**

d a

XXX, rappresentato e difeso dall'Avv.to Danilo MINA di Brescia, domiciliatario giusta delega a margine del ricorso in appello

RICORRENTE APPELLANTE

OGGETTO:

Prestazione: indennità rendita vitalizia INAIL o equivalente – altre ipotesi

c o n t r o

I.N.A.I.L., in persona del Direttore Regionale pro tempore, rappresentato e difeso dall'Avv.to Sabina LUPO ed elettivamente domiciliato presso l'Avvocatura Distrettuale I.N.A.I.L di Brescia giusto mandato generale alle liti

RESISTENTE APPELLATO

In punto: appello a sentenza n. 471/08 del 15/05/2008 del Tribunale di Brescia.

CONCLUSIONI

Del ricorrente appellante:

come da ricorso in appello

Del resistente appellato:

come da memoria di costituzione e risposta

Svolgimento del processo

Con ricorso al Tribunale di Brescia, giudice del lavoro, depositato il 6.7.2007 Innocente XXX conveniva l'INAIL per sentirlo condannare a corrispondergli le prestazioni di legge in riferimento ad una grave e complessa patologia cerebrale che aveva origine professionale. Esponeva, in fatto, di aver svolto attività di dirigente d'azienda dal 1981, e, da ultimo, presso la S.p.a. Sangiacomo dal 2.9.1991 al 26.9.2003; che in tale mansione aveva utilizzato il telefono cellulare e il cordless per una media di 5 - 6 ore al giorno e per un periodo di 12 anni; che, essendo destrimane, teneva l'apparecchio all'orecchio sinistro in quanto con la mano destra rispondeva al telefono fisso collocato sulla scrivania o prendeva note e appunti; che detta attività gli aveva provocato una grave patologia per la quale il 17.11.03 aveva chiesto all'INAIL le corrispondenti prestazioni di legge, che l'Istituto aveva rifiutato la richiesta, negando il nesso causale fra l'attività lavorativa e le affezioni denunciate. Pertanto insisteva nella domanda, deducendo prova per testi sulle modalità lavorative ed allegando un'approfondita relazione medico-legale del neurochirurgo dott.Giuseppe Grasso.

L'INAIL si opponeva al ricorso, sempre sotto il profilo della

carenza del nesso causale, e deduceva controprova orale, producendo varia documentazione.

Esperita l'istruttoria testimoniale, che accertava in fatto l'uso intenso di cellulare e cordless, ed assunta consulenza tecnica d'ufficio, il primo giudice respingeva la domanda per carenza del nesso causale, aderendo alle considerazioni svolte dal CTU, aspramente criticate dal ricorrente.

Appellava il XXX, depositando ulteriore e ponderoso elaborato del consulente di parte, riportandosi alle considerazioni critiche ivi svolte e chiedendo che, previo rinnovo della consulenza, l'INAIL fosse condannato alle prestazioni di legge.

Si costituiva l'INAIL per la conferma, ricordando l'inesistenza di studi scientifici attendibili in ordine alla nocività delle onde elettromagnetiche.

Questa Corte rinnovava la consulenza e, a fronte delle osservazioni svolte dall'INAIL, concedeva termine all'appellante per depositare le sue controdeduzioni: all'esito, all'udienza odierna, le parti discutevano e la Corte decideva con sentenza del cui dispositivo veniva data immediata lettura.

Motivi della decisione

La CTU disposta in questo grado di giudizio, molto documentata ed accuratamente motivata, individua il nesso, quanto meno concausale, tra l'utilizzo dei telefoni e la patologia.

Innanzitutto, occorre osservare che l'allegato utilizzo di cellulare e radiotelefono per molte ore lavorative (5-6 ore / die) e

l'uso principalmente dell'orecchio sinistro, che consentiva il libero impiego della mano destra per note scritte, hanno trovato piena conferma testimoniale e non sono più oggetto di contestazione da parte dell'INAIL.

Da questo dato, che quantifica il livello di esposizione, doveva dunque partire il consulente per la sua indagine sul nesso causale.

Dall'anamnesi clinica risulta che nel giugno 2002 è comparsa ipoanestesia (perdita parziale della sensibilità) dell'emiviso di sinistra: eseguita la Risonanza Magnetica Nucleare veniva formulata diagnosi di "neurinoma del Ganglio di Gasser" che è un tumore benigno che colpisce i nervi cranici, in particolare il nervo acustico, mentre più rara è la localizzazione al V nervo cranico (Trigemino), come nel caso in specie. Secondo la spiegazione fornita dal consulente, deriva dalle cellule (cellule di Schwann) della guaina di rivestimento da cui anche la denominazione di Schwannoma. La localizzazione anatomica di questo tumore dà ragione della severità delle manifestazioni cliniche correlate.

Subiva quindi un intervento neurochirurgico l'8 novembre 2002 (Ospedale S. Anna di Lucerna) con resezione branca mandibolare del nervo in quanto non dissociabile, asportazione del ganglio di Gasser, ma permaneva residuo tumorale dimostrato a RMN post operatoria.

Gli esiti post intervento possono così essere riassunti: 1) ulcera corneale sin. (da iposecrezione lacrimale e disturbo

neurologico). Attuò vari trattamenti per risolverla. E' in atto un trattamento cronico con autosiero. Graduale deficit del visus (3-4 diottrie); 2)Sindrome algo-distrofica dell'emiviso di sinistra con dolore cronico severo. Attuati vari e ripetuti trattamenti con farmaci attivi sul dolore neuropatico con scarso o nullo beneficio. Dall'agosto 1.08.2005 assunzione orale di morfina (Oxicontin 20mg x3 /die) in associazione con pregabalin (Lyrica 75mg x 3. trattamento del dolore neuropatico periferico e centrale negli adulti). Valutazione dolore di 5-6 con scala numerica verbale (scala di valutazione del dolore cronico. Valori da 1-nessun dolore- a 10-il peggiore dolore immaginabile-. Il dolore si considera controllato per un indice ~4); 3) Persistenti parestesie sempre all'emiviso; 4) Disturbi della meccanica masticatoria da mal occlusione secondaria ad atrofia dei muscoli temporale massetere di sinistra (attua fisiochinesiterapia per mantenere il trofismo); 5) Incostante diplopia (visione doppia, in senso orizzontale o verticale, di uno stesso oggetto) 6) Epilessia parziale complessa a genesi temporale da encefalomalacia (rammollimento cerebrale legato al trauma chirurgico con perdita di funzione possibile origine di foci epilettogeni); 7) Deficit cognitivo (disturbo della fissazione mnesica e ed attenzione); 8) Disturbo dell'adattamento (Nel Diagnostic and Statistical Manual of mental disorders [DSM-N] i disturbi dell'adattamento sono definiti come "sintomi emozionali o comportamentali clinicamente significativi" che si sviluppano "in risposta a uno o più fattori stressanti psicosociali identificabili"); 9) sindrome del lobo temporale (sindrome complessa

da danno del lobo temporale con vari disturbi-olfattivi, gustativi, dell'equilibrio, visivi, disturbi uditivi e psichiatrici)

Tutte queste situazioni cliniche sono ampiamente documentate negli atti. Ogni patologia è suffragata da consulenze cliniche specialistiche, anche ripetute, e da opportune indagini strumentali ed ematochimiche.

Nel 2003 diagnosi di neoformazione surrenalica di destra 5x3 cm con normofunzione. Intervento 30.06.2004 Istituto Europeo di Oncologia con diagnosi istologica di feocromocitoma (tumore raro con possibile secrezione di catecolamine [sostanze ormoni e neurotrasmettitori- prodotte dalla porzione interna del surrene e da alcuni neuroni] II tumore in presenza di secrezione di catecolamine si caratterizza per una sindrome clinica peculiare).

Nel caso in specie, non fu dimostrata secrezione di catecolamine.

E' seguito da uno psichiatra dal 2003 ed è in terapia con paroxetina; due ricoveri c/o Servizio Psichiatrico di Diagnosi e Cura l'ultimo nel marzo 2008.

Attuale terapia comprende Morfina orale, Pregabalin, Paroxetina (farmaco antidepressivo appartenente alla classe degli SSRI-inibitori selettivi della ricaptazione della serotonina), Dolore attuale riferito 5-6 con scala numerica verbale.

Si presente come soggetto magro in mediocri condizioni generali. Normale sanguinificazione. Sensorio normale orientamento spazio temporale. Tono dell'umore depresso. Crisi di pianto in corso

della valutazione clinica. Paresi del VII nervo cranico con ipostenia del muscolo orbicolare. Anestesia dolorifica abolita sensibilità tattile. Asimmetria degli emivisi. Dolorabilità alla pressione su articolazione temporo-mandibolare sin. Lieve tendenza all'intrarotazione mano dx in estensione.

La prima valutazione del consulente è che gli esiti della neoplastica son assolutamente severi e del tutto documentati e che la qualità di vita del sig. XXX è sicuramente gravemente compromessa da tali esiti.

Quanto alla questione centrale (non essendovi contestazione sulle conseguenze subite dall'appellante) relativa al nesso causale tra l'uso dei telefoni e insorgere della patologia, il consulente osserva innanzitutto che, vel periodo in cui ha lavorato alla San Giacomo SPA per i primi 3 anni utilizzava telefono cellulare - cordless (deposte 5-6 h al di), dal 1993-4 al cordless fu associato l'uso di telefono cellulare fino al settembre 2003.

Orbene, i telefoni mobili (cordless) e i telefoni cellulari funzionano attraverso le onde -elettromagnetiche. Secondo il CTU "In letteratura gli studi sui tumori cerebrali per quanto riguarda il neurinoma considerano il tumore con localizzazione al nervo acustico che è il più frequente. Trattandosi del medesimo istotipo è del tutto logico assimilare i dati al neurinoma del trigemino".

Nella CTU, con una tabella molto chiara a cui ci si riporta, sono riassunti alcuni studi dal 2005 al 2009. "In tre di essi (Hardell group) e si evidenzia un aumento significativo del rischio relativo di

neurinoma. (Rischio relativo: misura di associazione fra l'esposizione ad un particolare fattore di rischio e l'insorgenza di una definita malattia, calcolata come il rapporto fra i tassi di incidenza negli esposti [numeratore] e nei non esposti [denominatore]. Esempio: un rischio relativo di 3 sta a significare che il tasso di incidenza negli esposti è 3 volte maggiore dei non esposti. Nella tabella il rischio relativo è derivato dall'odd ratio)

Un recente lavoro, sempre del gruppo di Hardel (Mobile phones, cordless phones and the risk far brain tumours L. Hardell and M. Carlberg INTERNATIONAL JOURNAL OF O COLOGY 35: 5-17, 2009), che si basa sulla revisione degli studi già pubblicati dallo stesso gruppo, considera altri elementi quali: età dell'esposizione, ipsilaterlaità e tempo di esposizione. Per quanto riguarda il neurinoma (dell'acustico) i risultati indicano un Odd ratio per l'uso del cordless di 1,5 e per il telefono cellulare di 1,7. Considerando l'uso> di 10 anni, gli Odd ratio sono rispettivamente di 1,3 e di 1,9.

L'Odds ratio è il rapporto tra la frequenza con la quale un evento si verifica in un gruppo di pazienti e la frequenza con la quale lo stesso evento si verifica in un gruppo pazienti di controllo. Se il valore dell'odds ratio è superiore a 1 significa che la probabilità che si verifichi l'evento considerato (per esempio una malattia) in un gruppo (per esempio tra gli esposti) è superiore rispetto a quella di un altro gruppo (per esempio tra i non esposti). Significato opposto ha un valore inferiore a 1 (riduzione del rischio legato all'esposizione). Se il valore è pari a 1 significa che non vi è differenza tra i gruppi. In caso

di eventi rari l'odds ratio ha un valore molto vicino a quello del rischio relativo. L'odds ratio è una misura particolarmente utile negli studi caso-controllo come stima del rischio relativo, che in questo tipo di studi non può essere misurato direttamente.

Una recente review della The International Commission on Non-Ionizing Radiation Protection (Exposure to high frequency electromagnetic fields, biological effects and health consequences 100 kHz-300 GHz. Review of the scientific evidence on dosimetry, biologicaI effects, epidemiological observations, and health consequences concerning exposure to high frequency electromagnetic fields 100 kHz to 300 GHz, 2009) evidenzia i limiti degli studi epidemiologici fin'ora attuati. I principali "bias" riguardano la modalità di arruolamento, spesso l'assenza di un gruppo di controllo con ricorso a registri di popolazione, l'impossibilità di standardizzare l'entità e la durata complessiva di esposizione. Gli autori concludono che, allo stato attuale, non vi è una convincente evidenza del ruolo delle radiofrequenze nella genesi dei tumori, ma aggiungono che gli studi non ne hanno escluso l'associazione (pag 336 "Results of epidemiological studies to date give no consistent or convincing evidence of a causal relation between RF exposure and any adverse health effect. On the other hand, these studies have too many deficiencies to rule out an association").

A questo punto è utile dedicare spazio ad un'altra review del tutto autorevole.

Kundi nel 2009 (The Controversy about a Possible

Relationship between Mobile Phone Use and Cancer Michael Kundi Environ Health Perspect. 2009 March; 117,3: 316-324) L'autore conferma i dubbi che gli studi epidemiologici inducono per quanto riguarda il tempo di esposizione e conclude per rischio individuale basso, ma presente. L'esposizione può incidere sulla storia naturale della neoplasia in vari modi: interagendo nella fase iniziale di induzione, intervenendo sul tempo di sviluppo dei tumori a lenta crescita, come i neurinomi, accelerandola ed evitando la possibile naturale involuzione.

L'analisi della letteratura non porta a un giudizio esaustivo, ma con tutti i limiti insiti nella tipologia degli studi, un rischio aggiuntivo per i tumori cerebrali, ed in particolare per il neurinoma, è documentato dopo esposizione per anni (>10) a radiofrequenze emesse da telefoni portatili e cellulari.

Il dato anamnestico di esposizione supera il limite dei 10 anni.

Il tempo di esposizione è elemento valutativo molto rilevante. Nello studio di 2006 l'esposizione per più di 10 anni comportava un rischio relativo calcolato di 2,9 sicuramente significativo (pur considerando i limiti metodologici già illustrati) Si tratta quindi di una situazione "individuale" che gli esperti riconducono al "modello probabilistico-induttivo" ed alla "causalità debole" (Angelo Fiori Atti VI Convegno Nazionale di Medicina Legale Previdenziale 2006), che ha comunque valenza in sede previdenziale.

Un ruolo quindi, almeno concausale, delle radiofrequenze

nella genesi della neoplasia che ha patito il sig. XXX è "probabile" (probabilità qualificata)" (vedi CTU dott. Di Stefano pag. 8 e 9).

L'esito della malattia ha condotto ad una menomazione stimata nella misura, incontestata, del 80%.

L'Inail ha criticato l'elaborato sostenendo in primo luogo che la prima asserzione del CTU sarebbe errata: utilizzare gli studi relativi al neurinoma dell'acustico per analizzare un caso di neurinoma del trigemino non sarebbe possibile in quanto si tratterebbe di tumore a diversa localizzazione in quanto relativa a diverso distretto anatomico.

La censura non merita accoglimento: infatti, come correttamente spiegato dal consulente, i due neurinomi appartengono al medesimo distretto corporeo in quanto entrambi i nervi interessati si trovano nell'angolo ponto-cerebellare, che è una porzione ben definita e ristretta dello spazio endocranico certamente compresa nel campo magnetico che si genera dall'utilizzo dei telefoni cellulari e cordless.

Una seconda censura riguarda gli studi utilizzati dal CTU per rispondere positivamente al quesito: si tratterebbe infatti di studi su un basso numero di casi mentre lo studio del 2000 dell'OMS ha escluso effetti negativi sulla salute. Anche questa censura però non coglie nel segno.

Lo studio dell'OMS, risalente appunto al 2000 e basato su dati, ovviamente, ancora più risalenti, non tiene conto dell'uso più recente, ben più massiccio e diffuso, di tali apparecchi e del fatto che

si tratta di tumori a lenta insorgenza: pertanto gli studi del 2009, basati su dati più recenti, sono di per sé più attendibili. Inoltre, come ha osservato nelle controdeduzioni il ct di parte del XXX, non si tratta di studi su un basso numero di casi, ma, al contrario, del tutto esaustivo in quanto tratta di 678 casi, che sono il numero totale che si verifica in un anno in Italia (trattandosi di tumore non frequente). Inoltre, a differenza dello studio della IARC, co-finanziato dalla ditte produttrici di telefoni cellulari, gli studi citati dal dott. Di Stefano sono indipendenti.

Naturalmente, secondo il costante insegnamento della Suprema Corte, nel caso di malattia professionale non tabellata, come anche in quello di malattia ad eziologia multifattoriale, la prova della causa di lavoro, che grava sul lavoratore, deve essere valutata in termini di ragionevole certezza, nel senso che, esclusa la rilevanza della mera possibilità dell'origine professionale, questa può essere invece ravvisata in presenza di un rilevante grado di probabilità. A tale riguardo, il giudice deve non solo consentire all'assicurato di esperire i mezzi di prova ammissibili e ritualmente dedotti, ma deve altresì valutare le conclusioni probabilistiche del consulente tecnico in tema di nesso causale, considerando che la natura professionale della malattia può essere desunta con elevato grado di probabilità dalla tipologia delle lavorazioni svolte, dalla natura dei macchinari presenti nell'ambiente di lavoro, dalla durata della prestazione lavorativa e dall'assenza di altri fattori extralavorativi, alternativi o concorrenti, che possano costituire causa della malattia.

Nel caso di specie, il CTU ha spiegato i valori di odd ratio che lo portano a sostenere la probabilità qualificata del ruolo, quanto meno, concausale, dell'uso dei telefoni nella causazione dell'infortunio, ma, per rendere più evidente la reale portata di quanto affermato, appare utile dar conto dell'esempio che il ct di parte ha fatto nelle contro-osserazioni depositate il 25.11.2009. Partendo dai dati indicati dal CTU, il dott. Grasso afferma essere utile confrontare il dato di rischio individuale ottenuto dal consulente (2,9) con quello ricavato per il fattore di rischio, universalmente riconosciuto, dell'esposizione alle radiazioni ionizzanti. Afferma il dott. Grasso: "Orbene: nei soggetti esposti a 1Gy di RI, come i sopravvissuti alle esplosioni atomiche giapponesi di Hiroshima e Nagasaki, è stato accertato un rischio relativo di tipo oncologico di 1,39 per "tutti i tumori" con un minimo di 1,22 per i tumori di "utero e cervice" ed un massimo di 4,92 per la "leucemia", il che significa che il rischio oncogeno medio delle RI è inferiore a quello che si ha per l'esposizione alle radio frequenze in riferimento ai neurinomi endocranici" (vedi relazione dott. Grasso depositata il 25.11.2009 pag.7 e 8).

Appare, quindi, evidentemente integrato il requisito di elevata probabilità che integra il nesso causale richiesto dalla normativa.

Ne consegue che l'INAIL deve essere condannato a corrispondere al XXX la rendita per malattia professionale prevista per l'invalidità all'80%, con arretrati ed interessi di legge.

Le spese seguono la soccombenza e si liquidano per il primo

grado in €.1.800,00 (di cui euro 650 per diritti) e per il presente grado in €.2.000,00 (di cui euro 700 per diritti) con distrazione in favore del procuratore antistatario. Le spese di CTU restano definitivamente a carico dell'INAIL.

P.Q.M.

In riforma della sentenza n. 471/08 del Tribunale di Brescia condanna l'INAIL a corrispondere all'appellante la rendita per malattia professionale prevista per l'invalidità all'80%, con arretrati ed interessi di legge; condanna l'INAIL alla rifusione delle spese di ambo i gradi liquidate per il I grado in €.1.800,00 e per il presente grado in €.2.000,00.

Brescia 10.12.2009

Il Consigliere est. Il Presidente

COMMENT

Appeals that matter or not on a moratorium on the deployment of the fifth generation, 5G, for microwave radiation

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Abstract. Radiofrequency (RF) radiation in the frequency range of 30 kHz-300 GHz is classified as a 'possible' human carcinogen, Group 2B, by the International Agency for Research on Cancer (IARC) since 2011. The evidence has since then been strengthened by further research; thus, RF radiation may now be classified as a human carcinogen, Group 1. In spite of this, microwave radiations are expanding with increasing personal and ambient exposure. One contributing factor is that the majority of countries rely on guidelines formulated by the International Commission on Non-Ionizing Radiation Protection (ICNIRP), a private German non-governmental organization. ICNIRP relies on the evaluation only of thermal (heating) effects from RF radiation, thereby excluding a large body of published science demonstrating the detrimental effects caused by non-thermal radiation. The fifth generation, 5G, for microwave radiation is about to be implemented worldwide in spite of no comprehensive investigations of the potential risks to human health and the environment. In an appeal sent to the EU in September, 2017 currently >260 scientists and medical doctors requested for a moratorium on the deployment of 5G until the health risks associated with this new technology have been fully investigated by industry-independent scientists. The appeal and four rebuttals to the EU over a period of >2 years, have not achieved any positive response from the EU to date. Unfortunately, decision makers seem to be uninformed or even misinformed about the risks. EU officials rely on the opinions of individuals within the ICNIRP and the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), most of whom have ties to the industry. They seem to dominate evaluating bodies and refute risks. It is important that these circumstances are described. In this article, the warnings on the health risks associated with RF presented in the 5G appeal and the letters to the EU Health Commissioner since September, 2017 and the authors' rebuttals are summarized. The responses from the EU seem to have thus far prioritized industry profits to the detriment of human health and the environment.

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Key words: appeals, microwave radiation, radiofrequency radiation, radiofrequency electromagnetic field, EMF, 5G, EU, WHO, moratorium, health risks, non-ionizing radiation guidelines

Introduction

Over the years, numerous international appeals on radiofrequency (RF) radiation and health and the environment have been published (e.g., www.emfscientist.org). These seem to have had little or no impact on those proposing limits on RF radiation and on the deployment of this technology. On the contrary, ambient RF radiation exposure has increased and is a potential health risk based on the current knowledge of the biological effects of RF radiation (1-8). There seems to be an 'unholy' alliance between the telecom industry and certain scientists, organizations (even WHO), and some politicians, thus reducing the potential for precautionary actions (9,10).

The International Agency for Research on Cancer (IARC) of WHO in 2011 classified RF radiation in the frequency range of 30 kHz-300 GHz as a 'possible' human carcinogen, Group 2B (11,12). Since then, the evidence of the adverse effects of RF radiation has strengthened based on human epidemiological (7,8,13) and animal studies (14-16). These results add scientific evidence to a previous evaluation (17). Thus, RF radiation may now be classified as a human carcinogen, Group 1. That is the strongest classification, which is the same as that for e.g., asbestos and smoking.

The IARC cancer classification seems to have had little or no impact on protecting the public against risks associated with RF exposure. A major hampering factor has been the exposure guidelines by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) based only on the acute and very short-term thermal (heating) effects of RF radiation. These guidelines are used by the majority of countries worldwide. These guidelines were initially published approximately 20 years ago (18) and were updated in 2009 (19); however, no changes were made to adapt to the

rapidly increasing evidence of the harmful effects of RF and new RF signal characteristics and exposure from new technologies. ICNIRP, with the support of the WHO (10) and the major telecom companies, has made considerable efforts to convince countries worldwide to follow their guidelines. However, with the deployment of the 5th generation of microwave radiation, 5G, even the obsolete ICNIRP guidelines may be exceeded and may become an obstacle for the deployment of 5G (20). Thus, ICNIRP is preparing new guidelines that are briefly commented on below. However, as already published (9,10), the ICNIRP guidelines may be contradictory to a vast number of existing scientific reports demonstrating the harmful effects of RF radiation (21). Furthermore, there may perhaps also be conflicts of interests in terms of ties to the industry.

ICNIRP

On July 11, 2018, the ICNIRP released a draft of the guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (100 kHz-300 GHz). It was open for public consultations until October 9, 2018. Appendix B was based on the assessment of the health risks based on a literature survey (https://www.icnirp.org/en/activities/public-consultation/index.html).

Of note, in the background material to the new ICNIRP guidelines, the IARC classification from 2011 of RF exposure as class 2B, 'possibly' carcinogenic to humans (11,12) was not included. Notably, one of the ICNIRP commission members, Martin Röösli (https://www.icnirp.org/en/about-icnirp/commission/index.html), was also one of the IARC experts evaluating the scientific RF carcinogenicity in May, 2011 (https://monographs.iarc.fr/wp-content/uploads/2018/06/mono102-F05.pdf), which classified RF exposure as a class 2B 'possible' carcinogen. Thus, he should be aware of the IARC classification. Of note, one of the authors of this article (L.H.) was a member of the IARC expert group.

Below, eight excerpts/quotes from the 2018 ICNIRP draft guidelines are presented (https://www.icnirp.org/cms/upload/consultation_upload/ICNIRP_RF_Guidelines_PCD_2018_07_11.pdf). These assertions in the ICNIRP evaluation do not seem to represent the valid evaluation of the published literature on the health risks associated with RF:

i) ***Brain physiology and function.*** *'In summary, there is no evidence of effects of radiofrequency EMF [electromagnetic field] on physiological processes or eye pathology that impair health in humans. Some evidence of superficial eye damage has been shown in rabbits at exposures of at least 1.4 kW m-2, although the relevance of this to humans has not been demonstrated'.*

ii) ***Auditory, vestibular, and ocular function.*** *'In summary, no effects on auditory, vestibular, or ocular function relevant to human health have been substantiated'.*

iii) ***Neuroendocrine system.*** *'In summary, the lowest level at which an effect of radiofrequency EMF on the neuroendocrine system has been observed is 4 W kg-1 (in rodents and primates), but there is no evidence that this translates to humans or is relevant to human health. No other effects have been substantiated'.*

iv) ***Neurodegenerative diseases.*** *'In summary, no adverse effects on neurodegenerative diseases have been substantiated'.*

v) ***Cardiovascular system, autonomic nervous system and thermoregulation.*** *'In summary, no effects on the cardiovascular system, autonomic nervous system, or thermoregulation that compromise health have been substantiated for exposures with whole body average SARs below approximately 1 W kg-1, and there is some evidence that 4 W kg-1 is not sufficient to alter body core temperature in hamsters. However, there is strong evidence that whole body exposures in rats that are sufficient to increase body core temperature by several degrees centigrade can cause serious adverse health effects in rats'.*

vi) ***Immune system and hematology.*** *'The few human studies have not indicated any evidence that radiofrequency EMF affects health in humans via the immune system or haematology'.*

vii) ***Fertility, reproduction and childhood development.*** *'In summary, no adverse effects of radiofrequency EMF exposure on fertility, reproduction or development relevant to human health have been substantiated'.*

viii) ***Cancer.*** *'In summary, no effects of radiofrequency EMF on cancer have been substantiated'.*

Since the ICNIRP 2018 draft guidelines excluded a large number of science-based evidence of health hazards from RF radiation, numerous rebuttals have been sent to the ICNIRP. However, it remains unknown as to whether these rebuttals have been taken into account or not.

Thus, the ICNIRP does not acknowledge the health effects caused by RF radiation. This has been rebutted by several scientists (21-24).

Details and proofs of scientific misinterpretation were outlined in a comprehensive response by Dr Martin Pall (21). He demonstrated that the denials of scientific facts concerning health risks seem to be the rule in the Health Risk Assessments of the ICNIRP 2018 Draft Guidelines. ICNIRP confirmed that Pall's response was received on October 8, 2018 (tinyurl.se/pall). As outlined above in all eight summarizing statements, the ICNIRP denies that any scientific reports exist which demonstrate harmful effects below the ICNIRP guidelines. However, as Dr Pall demonstrated, a large number of peer-reviewed studies have been published over a period of >20 years contradicting the ICNIRP evaluations. Independent peer-reviewed scientific articles (1,7,8) have demonstrated the harmful effects even far below the current public safety limits based on ICNIRP 1998 reference levels 10 W/m^{2}for 2-300 GHz and 2-10 W/m^2 for 400 to 2,000 MHz (18).

The ICNIRP also seems to have disregarded previously published animal studies (14-16) on carcinogenesis. The NTP results have been discussed in a commentary (25) and clarified to that degree that they should have been considered in full. These findings supported human epidemiology results on cancer risks from RF radiation (6,26). The final new ICNIRP guidelines have yet to be published.

In fact, a hint of the ICNIRP final document may be found in a presentation by the ICNIRP chairman Eric van Rongen at a meeting held on April 17, 2019 https://www.anfr.fr/fileadmin/mediatheque/documents/expace/workshop-5G/20190417-Workshop-ANFR-ICNIRP-presentation.pdf.

van Rongen stated that there is no evidence that RF EMF causes diseases, such as cancer and that the US NTP (14-15) and Ramazzini Institute (16) studies are not convincing for carcinogenesis. ICNIRP seems still to hold the view, which is clearly beneficial to the industry, that only thermal effects exist for RF radiation and not any non-thermal effects, which have been proven in research by the majority of scientists in this field.

ICNIRP recently published a note on the NTP and Ramazzini Institute animal studies (27). Some of their incorrect statements are commented on below. The ICNIRP claims that there is no verified mechanism for RF radiation carcinogenesis, in spite of well-designed studies showing the contrary, e.g., oxidative stress (25,28) and DNA damage (25,29). The ICNIRP claims that the histopathological evaluation was not blinded in these studies; however, this is not true, as supported by the methods described in these studies. Furthermore, the ICNIRP claims that the body core temperature was increased in the NTP study (15) and suggested it to be a factor increasing cancer risk, although heat is not a known carcinogen. The ICNIRP also claims that only the Hardell group found an increased risk for acoustic neuroma although the Interphone study had similar findings (7). ICNIRP does not seem to take into account the concordance between the tumor types found in human epidemiological and animal studies. These are just a few examples.

It is noteworthy that ICNIRP repeats certain debatable statements in spite of being rebutted by Melnick (25) and should have been known to the 13 ICNIRP Commission members (https://www.icnirp.org/en/about-icnirp/commission/index.html) with their names listed at the end of the article (27). Perhaps this ICNIRP article lacks scientific authorization. As previously suggested, they seem to create doubt (30,31). Thus, one must be cautious when also interpreting other publications by the 13 Commission members.

The ICNIRP points out an important scientific problem: How incorrect data can achieve lives of their own and gain respectability and credence with inappropriate repetition. Corrections and clarifications (25), seem to have difficult time to counteract any possible errors, which is to the disadvantage of both good science and public health. Of note, President Franklin D. Roosevelt stated that *'Repetition does not transform a lie into a truth'* (https://www.azquotes.com/quote/377323).

Finland, in a new regulation, 1045/2018, dated December 15, 2018, allowed higher average radiation, 200 W, in narrow areas of 1x1 cm (1 cm^2) (please see Table 1.5, Note 3 (in Swedish): (https://www.finlex.fi/data/sdliite/liite/6943.pdf). This was probably decided in order to accommodate the steerable, beam-formed, narrow 5G fields, which will be used by most 5G equipment. The Director of the Radiation Safety Agency in Finland claims that this is no problem, as if you disperse the effect of 200 W (on 1 cm^2) upon a whole square meter it will still be within the ICNIRP guideline of 10 W/m^2 (private communication from Petteri Tiippana, 2018, please see https://www.dropbox.com/s/89cm7bmb4l0em8w/200W%3Am2-STUK.pdf?dl=0).

On top of the other flaws which ICNIRP members are presenting, they also suggest that only the 'mean values' of RF radiation should be measured. However, the interferences and the supra-additive effects between pulses from different RF radiation sources can lead to 'hundreds of thousands higher density' short-time pulses than the power density mean values with the guideline of 10 W/m^2. This has been well-documented in a report from the Finnish Radiation Safety Agency (32). Panagopoulos (29) has clearly demonstrated that using mean values for RF radiation may underestimate the risk. Intensity, frequency, exposure duration, polarization, pulsing and modulation are crucial parameters for the bioactivity. Puranen (32) states that the instant effect density can be much stronger than the mean values. However, the guidelines only consider the mean values.

Appeals to the EU and responses from the EU

The impact of the many international appeals on RF radiation safety, if any, is unclear. However, they will be historical documents on warnings that have been thus far ignored by the EU and the WHO. This is exemplified below.

The deployment of 5G for microwave radiation has given increasing awareness and concern among individuals regarding the risks to human health and the environment resulting in massive protests and even a moratorium in certain EU countries and US cities (https://tinyurl.se/5gstoppers). 5G uses a different technology compared with previous generations, such as 2G, 3G and 4G. In the following, our 5G appeal to EU is discussed (www.5Gappeal.eu). This has currently been signed by >260 scientists and medical doctors from a number of countries. It is still open for endorsement.

a) The 5G Appeal, September 13, 2017 and response. Below, the full text, with included links to references, is presented although it can also be found online (www.5gappeal.eu), and also at (https://www.environmentandcancer.com/5g-appeal/).

Scientists and doctors warn of potential severe health effects of 5G. *'We the undersigned scientists and doctors recommend a moratorium on the roll-out of the fifth generation, 5G, for telecommunication until potential hazards for human health and the environment have been fully investigated by scientists independent from industry. 5G will substantially increase exposure to radiofrequency electromagnetic fields (RF-EMF)*

on top of the 2G, 3G, 4G, Wi-Fi, etc. for telecommunications already in place. RF-EMF has been proven to be harmful for humans and the environment'.

5G leads to the marked increase of mandatory exposure to wireless radiation. *'5G technology is effective only over short distance. [The range of 5G radiation is decreased due to its increased carrier frequency (up to ~100 GHz) compared to previous mobile telephony generations and other existing microwave telecommunications radiations such as Wi-Fi (up to 2.6 GHz), and according to Rayleigh's law which explains that the intensity of scattered electromagnetic radiation (Jscat) is proportional to f 4 (where f is the frequency of the radiation) when the dimensions of the scattering particles - such as the molecules of the air, of the building materials, etc. - are smaller than the wavelength (which is the case for all mobile telephony radiations): Jscat ∝ f 4 (33)]. It is poorly transmitted through solid material. Many new [base] antennas will be required and full-scale implementation will result in antennas every 10 to 12 houses in urban areas,* ***thus massively increasing mandatory exposure****'.*

'[Moreover, apart from the increase in background exposure, 5G is likely to induce significant thermal effects in addition to the already non-thermal ones, again due to its significantly higher frequency (34)]'.

'With "the ever more extensive use of wireless technologies," (35) nobody can avoid to be exposed. Because on top of the increased number of 5G-transmitters (even within housing, shops and in hospitals) according to estimates, "10 to 20 billion connections" (36) (to refrigerators, washing machines, surveillance cameras, self-driving cars and buses, etc.) will be parts of the Internet of Things. All these together can cause a substantial increase in the total, long term RF-EMF exposure to all EU citizens'.

Harmful effects of RF-EMF exposure have already been proven. *'Over 230 scientists from >40 countries [now 252 scientists from 43 nations] (37) have expressed their "serious concerns" regarding the ubiquitous and increasing exposure to EMF generated by electric and wireless devices already before the additional 5G roll-out. They refer to the fact that "numerous recent scientific publications have shown that EMFs affect living organisms at levels well below most international and national guidelines". Effects include increased cancer risk, cellular stress, increase in harmful free radicals, genetic damages, structural and functional changes of the reproductive system, learning and memory deficits, neurological disorders, and negative impacts on general well-being in humans. Damage goes well beyond the human race, as there is growing evidence of harmful effects (38) to both plants (39) and animals (40)'.*

'After the scientists' appeal was written in 2015 additional research has convincingly confirmed serious health risks from RF-EMF fields from wireless technology. The world's largest study (25 million US dollar) National Toxicology Program (NTP) (41), shows statistically significant increase in the incidence of brain and heart cancer in animals exposed to EMF [intensities] below the ICNIRP (International Commission on Non-Ionizing Radiation Protection) guidelines followed by most countries. These results support results in human epidemiological studies (17) on RF radiation and brain tumour risk. A large number of peer-reviewed scientific reports (2) demonstrate harm to human health from EMFs'.

'The International Agency for Research on Cancer (IARC), the cancer agency of the World Health Organization (WHO), in 2011 concluded that EMFs of frequencies 30 KHz - 300 GHz are possibly carcinogenic to humans (Group 2B) (12,42). However, new studies like the NTP study mentioned above and several epidemiological investigations including the latest studies on mobile phone use and brain cancer risks confirm that RF-EMF radiation is carcinogenic to humans (17)'.

'The EUROPA EM-EMF Guideline 2016 (1) states that "there is strong evidence that long-term exposure to certain EMFs is a risk factor for diseases such as certain cancers, Alzheimer's disease, and male infertility…Common EHS (electromagnetic hypersensitivity) symptoms include headaches, concentration difficulties, sleep problems, depression, lack of energy, fatigue, and flu-like symptoms"'.

'An increasing part of the European population is affected by ill health symptoms that have for many years been linked to exposure to EMF and wireless radiation in the scientific literature. The International Scientific Declaration on EHS & multiple chemical sensitivity (MCS), Brussels (43), declares that: "In view of our present scientific knowledge, we thereby stress all national and international bodies and institutions… to recognize EHS and MCS as true medical conditions which acting as sentinel diseases may create a major public health concern in years to come worldwide i.e. in all the countries implementing unrestricted use of electromagnetic field-based wireless technologies and marketed chemical substances… ***Inaction is a cost to society*** *and is not an option anymore… we unanimously acknowledge this serious hazard to public health…that major primary prevention measures are adopted and prioritized, to face this* ***worldwide pan-epidemic*** *in perspective"'.*

Precautions. *'The Precautionary Principle (44) was adopted by EU 2005 (45): "When human activities may lead to morally unacceptable harm that is scientifically plausible but uncertain, actions shall be taken to avoid or diminish that harm"'.*

'The Council of Europe Resolution 1815 (46): "Take all reasonable measures to reduce exposure to electromagnetic fields, especially to radio frequencies from mobile phones, and particularly the exposure to children and young people who seem to be most at risk from head tumours…Assembly strongly recommends that the ALARA (as low as reasonably achievable) principle is applied, covering both the so-called thermal effects and the athermic [non-thermal] or biological effects of electromagnetic emissions or radiation" and to "improve risk-assessment standards and quality"'.

'The Nuremberg code (47) applies to all experiments on humans, thus including the roll-out of 5G with new, higher

RF-EMF exposure. All such experiments: "should be based on previous knowledge (e.g., an expectation derived from animal experiments) that justifies the experiment. No experiment should be conducted, where there is an a priori reason to believe that death or disabling injury will occur; except, perhaps, in those experiments where the experimental physicians also serve as subjects," Nuremberg code pts 3-5 (47). Already published scientific studies show that there is "a priori reason to believe" in real health hazards'.

'The European Environment Agency (48) is warning for "Radiation risk from everyday devices" in spite of the radiation being below the WHO/ICNIRP standards (49). EEA also concludes: "There are many examples of the failure to use the precautionary principle in the past, which have resulted in serious and often irreversible damage to health and environments…harmful exposures can be widespread before there is both 'convincing' evidence of harm from long-term exposures, and biological understanding [mechanism] (50) of how that harm is caused"'.

'Safety guidelines' protect the industry, not health. *'The current ICNIRP "safety guidelines" are obsolete. All proofs of harm mentioned above arise although the radiation is below the ICNIRP "safety guidelines" (49). Therefore new safety standards are necessary. The reason for the misleading guidelines is that "conflict of interest of ICNIRP members (10) due to their relationships with telecommunications or electric companies undermine the impartiality that should govern the regulation of Public Exposure Standards for non-ionizing radiation…To evaluate cancer risks it is necessary to include scientists with competence in medicine, especially oncology'.*

'The current ICNIRP/WHO guidelines for EMF are based on the obsolete hypothesis that "The critical effect of RF-EMF exposure relevant to human health and safety is heating of exposed tissue" (51). However, scientists have proven that many different kinds of illnesses and harms are caused without heating ("non-thermal effect") (52) at radiation levels well below ICNIRP guidelines'.

The authors thus urge the EU to carry out the following. *i) 'To take all reasonable measures to halt the 5G RF-EMF expansion until independent scientists can assure that 5G and the total radiation levels caused by RF-EMF (5G together with 2G, 3G, 4G, and WiFi) will not be harmful for EU-citizens, especially infants, children and pregnant women, as well as the environment'. ii) 'To recommend that all EU countries, especially their radiation safety agencies, follow Resolution 1815 and inform citizens, including, teachers and physicians, about health risks from RF-EMF radiation, <u>how</u> and <u>why</u> to avoid microwave radiation, particularly in/near e.g., daycare centers, schools, homes, workplaces, hospitals and elderly care'. iii) 'To appoint immediately, without industry influence, an EU task force of independent, truly impartial EMF-and-health scientists with no conflicts of interest (to re-evaluate the health risks and: a) To decide about new, safe "maximum total exposure standards" for all microwave radiation within EU. b) To study the total and cumulative exposure affecting EU-citizens. c) To create rules that will be prescribed/enforced within the EU about how to avoid exposure exceeding new EU "maximum total exposure standards" concerning all kinds of EMFs in order to protect citizens, especially infants, children and pregnant women'. iv) 'To prevent the wireless/telecom industry through its lobbying organizations from persuading EU-officials to make decisions about further propagation of RF radiation including 5G in Europe'. v) 'To favor and implement wired digital telecommunication instead of wireless'.*

First reply from the EU. A reply from the EU was sent on October 13, 2017 by the Directorate-General Health and Food Safety (Public health, country knowledge, crisis management) in Luxembourg. It was not replied to by the Commissioner Andriukaitis, but instead by Mr. John F. Ryan, Director (for the full text please see: http://www.5gappeal.eu/wp-content/uploads/2018/06/reply_ryan.pdf). Some paragraphs are presented below:

'It is worth underlining that for the Commission health protection is always taken into account in all of its proposals. There is consistent evidence presented by national and international bodies (International Commission on Non Ionising Radiation Protection - ICNIRP, Scientific Committee on Emerging and Newly Identified Health Risks - SCENIHR) that exposure to electromagnetic fields does not represent a health risk, if it remains below the limits set by Council Recommendation 1999/519/EC (https://ec.europa.eu/health//sites/health/files/electromagnetic_fields/docs/emf_rec519_en.pdf)'.

'The Scientific Committee on Emerging and Newly Identified Health Risks, which is independent of the Commission, has a standing mandate to provide this update'.

'It has already produced five opinions. The last opinion was adopted in January 2015 on "Potential health effects of exposure to electromagnetic fields". (https://ec.europa.eu/health/scientific_committees/emerging/docs/scenihr_o_041.pdf)'.

'These scientific opinions have not provided any scientific justification for revising the exposure limits (basic restrictions and reference levels) under Council Recommendation 1999/519/EC'.

'Digital technologies and mobile communication technologies, including high speed internet, will be the backbone of Europe's future economy, allowing all citizens to be connected. At the same time, all citizens deserve appropriate protection against electromagnetic fields from all types of sources including from wireless devices'.

'Most 5G networks are expected to use smaller cells than previous generations with lower electromagnetic fields exposure levels. This is confirmed by the experience so far gained. The introduction of 3G and 4G has not increased exposure from environmental fields and this has been published also in peer-reviewed journals. In particular, the introduction of 3G has lowered exposure of mobile phone users for calls, compared to 2G'.

'Related to the issue of the alleged conflicts of interests, the Commission is not aware of any conflicts of interests of members of international bodies such as ICNIRP or the members of SCENIHR. Please be informed that the Ombudsman conclusion in case 208/2015/P concerning conflicts of interests in a Commission expert group on electromagnetic fields is that there was no maladministration by the European Commission (https://www.ombudsman.europa.eu/en/cases/decision.faces/en/78175/html.bookmark)'.

'Please be assured that the Commission will pursue scrutiny of the independent scientific evidence available to ensure the highest health protection of our citizens'.

Comment: There are obvious misconceptions in this reply such as: *'The introduction of 3G and 4G has not increased exposure from environmental fields and this has been published also in peer-reviewed journals'*. On the contrary, numerous peer-reviewed articles have demonstrated that exposure to ambient RF radiation has increased substantially, as discussed (3-6).

In addition, the statement that: *'the Commission is not aware of any conflicts of interests of members of international bodies such as ICNIRP or the members of SCENIHR'* does not represent the scientific evidence of inherent conflicts of interest both in ICNIRP and SCENIHR (9,10). The very Commission seems to be ill-informed or even misinformed, as the EU seems to take information mainly from these two fraudulent organizations, but not from independent researchers. The EU does not seem to rely on sound science and thereby downplays the RF-related risks (7-12,53,54).

b) First rebuttal to the EU and the response. On November 13, 2017, a rebuttal was sent to the EU Commissioner of Health, Dr Andriukaitis. The whole letter can be found at: https://www.environmentandcancer.com/letter-to-vytenis-andriukaitis-13-11-2017/.

'We suppose that you know that Director John F. Ryan, October 13, 2017 replied (Ares 2017 5015409 - Reply to the EU 5G-appeal, and that he said: "There is ***consistent evidence*** *that exposure to electromagnetic fields* ***does not represent a health risk… if below the limits*** *…" His conclusion is based on the opinions of ICNIRP and SCENIHR'.*

'As early as February 1, 2016, in a Comment on SCENIHR to Mr. Ryan it was shown in article and letter by Drs. *[Sage], Carpenter and Hardell, representing BioInitiative and ECERI, that: "The evidence in the SCENIHR Final Opinion on EMF* ***clearly and convincingly establishes the potential for health effects of exposure to electromagnetic fields*** *[EMF]. Based on the evidence provided in this Opinion, the Committee is obligated to draw to the attention of the [EU] Commission that EMF is a new and emerging problem that may pose an actual or potential threat"' (55).*

'In spite of all this, Mr Ryan in his reply to us still continues to claim that EMF 'does not represent a health risk' and - without any other references than ICNIRP and SCENIHR - defends industry's standpoint that EMFs are harmless if below the ICNIRP "safety guidelines". In addition he ignores the IARC evaluations on both ELF-EMF and RF-EMF to be 'possible' human carcinogens, Group 2B'.

'In the 5G-Appeal we urge EU to appoint a truly independent expert group of EMF-and-health researchers (contrary to ICNIRP and SCENIHR) to decide about new safe guidelines for EMF exposure. It is imperative to ***immediately apply EU:s Precautionary Principle (and ALARA)*** *enabling rapid response to stop distribution of 5G products in order to diminish the harm that has already been proven by scientists. A* ***European pan-epidemic*** *may follow if you don't do so'.*

Second reply from EU on 29 November, 2017. This was sent from the European Commission, Cabinet of Commissioner Vytenis Andriukaitis, Head of Cabinet Brussels, written by Arūnas Vinciunas. The full reply can be found at: http://www.5gappeal.eu/wp-content/uploads/2018/06/reply_vinciunas.pdf).

'When Mr Ryan answered your email, in which you stated your disagreement with the Commission's stance on the 5G appeal, he presented the conclusions of roughly two decades of research on the potential health effects of EMF, and the views expressed in the Scientific Opinions produced by the independent Scientific Committees. [ICNIRP - International Commission on Non-Ionizing Radiation Protection and SCENIHR - Scientific Committee on Emerging and Newly Identified Health Risks]. The Committee's last Opinion on EMF, published in 2015, is based on hundreds of peer-reviewed studies published worldwide and is the fourth Opinion on EMF published since EMF legislation was adopted in 1999. The Committee's conclusion in this latest Opinion was based on exposure studies, epidemiological studies and in vivo and in vitro studies, and studies on any suggestions of causality were considered for the weighting'.

'The Commission services are confident that the advice provided by the Scientific Committees is unbiased, accurate and scientifically sound and therefore do not feel it necessary to appoint an independent expert group of EMF-and-health researchers to discuss new safe guidelines for EMF exposure'.

'The recourse to the EU's Precautionary Principle to stop the distribution of 5G products appears too drastic a measure. We first need to see how this new technology will be applied and how the scientific evidence will evolve. Please rest assured that the Commission will keep abreast of future developments in view of safeguarding the health of the European citizens at the highest level possible and in line with its mandate'.

Comment: This reply from EU is far from adequate. It does not represent a sound evaluation of the RF-related radiation risks based on published peer-reviewed studies. This is again outlined in our response to the EU.

c) Second rebuttal to the EU and the response. On January 17, 2018, a letter was sent to Dr. Vytenis Andriukaitis, EU Commissioner of Health. Sections of this letter are presented

below and the full text can be found at: https://www.environmentandcancer.com/letter-to-vytenis-andriukaitis-and-donald-tusk-17-01-18/.

'Following the letter and the Scientist Appeal calling for a moratorium on 5G ("The 5G Appeal"), which we sent to your office, we received a response from Director John F. Ryan on October 13, 2017 and then, upon our reply, a letter from Mr. Arūnas Vinciūnas dated 29.11.2017'.

'Despite the conclusive evidence presented in our letters, both Director Ryan and Mr. Vinciūnas gave generic responses and continued to claim that EMF "does not represent a health risk". In doing so they only refer to ICNIRP and SCENIHR opinions without explaining why they disregarded the compelling evidence and references under the 5G-Appeal headline: "Harmful effects of RF-EMF exposure are already proven"'.

'The ICNIRP exposure limits are dependent on an unproven hypothesis that "only heat from EMF can cause health hazards". This hypothesis has clearly been rejected in a large number of scientific studies'.

'Both EU officials defend the industry-supportive standpoint that EMFs are harmless if below the ICNIRP "guidelines". However, many of the scientists on both ICNIRP's and SCENIHR's committees are connected to the telecom industry with obvious conflicts of interest'.

'Mr Vinciūnas stated in his letter: "The recourse to the EU's Precautionary Principle to stop the distribution of 5G products appears ***too drastic a measure****." Mr Vinciūnas finishes his letter: "we need to see … how the scientific* ***evidence will evolve****"'.*

'According to Communication from the Commission on the precautionary principle: "Whether or not to invoke the Precautionary Principle is a decision exercised where scientific information is insufficient, inconclusive, or uncertain and where there are indications that the possible effects on the environment, or human, animal or plant health may be potentially dangerous and inconsistent with the chosen level of protection." That describes the situation with 5G perfectly. Existing data shows that 5G frequencies [radiations] are hazardous. However, additional studies will be necessary to fully determine the extent of the risk'.

Third reply from the EU. This letter was replied to on April 27, 2018 by Mr. Arūnas Vinčiūnas from the Cabinet of Commissioner Vytenis Andriukaitis. For the full third reply to our appeals please see: https://www.environmentandcancer.com/answer-from-arunas-vinciunas-27-04-2018/.

'Thank you very much for your letter of 15 March 2018 which was also transmitted by email on 19 March. Commissioner Andriukaitis has asked me to reply to you on his behalf'.

'Finally, let me refer to the previous correspondence you have had with John F. Ryan, Director of Public Health and me (29 November 2017, 13 October 2017 and 19 February 2018) where we have comprehensively explained our position with regard to the arguments you have raised. It is my view that we have now extensively deliberated on the matter and that we should refrain from further repetition'.

'Please rest assured that the Commission will remain committed to safeguarding the health of the European citizens, at the highest level possible and in line with his mandate'.

d) Third rebuttal to the EU and the response. This rebuttal had the title *"Request for a moratorium on the 5G rollout. Request for guidelines based on independent research. Request for documents showing that 5G is safe"*. On May 20, 2019 a letter with these requests was sent to Dr Karmenu Vella, EU Commissioner of Environment and Dr Vytenis Andriukaitis, EU Commissioner of Health. For the full text please see: https://www.environmentandcancer.com/letter-to-vytenis-andriukaitis-20-05-2019/.

'We make reference to the Precautionary Principle (PP) (56) It "enables a rapid response to be given in the face of a possible danger to human health…institutions may take protective measures without having to wait until the reality… of risks become apparent … preventive action should be taken" (57). Research confirms 5G to be a risk to all life on earth'.

'With this communication we touch upon three points:' i) 'Firstly, we request in the 5G Appeal to EU (www.5gappeal.eu), of which you are a public servant and representative, to declare an immediate ***moratorium*** *on 5G deployment. The 5G appeal to EU is now confirmed by 230+ truly independent scientists and physicians from 36 countries. The Space 5G appeal (58) has more than 83,000 affirmations from 168 countries. According to PP (56) and EU IP/00/96 (59) "protection of health takes precedence over economic considerations."' ii) 'Secondly, we ask for groups of truly industry-independent researchers to establish* ***new guidelines for exposure****. An "In-depth analysis" of the deployment of 5G (60), published by EU in April 2019, needs to be seriously considered. It stated that" One aspect, for example, that is not well understood today is the unpredictable propagation patterns that could result in unacceptable levels of human exposure to electromagnetic radiation."(p. 6)'. iii) 'Thirdly, with this letter we are formally requesting, in accordance with Art. 42 (61) on EU Fundamental Rights,* ***access to all documents*** *in your possession, either created by you or at your disposal, related to the effects of EMF to human health and the environment. Once in possession of such a list, we will decide which of those documents, if any, are of interest and show that 5G is safe. The list of the documents, and the ways to access them, should be sent to the email addresses below'.*

'We note that, while the EU is eagerly promoting the rollout of 5G, a new EU report admits (60) "the problem is that currently it is not possible to accurately simulate or measure 5G emissions in the real world" (p. 12). *"Significant concern is emerging over the possible impact on health and safety arising from potentially much higher exposure to radiofrequency electromagnetic radiation arising from 5G" (p. 4). The EU report also stresses dangers: "Increased exposure*

may result not only from the use of much higher frequencies in 5G but also from the potential for the aggregation of different signals, their dynamic nature, and the complex interference effects that may result, especially in dense urban areas." (p. 11)'.

Fourth reply from the EU. Finally, a response was delivered by the EU on September 5, 2019, although with reference to the wrong date of our letter. It was sent by Arunas Vinciunas from the Cabinet of Commissioner Vytenis Andriukaitis. The full response can be read at: https://www.environmentandcancer.com/answer-from-arunas-vinciunas-05-09-2019/.

'Thank you for your email of 7 July 2019 to Commissioner Andriukaitis in which you request to halt the 5G expansion in the EU immediately in order to allow a moratorium for industry independent research. Commissioner Andriukaitis has asked me to reply to you on his behalf'.

'In my latter note to you I already expressed my view that we had extensively deliberated on the matter and that we should refrain from further repetition'.

'As regards your request to halt the launch of the new 5G technology, I would like to confirm the view already expressed in my note of 29 November 2017 to you that stopping the distribution of 5G products appears too drastic a measure. I repeat that first there is a need to see how this new technology will be applied and how the scientific evidence will evolve'.

'Concerning your call for a scientific evaluation and new guidelines for exposure, the second point you have raised, let me stress that the Commission will review the situation once the review of the guidelines issued by the International Commission on Non-Ionizing Protection (ICNIRP) will be finalised which is expected in due course'.

'As regards your third point, documents related to the effects of electromagnetic fields to human health and the environment, please be referred to the opinion of the Commission's Scientific Committee on Emerging and Newly Identified Health Risks of 20 January 2015 on potential health effects of exposure to electromagnetic fields (EMF) (https://ec.europa.eu/health/scientific_committees/emerging/docs/scenihr_o_041.pdf) that provides an extensive list of references to scientific literature on this issue'.

Comment on the fourth reply from the EU appeal: There is no new evidence of the safety in this letter from EU compared with the earlier replies. Of note, the EU relies on documentation of risk only on old and biased selection of references in one single report from SCENIHR (https://ec.europa.eu/health/scientific_committees/emerging/docs/scenihr_o_041.pdf). Thus, EU officials still seem to base the evaluation of the health risks on reports from the ICNIRP and SCENIHR that have been seriously criticized. Of note, the EU relies on a report from 2015 as to scientific publications on the safety of 5G, a technology that was not developed during that time. This suggests that perhaps the EU is reluctant to deal with the safety issues associated with 5G technology.

e) Fourth rebuttal to the EU. On October 24, 2019 a fourth rebuttal was sent to the EU (https://www.environmentandcancer.com/letter-to-arunas-vinciunas-24-10-2019). We wrote that *'Specifically now, as we wish to assist the Commissioner in giving due response, it can be further specified from this side that we need the **list of documents** related to EMFs created by RF/Radiofrequencies (so: not by ELF) and even more specifically, to the list of those documents based on which the Commission is basing its current position that 5G should not be stopped nor subject to a moratorium (see the statement of your letter that "first there is a need to see how this new technology will be applied and how the scien6fic evidence will evolve"). We leave aside our total disagreement on the merits of such position at this time: formally, we are entitled to receive from you such a list of documents based on which the Commissioner determined that 5G is safe. Based on that list we will decide which of those documents, are of interest. Please provide such list by email no later than October 31, 2019. This is urgent'.*

Fifth reply from the EU. In this response, dated December 19, 2019, it was stated that new ICNIRP guidelines are expected. Thus, the same approach to this issue as previously and no new commitment (https://www.environmentandcancer.com/answer-from-martin-seychell-19-12-2019).

Appeals to the Nordic Prime Ministers

The 5G Appeal was also sent to the Nordic Prime Ministers (https://www.environmentandcancer.com/letter-to-nordic-ministers-27-6-2018/); (https://www.environmentandcancer.com/letter-to-nordic-ministers-5-3-2019/). The only reply, dated March 29, 2019, was sent from the Swedish government (Ministry of Enterprise and Innovation, Mari Mild). It was stated that the government relies on Swedish Radiation Safety Authority (SSM) and their yearly update of health risks and that no new health risks have been reported. According to the letter there is no reason for a moratorium on the deployment of 5G, see (in Swedish) (https://www.miljoochcancer.com/svar-fran-naringsdepartementet-29-3-2019/). SSM relies on ICNIRP.

Discussion

Our experience with the EU and the Governments of the Nordic countries suggests that the majority of decision makers are scientifically uninformed on health risks from RF radiation (62). In addition, they seem to be uninterested to being informed by scientists representing the majority of the scientific community, i.e., those scientists who are concerned about the increasing evidence or even proof of harmful health effects below the ICNIRP guidelines (www.emfscientist.org). Instead, they rely on evaluations with inborn errors of conflicts, such as ICNIRP. In fact, the ICNIRP, with the support of WHO and major telecommunications companies, has been rather successful in implementing their views in the EU and worldwide. Their guidelines seem to be based on the omission of scientific facts. Thus, their possible ignorance of the health risks is of concern, as well as their reluctance to adhere to warnings from large numbers of scientists around the world.

It is striking that 5G is deployed without previous scientific evaluation of health risks. Not only cancer risks, but also other health effects such as fertility, cognitive and neurobehavioral effects, oxidative stress and electromagnetic hypersensitivity (EHS) have been associated with RF exposure [for a more detailed discussion on this tope, please see previous publications (1,7,8,28,35)]. It is thus noteworthy that the ICNIRP thermal paradigm is still used for the evaluation of the health risks associated with RF radiation. One issue of major concern is that there seems to be conflicts of interest among persons in the evaluating groups. Furthermore the same persons may often be found in different bodies, thereby in fact citing themselves representing a cartel (https://www.saferemr.com/2018/07/icnirps-exposure-guidelines-for-radio.html). This has been outlined in peer-reviewed publications (9,10).

This is also an ethical question. Thus, it would not be possible to test a new drug on individuals without information and signed permission by each individual. Certainly, this principle should apply to 5G that is furthermore, mandatory. Exposure to RF radiation from 5G must be regarded as a medical experiment with potential health risks, some known and expected based on current knowledge, some unknown since this is a new untested technology. A letter of information to those exposed must be sent for informed consent. However, it must be concluded that such a letter, affirming no risk, cannot be formulated based on the limited number of studies on 5G, in fact most of them with no assurance of no risks.

This is also a moral question for all the individuals involved in the propagation of 5G. It is to be noted that individuals within e.g., ICNIRP, national governmental bodies and the EU, partly a cartel, seem to neglect scientific warnings. They instead seem to follow the no-risk paradigm. It is thus questionable as to how it is possible to thereby disregard the diseases caused by this technology and to not consider the affected persons.

Taking the history of e.g., tobacco and smoking and the long period of time it took for cancer classification into account, it is fully understandable that RF radiation is still in the beginning of that history. However, if no action is currently taken, the costs to society will most likely be very high in terms of premature deaths, deteriorated public health and damage to the ecological system. It is however, important to publish the history of neglected RF radiation warnings. The EU seems to perhaps lacking in that respect. It must be concluded that the polluter has to pay the full cost of harm from this technology (63). Those in responsible positions in governments and organizations intended to protect the public and the environment from harm (WHO and ICNIRP), but who fail to do so by ignoring the increasing warnings from scientists worldwide about the dangers of 5G, should also be held responsible for the harm to the public that they thereby induce (64). No doubt damage to the environment by the business sector may be substantial (https://www.theguardian.com/environment/2010/feb/18/worlds-top-firms-environmental-damage).

The EU principle that the Polluter Pays (Article 191, pt 2) states: *'Union policy on the environment shall aim at* ***a high level of protection taking*** *into account the diversity of situations in the various regions of the Union.* ***It shall be based on the precautionary principle*** *and on the principles that* ***preventive action should be taken****, that environmental damage should as a priority be rectified at source and that* ***the polluter should pay****'.* (https://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:12008E191:EN:HTML).

'*The fundamental principle of this Directive should therefore be that an operator whose activity has caused the environmental damage or the imminent threat of such damage is to be held financially liable, in order to induce operators to adopt measures and develop practices to minimise the risks of environmental damage so that their exposure to financial liabilities is reduced' (65) (https://eur-lex.europa.eu/legal-content/EN/TXT/HTML/?uri=CELEX:32004L0035&from=EN).*

The industry tries to convince us that the super high frequencies of 5G are so weak and its millimeter waves will penetrate only the outer surface of the skin. The opposite was proven in USSR research already in 1977 (https://www.cia.gov/library/reading-room/docs/CIA-RDP88B01125R000300120005-6.pdf). High frequencies (37-60 GHz), which will be used in 5G, caused several kinds of detrimental effects in experimental rats. The high frequencies seem to be worse than the lower frequencies. The USSR experiments were made more than 40 years ago - when we had no digital pulsed radiation - with a generator producing sinus curves. Peaks of pulsed radiation used in 5G with unpredictable intensity changes seem to be an important parameter for the bioactivity of RF radiation (29).

In conclusion, this article demonstrates that the EU has given mandate to a 13-member, non-governmental private group, the ICNIRP, to decide upon the RF radiation guidelines. The ICNIRP, as well as SCENIHR, are well shown not to use the sound evaluation of science on the detrimental effects of RF radiation, which is documented in the research which is discussed above (9,10,21-24,54,55). These two small organizations are producing reports which seem to deny the existence of scientific published reports on the related risks. It should perhaps be questioned whether it is in the realm of protecting human health and the environment by EU and whether the safety of EU citizens and the environment can be protected by not fully understanding the health-related risks.

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Availability of data and materials

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Authors' contributions

Both authors (LH and RN) participated in the conception, design and writing of the manuscript, and have read and approved the final version.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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REPUBBLICA ITALIANA

IN NOME DEL POPOLO ITALIANO

Il Tribunale Amministrativo Regionale per il Lazio

(Sezione Terza Quater)

ha pronunciato la presente

SENTENZA

sul ricorso numero di registro generale 8373 del 2018, proposto da

Associazione per la prevenzione e la lotta all'elettrosmog, in persona del legale rappresentante pro tempore, rappresentata e difesa dagli avvocati Renato Ambrosio, Stefano Bertone, Chiara Ghibaudo, Luigi M. Angeletti, con domicilio digitale come da PEC da Registri di Giustizia e domicilio eletto presso lo studio dell'avv.to Marco De Fazi in Roma, via della Giuliana n. 44;

contro

Ministero della Salute; Ministero dello Sviluppo Economico; Ministero dell'Istruzione, dell'Università e della Ricerca; Ministero dell'Ambiente e della Tutela del Territorio e del Mare, in persona del legale rappresentante pro tempore, rappresentati e difesi dall'Avvocatura Generale dello Stato, domiciliata ex lege in Roma, via dei Portoghesi n. 12;

nei confronti

Brondi s.p.a., in persona del legale rappresentante pro tempore, rappresentata e difesa dagli avvocati Riccardo Prandi, Alessandro Massaia, Gianluca Contaldi, con domicilio digitale come da PEC da Registri di Giustizia e domicilio eletto presso lo studio dell'avv.to Gianluca Contaldi in Roma, via Pier Luigi da Palestrina n. 63;

per l'accertamento e la declaratoria dell'illegittimità

dell'inerzia serbata dalle Autorità intimate in relazione all'atto di diffida del 28 giugno 2017, formulato dalla ricorrente e diretto a promuovere l'adozione di tutti i provvedimenti finalizzati all'informazione capillare della popolazione, compresa la fascia dei soggetti più a rischio (bambini, adolescenti) sui rischi a breve e lungo termine per la salute dovuti all'uso dei telefoni mobili (cellulari e cordless) e sulle indispensabili misure cautelative da adottare durante il loro utilizzo;

per il conseguente accertamento dell'obbligo di provvedere in capo alle Autorità intimate in relazione al medesimo atto di diffida, mediante l'adozione di ogni idoneo provvedimento espresso, finalizzato ad assicurare alla popolazione idonea informazione sui rischi per la salute dei cittadini, a breve e lungo termine, quali descritti nelle più recenti acquisizioni scientifiche, dovuti all'uso dei telefoni mobili (cellulari e cordless) e sulle indispensabili misure cautelative da adottare durante il loro utilizzo, con particolare riferimento alla fascia dei soggetti più a rischio (bambini, adolescenti);

nonché per l'accertamento

dell'obbligo in capo al Ministero della Salute, e/o al Ministero dell'Ambiente, e/o al Ministero dello Sviluppo Economico (già Ministero dell'Industria) e/o al Ministero dell'Istruzione, dell'Università e della Ricerca, tenendo conto della multiculturalità presente in Italia, di provvedere all'emanazione senza ritardo del decreto di cui all'art. 12 della legge 22 febbraio 2001, n. 36 "Legge quadro sulla protezione dalle esposizioni a campi elettrici, magnetici ed elettromagnetici", anche al fine di indicare al pubblico "le informazioni che i fabbricanti di apparecchi e dispositivi, in particolare di uso domestico, individuale o lavorativo, generanti campi elettrici, magnetici ed elettromagnetici, sono tenuti a fornire agli utenti, ai lavoratori e alle lavoratrici, mediante apposite etichettature o schede informative";

Visti il ricorso e i relativi allegati;
Visti gli atti di costituzione in giudizio del Ministero della Salute, del Ministero dello Sviluppo Economico, del Ministero dell'Istruzione dell'Università e della Ricerca, del Ministero dell'Ambiente e della Tutela del Territorio e del Mare e della società Brondi s.p.a.
Visti tutti gli atti della causa;
Relatore nella camera di consiglio del giorno 13 novembre 2018 il dott. Paolo Marotta e uditi per le parti i difensori come specificato nel verbale;

Con ricorso notificato in data 30 giugno 2018 e depositato il 13 luglio successivo, l'Associazione ricorrente ha impugnato il silenzio – inadempimento asseritamente formatosi sulla istanza – diffida del 28 – 30 giugno 2017, diretta all'adozione da parte delle Autorità intimate di tutte le iniziative finalizzate ad informare la popolazione sui danni a breve e lungo termine connessi all'uso dei telefoni mobili (cordless e cellulari).
La parte ricorrente ha formulato anche istanza cautelare.
A fondamento della propria legittimazione ad agire, la ricorrente richiama il proprio Statuto e segnatamente l'art. 6 dello Statuto, che individua quale scopo dell'associazione quello di tutelare la salute degli esseri viventi e dell'ambiente dall'esposizione ai campi magnetici ed elettromagnetici.
Quale fondamento giuridico dell'obbligo di provvedere, la ricorrente richiama le seguenti fonti normative: l'art. 32 della Costituzione; la legge 13 marzo 1958 n. 296; gli artt. 1, 4, 10 e 12 della l. 22 febbraio 2001 n. 36 (legge quadro sulla protezione dalle esposizioni ai campi elettrici, magnetici ed elettromagnetici), la direttiva 1999/5/CE; l'art. 2043 c.c.
Si sono costituite le Amministrazioni intimate, eccependo, in via preliminare, l'inammissibilità del ricorso sotto diversi profili e contestando, nel merito, la fondatezza delle doglianze formulate dalla parte ricorrente.
Si è costituita in giudizio anche la società Brondi s.p.a., eccependo l'inammissibilità del ricorso per difetto di giurisdizione del giudice adito, il difetto di legittimazione della ricorrente, l'insussistenza dell'obbligo di provvedere, il difetto di integrità del contraddittorio, il difetto dei presupposti per la concessione della misura cautelare.
Con memoria depositata in data 13 ottobre 2018 la ricorrente, pur riconoscendo l'inammissibilità del rito del silenzio nei confronti degli atti di natura normativa, si è soffermata a considerare la mancata attuazione della campagna di informazione e di educazione ambientale, di cui all'art. 10 della l. n. 36/2001.
Nel corso del giudizio le parti costituite hanno avuto modo di rappresentare compiutamente le rispettive tesi difensive.
All'udienza camerale del 13 novembre 2018, su richiesta delle parti, come da verbale, il ricorso è stato trattenuto in decisione.
Preliminarmente, in accoglimento della eccezione sollevata dalle Amministrazioni resistenti, deve essere dichiarata l'inammissibilità parziale del ricorso, per difetto assoluto di giurisdizione, in ordine al mancato esercizio da parte delle Amministrazioni intimate di poteri di natura normativa.
In prima istanza, infatti, l'Associazione ricorrente si duole dell'inerzia delle Amministrazioni intimate rispetto alla attuazione dell'art. 12, comma 1, della l. n. 36/2001, a norma della quale: "1. Con decreto del Ministro dell'ambiente, di concerto con il Ministro della sanità, previo parere del Comitato e sentite le competenti Commissioni parlamentari, sono stabilite, entro centoventi giorni dalla data di entrata in vigore della presente legge, tenendo conto anche degli orientamenti e degli atti dell'Unione europea in materia di inquinamento elettromagnetico, tutela dei consumatori e istruzioni per l'uso dei prodotti, le informazioni che i fabbricanti di apparecchi e dispositivi, in particolare di uso domestico, individuale o lavorativo, generanti campi elettrici, magnetici ed elettromagnetici, sono tenuti a fornire agli utenti, ai lavoratori e alle lavoratrici, mediante apposite etichettature o schede informative. Le

informazioni devono riguardare, in particolare, i livelli di esposizione prodotti dall'apparecchio o dal dispositivo, la distanza di utilizzo consigliata per ridurre l'esposizione al campo elettrico, magnetico ed elettromagnetico e le principali prescrizioni di sicurezza. Con lo stesso decreto sono individuate le tipologie di apparecchi e dispositivi per i quali non vi è emissione di campo elettrico, magnetico ed elettromagnetico, o per i quali tali emissioni sono da ritenersi cosi basse da non richiedere alcuna precauzione".

Orbene, risulta non controverso tra le parti che questa disposizione normativa non ha ancora trovato attuazione. Sennonché il rito del silenzio – inadempimento non può essere utilizzato per costringere le Amministrazioni intimate alla adozione del decreto ministeriale, cui fa riferimento la disposizione normativa sopra richiamata, costituendo esso un atto di natura normativa (più precisamente, regolamentare).

Per pacifica giurisprudenza infatti, è esclusa, ai sensi dell'art. 7, co. 1, ultimo periodo, c.p.a., la possibilità di sindacare, con lo speciale rito del silenzio, la mancata adozione, da parte degli organi titolari del relativo potere, di atti normativi (leggi, atti aventi forza di legge, regolamenti), venendo in rilievo ambiti nei quali l'Amministrazione esprime scelte di natura politica (Consiglio di Stato, sez. V, 22 gennaio 2015 n. 273).

Ne consegue che la domanda formulata dalla odierna ricorrente deve essere considerata in parte qua inammissibile, per difetto assoluto di giurisdizione, dovendo ritenersi che la mancata adozione del decreto ministeriale, di cui all'art. 12 della l. n. 36/2001, assuma rilevanza solo sul piano della responsabilità politica degli organi di governo e, comunque, non sia coercibile sul piano giuridico con il ricorso al rito del silenzio – inadempimento.

Sempre in via preliminare, deve essere esaminata l'eccezione di inammissibilità del ricorso, per difetto di legittimazione attiva dell'Associazione ricorrente, sollevata dalle Amministrazioni resistenti. Queste ultime sostengono che, non figurando tra le Associazioni di protezione ambientale individuate con decreto del Ministro dell'Ambiente di cui all'art. 13 della l. n. 349/1986, l'Associazione ricorrente sarebbe priva di legittimazione ad agire.

L'eccezione è infondata.

Ritiene il Collegio di aderire a quell'orientamento giurisprudenziale secondo il quale l'esplicita legittimazione, ai sensi degli artt. 13 e 18 della legge 8 luglio 1986, n. 349, delle Associazioni ambientalistiche di dimensione nazionale e ultraregionale all'azione giudiziale non esclude, di per sé sola, analoga legittimazione ad agire in un ambito territoriale ben circoscritto, e ciò anche per i comitati che si costituiscono al precipuo scopo di proteggere l'ambiente, la salute e/o la qualità della vita delle popolazioni residenti su tale circoscritto territorio. Le previsioni normative citate hanno introdotto un criterio di legittimazione "legale" "aggiuntivo", e non "sostitutivo", rispetto ai criteri elaborati precedentemente dalla giurisprudenza per l'azionabilità in giudizio dei c.d. "interessi diffusi". Ne consegue che il giudice amministrativo può riconoscere, caso per caso, la legittimazione a impugnare atti amministrativi a tutela dell'ambiente a favore di associazioni locali (indipendentemente dalla loro natura giuridica), purché le stesse a) perseguano statutariamente in modo non occasionale obiettivi di tutela ambientale, b) abbiano un adeguato grado di rappresentatività e stabilità e c) svolgano la propria attività in un'area di afferenza ricollegabile alla zona in cui è situato il bene a fruizione collettiva che si assume leso (Consiglio di Stato, sez. V, 17 ottobre 2012 n. 5295; in senso conforme, Consiglio di Stato, sez. VI, 12 giugno 2015 n. 2894).

Orbene, dagli atti depositati in giudizio emerge che:

- l'Associazione ricorrente è stata costituita ai sensi della l. 7 dicembre 2000 n. 383;
- lo Statuto dell'Associazione stabilisce, all'art. 5, che la durata dell'Associazione è illimitata, e all'art. 6, che lo scopo principale dell'Associazione medesima è quello di "promuovere, attraverso l'azione dei suoi Soci, la tutela della salute e della integrità degli esseri viventi e dell'ambiente dall'esposizione ai campi elettrici, magnetici ed elettromagnetici, statici o variabili, generati artificialmente e da tutte le forme di inquinamento chimico, fisico, radioattivo e biologico";
- l'ambito di operatività dell'Associazione è individuato nel territorio della Regione Veneto (art. 7 dello Statuto).

Ritiene conseguentemente il Collegio che conformemente all'orientamento giurisprudenziale sopra richiamato non vi siano ragioni per denegare alla Associazione ricorrente la legittimazione ad agire per la tutela della salute e

dell'ambiente dall'inquinamento elettromagnetico indotto dall'uso dei telefoni mobili (cellulari e cordless).

Il Collegio non ravvisa poi la necessità di disporre l'integrazione del contraddittorio, sulla base della eccezione sollevata dalla società Brondi s.p.a., non venendo in rilievo rispetto alla fattispecie dedotta in giudizio controinteressati in senso tecnico.

Nel merito, il Collegio rileva che, facendo seguito ad altre precedenti istanze, l'Associazione ricorrente, con istanza del 28 giugno 2017, ha diffidato il Ministero della Salute, il Ministero dell'Ambiente e il Ministero dello Sviluppo Economico ad adottare i seguenti atti:

a) ad emanare il decreto di cui all'art. 12 della l. n. 36/2001;

b) ad eseguire una campagna informativa rivolta alla intera popolazione, avente ad oggetto la indicazione delle modalità d'uso e dei rischi per la salute e per l'ambiente connessi all'uso di telefoni cellulari e cordless.

Con il ricorso in esame, dopo essersi lungamente soffermata sui rischi per la salute e per l'ambiente derivanti da un uso improprio degli apparecchi di telefonia mobile soprattutto per gli utenti più giovani di età, sulla base delle ultime ricerche scientifiche, l'Associazione ricorrente ha chiesto l'annullamento del silenzio – inadempimento formatosi per effetto dell'inerzia delle Amministrazioni intimate e che venga accertato l'obbligo delle predette Amministrazioni di provvedere, entro un determinato termine.

Orbene, come sopra evidenziato, la domanda (processuale) dell'Associazione ricorrente diretta ad ottenere l'emanazione del decreto ministeriale di cui all'art. 12 della l. n. 36/2001, è inammissibile per difetto assoluto di giurisdizione, venendo in rilievo il mancato esercizio di poteri di natura normativa.

Rimane invece da scrutinare la fondatezza della domanda formulata dalla ricorrente con riferimento al mancato avvio da parte dei Ministeri competenti ratione materiae di una campagna informativa rivolta alla intera popolazione, avente ad oggetto l'indicazione delle modalità d'uso e dei rischi per la salute e per l'ambiente connessi all'uso degli apparecchi di telefonia mobile (telefoni cellulari e cordless).

L'Associazione ricorrente individua il fondamento giuridico della predetta richiesta nell'art. 10 della l. n. 36/2001, a norma del quale: "Il Ministro dell'ambiente, di concerto con i Ministri della sanità (ora Ministro della salute, n.d.r.), dell'università e della ricerca scientifica e tecnologica e della pubblica istruzione (ora Ministro dell'Istruzione, dell'Università e della Ricerca, n.d.r.), promuove lo svolgimento di campagne di informazione e di educazione ambientale ai sensi della legge 8 luglio 1986 n. 349".

Nel corso del giudizio, l'Associazione ricorrente ha prodotto alcuni documenti tratti dalla letteratura scientifica, dai quali emerge che l'utilizzazione inadeguata dei telefoni cellulari o cordless, comportando l'esposizione di parti sensibili del corpo umano ai campi elettromagnetici, può avere effetti nocivi per la salute umana, soprattutto con riguardo ai soggetti più giovani e, quindi, più vulnerabili, potendo incidere negativamente sul loro sviluppo psico – fisico.

Orbene, i rischi per la salute paventati dall'Associazione ricorrente non sono stati efficacemente contestati dalle Amministrazioni resistenti, che si sono limitate ad invocare l'inammissibilità anche di questa seconda richiesta.

A tale riguardo, ritiene il Collegio che le campagne informative e di educazione ambientale di cui all'art. 10 della l. n. 36/2001 non possano essere sussunte nella categoria degli atti meramente materiali, come sostenuto dalle Amministrazioni resistenti, ma debbano essere ascritte al genus degli atti amministrativi generali, in quanto sono rivolte ad una pluralità indefinita di soggetti, trovano il fondamento giuridico in norme di rango legislativo, presuppongono lo svolgimento di un'attività istruttoria finalizzata alla individuazione dei rischi connessi all'esposizione del corpo umano ai campi elettromagnetici e alla individuazione delle precauzioni da adottare (sia da parte degli utenti che dei produttori dei predetti apparecchi) per limitarne gli effetti potenzialmente nocivi per la salute e hanno lo scopo di sensibilizzare gli utenti in merito ad un uso più consapevole degli apparecchi di telefonia mobile, al fine di salvaguardare il diritto alla salute che è un diritto costituzionalmente tutelato (art. 32 della Costituzione).

Dagli atti depositati in giudizio risulta che già, con nota prot. n. 0001080 –P del 16 gennaio 2012, il Ministero della Salute, in riscontro ad una precedente richiesta di uno dei procuratori della Associazione ricorrente, evidenziava: "…. il tema dei possibili rischi per la salute conseguenti all'uso del cellulare è alla costante attenzione del Ministero della Salute, in particolare a seguito della classificazione stabilita dall'Agenzia Internazionale per la Ricerca sul Cancro nel 2011, di agente possibilmente cancerogeno per l'uomo (categoria 2B) per i campi elettromagnetici in radiofrequenza".

Nella medesima nota, il Ministero della Salute, ha evidenziato che il Consiglio Superiore di Sanità, nel parere del 15 novembre 2011, tenuto conto della posizione formalmente assunta dall'Istituto Superiore di Sanità, "… ha rilevato che allo stato delle conoscenze scientifiche non è dimostrato alcun nesso di causalità tra esposizione a radiofrequenze e patologie tumorali, rimarcando tuttavia come l'ipotesi di un rapporto causale non possa essere del tutto esclusa in relazione ad un uso molto intenso del telefono cellulare…" e che lo stesso Consiglio Superiore di Sanità "… ha quindi raccomandato di mantenere vivo l'interesse della ricerca e della sorveglianza sul tema, in attesa che le nuove conoscenze risolvano le attuali aree di incertezza, suggerendo nel contempo l'avvio di una campagna d'informazione al pubblico al fine di promuovere e incoraggiare un uso responsabile del telefono, soprattutto in relazione ai bambini che tendono ad essere avvicinati all'uso del telefono cellulare in età sempre più precoce", precisando infine: "La campagna di informazione è in fase di preparazione e sarà basata sul quadro delle conoscenze desumibili dalle più autorevoli fonti e organismi nazionali e internazionali".

Nonostante il ragguardevole lasso di tempo intercorso, la preannunciata campagna informativa non risulta essere stata ancora attuata.

Deve conseguentemente essere dichiarato l'obbligo del Ministero dell'Ambiente, del Ministero della Salute e del Ministero dell'Istruzione, dell'Università e della Ricerca, ciascuno per il proprio ambito di competenza, di provvedere, in attuazione di quanto disposto dall'art. 10 della l. n. 36/2001, ad adottare una campagna informativa, rivolta alla intera popolazione, avente ad oggetto la individuazione delle corrette modalità d'uso degli apparecchi di telefonia mobile (telefoni cellulari e cordless) e l'informazione dei rischi per la salute e per l'ambiente connessi ad un uso improprio di tali apparecchi.

La predetta campagna di informazione e di educazione ambientale dovrà essere attuata nel termine di sei mesi dalla notifica o, se anteriore, dalla comunicazione in via amministrativa della presente sentenza, avvalendosi dei mezzi di comunicazione più idonei ad assicurare una diffusione capillare delle informazioni in essa contenute.

In ragione dell'accoglimento parziale delle domande formulate dall'Associazione ricorrente, ricorrono all'evidenza valide ragioni per disporre l'equa compensazione delle spese di giudizio.

P.Q.M.

Il Tribunale Amministrativo Regionale per il Lazio (Sezione Terza Quater), definitivamente pronunciando sul ricorso, come in epigrafe proposto, così dispone:

- dichiara inammissibile, per difetto assoluto di giurisdizione, la domanda di annullamento del silenzio – inadempimento sulla istanza della ricorrente relativa alla emanazione del decreto ministeriale, di cui all'art. 12 della l. n. 36/2001;

- accoglie la domanda di annullamento del silenzio – inadempimento sulla istanza presentata dalla ricorrente, sulla base dell'art. 10 della l. n. 36/2001, e, per l'effetto, dichiara l'obbligo del Ministero dell'Ambiente, del Ministero della Salute e del Ministero dell'Istruzione, dell'Università e della Ricerca, ciascuno per il proprio ambito di competenza, di provvedere (nei termini e con le modalità indicate in motivazione) ad adottare una campagna informativa, rivolta alla intera popolazione, avente ad oggetto l'individuazione delle corrette modalità d'uso degli apparecchi di telefonia mobile (telefoni cellulari e cordless) e l'informazione dei rischi per la salute e per l'ambiente connessi ad un uso improprio di tali apparecchi.

Spese compensate.

Ordina che la presente sentenza sia eseguita dall'autorità amministrativa.

Così deciso in Roma nella camera di consiglio del giorno 13 novembre 2018 con l'intervento dei magistrati:

Giuseppe Sapone, Presidente

Pierina Biancofiore, Consigliere

Paolo Marotta, Consigliere, Estensore

L'ESTENSORE	IL PRESIDENTE
Paolo Marotta	**Giuseppe Sapone**

IL SEGRETARIO

Radiofrequency radiation from nearby mobile phone base stations-a case comparison of one low and one high exposure apartment

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Abstract. Radiofrequency (RF) radiation in the frequency range of 30-300 GHz has, since 2011, been classified as a 'possible' human carcinogen by Group 2B, International Agency for Research on Cancer (IARC) at WHO. This was based on a number of human epidemiology studies on increased risk for glioma and acoustic neuroma. Based on further human epidemiology studies and animal studies, the evidence on RF radiation carcinogenesis has increased since 2011. In previous measurement studies, it has been indicated that high environmental RF radiation levels are present in certain areas of Stockholm Sweden, including in one apartment. Field spatial distribution measurements were performed in the previously measured apartment in Stockholm, which exhibited high RF radiation from nearby base stations. Based on the RF broadband analyzer spot measurements, the maximum indoor E-field topped at 3 V m^{-1} in the bedroom at the 7th floor. The maximum outdoor exposure level of 6 V m^{-1} was encountered at the 8th floor balcony, located at the same elevation and only 6.16 m away from the base station antennas. For comparison, a measurement was made in a low exposure apartment in Stockholm. Here, the maximum indoor field 0.52 V m^{-1} was measured at the corner window, with direct line of sight to the neighboring house with mobile phone base station antennas. The maximum outdoor field of 0.75 V m^{-1} was measured at the balcony facing the same next-door building with mobile phone base station antennas. The minimum field of 0.10 V m^{-1} was registered on the apartment area closest to the center of the building, demonstrating the shielding effects of the indoor walls. Good mobile phone reception was achieved in both apartments. Therefore, installation of base stations to risky places cannot be justified using the good reception requirement argument.

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Key words: radiofrequency radiation, microwaves, mobile phone, base stations, exposure, health, building

Introduction

Public exposure to radiofrequency (RF) electromagnetic fields (EMF) in today's cities may be caused by a number of sources, including mobile phone base stations, TV and radio towers, wireless local area networks (WLAN), emergency services radio network, RF-identification systems, microwave ovens, anti-theft gates etc. Additionally, individual's exposure may be significantly elevated by personal usage of mobile and cordless phones, 2-way radios, WLAN, Bluetooth and other wireless devices. In this study we have focused on the exposure from mobile phone base station antennas. Exposure in two apartments positioned close to mobile phone base station antennas is measured in detail.

Developments in telecommunications technologies have led to widespread use of mobile devices connected to the network in constantly increasing loads. This has resulted also in the public's exposure to RF EMFs. Temporal trends in RF EMF exposure in everyday environments were investigated across European cities of Basel, Ghent and Brussels in 2011-2012 (1). Within a year total RF exposure levels in all investigated outdoor locations combined raised 57.1%. The increase in exposure was most notably observed in outdoor locations due to mobile phone base stations (1).

In many European countries on-site RF exposure measurements have been conducted since the 1990's. Most studies have focused on the mobile communications' frequency bands. A comparative analysis concluded that due to the developments in telecommunications technology, the RF exposure is continuously increasing and is estimated >65% of the total exposure (2). Based on the personal exposure measurements in the EU, the mean RF is generally from 0.10 to 0.26 V m^{-1}; however most of these studies are based on outdoor measurements.

RF field exposure literature in Europe was reviewed, comparing indoor levels to outdoor levels (3). The mean RF

exposure from spot measurements in homes was determined to be 0.29 V m^{-1} and for outdoor 0.54 V m^{-1}. In outdoor studies the exposure levels rarely exceeded 1.0 V m^{-1}, whereas the highest exposure contributor was the downlink i.e., radiation from mobile phone base station antenna. A finding of the systematic review was that there was no distinct difference in exposure levels between European countries. However, studies done by different researchers across the Europe have used different procedures limiting the comparability between studies (3).

RF levels are exponentially higher when located closer to the mobile phone base station antenna. Therefore, proper safety measures must be applied when protecting public from the excess RF radiation. One of the main safety principles is creating sufficient distance between the public and the RF sources. This requirement may not be met in certain housing conditions. Mobile phone base stations installed on rooftops may become very close to people in nearby apartments.

RF field exposure from a mobile phone base station antenna, located at the rooftop showed that allowable maximum safety levels were exceeded when being closer than 30 m to the base station antenna (4). With the increasing distance, the RF power density is increasingly affected by the landscape topography, buildings, and trees that induce reflection and absorption. Also, RF power density depends on the numbers of channels in use by the base station antenna, the number of time intervals used and other mobile communication specific factors. The base station's maximum RF level varies across the day, which is an indicator of the mobile communications' service load. Also, RF power density distribution is greatly determined by the antenna's directional pattern. Values were measured highest in the balconies within the main radiation lobe of the antenna (4).

We have previously reported results from our measurements of RF radiation levels at certain places in Stockholm, Sweden such as at the Central Railway station (5), the Old Town (6), and in the City (7). High radiation was measured at a square, Järntorget, in the Old Town as further displayed in a recent publication displaying RF E-field distribution (8). Most of the radiation was downlink.

Of special concern is our results of measurements in a Stockholm apartment for everyday living purposes (9). Two groups of base stations are located close to the apartment. The total mean RF radiation level was 3,811 $\mu W/m^2$ (range 15.2-112,318 $\mu W/m^2$) for the measurement of the whole apartment, including balconies. Particularly high levels were measured on three balconies and in 3 of 4 bedrooms. High mean exposure levels in the bedrooms of growing children (one at 2,531 $\mu W/m^2$ and the other at 1,471 $\mu W/m^2$) with maximum peaks at 11,803 and 13,739 $\mu W/m^2$, respectively, may have deleterious effects on their physical and mental health (9,10).

The aim of this study was to further investigate radiation levels in the high exposure apartment (9) and to compare it with a low exposure apartment showing RF E-field distribution. This was a measurement study with no involvement of test persons. Thus, no ethical permission was needed.

Materials and methods

In the present study RF field levels were investigated in two apartments near mobile phone base station antennas. One of the apartments represented a high exposure living area, while the other was of low exposure area. Both of the apartments were near to mobile phone base stations but located at different city districts in Stockholm, Sweden. The locations of the base stations close to the apartment with high RF radiation exposure are shown in Figs. 1 and 2, whereas the base stations relating to the low exposure apartment are shown in Fig. 3.

The high exposure apartment's outdoor areas were positioned close to the mobile phone base station antennas, being as close as 6 m. The low exposure apartment's balcony was about 40 m away from the base station antennas, since these were installed on the neighboring building and significantly higher on the roof.

Field spatial distribution measurements were conducted in the investigated apartments. The following analyses bring forth the low and high exposure determinant factors. RF electric field was measured at each room of the apartments. Depending on the room size, the room was divided into two to ten quadrants (smaller imaginary squares). At each quadrant a spot measurement was conducted. At each spot the field was measured with slow circular movements to cover the area of about 1 m^2 at heights of 0.7-2 m. At each spot, the average and maximum electric field in Volts per meter (V m^{-1}) was recorded representing the measurement period of about 1 min.

The measurements were conducted on a working day during business hours (afternoon) in January 2019.

Field perturbation by the measurer was minimized by distancing the meter from the body-the meter was held at arm's length, with the extending probe outward. The measurements area was therefore at about 0.8-0.9 m from the investigator.

Spot distance to mobile phone base station antenna was measured by targeting the closest antenna element. Distance was measured by laser distance meter STABILA LE50, which provides precise distance measurements up to 100 m with the resolution of 0.001 m at the accuracy ±1.5 mm/m.

The measurements were conducted with a RF broadband analyzer, Narda NBM-520, with a E-field probe E0391 (Narda-Safety-Test-Solutions GmbH, Pfullingen, Germany). The Narda NBM-series meter is capable of time and spatial averaging and determining the maximum level during the period monitored. Narda EF0391 probe is intended by the manufacturer for base station measurements and has a frequency range from 100 kHz to 3 GHz.

The broadband meter Narda EF0391 covers a large range of RF transmissions, including different telecommunications protocols: frequency modulation (FM) radio broadcasting; television (TV) broadcasting; TETRA emergency services (police, rescue, etc.); global system for mobile communications (GSM) second generation mobile communications; universal mobile telecommunications systems (UMTS) third generation mobile communications, 3G; long-term evolution (LTE) fourth generation mobile communications standard, 4G; digital European cordless telecommunications (DECT) cordless telephone systems standard; Wi-Fi wireless local area network protocol, 2.45 GHz; worldwide interoperability for microwave access (WIMAX) wireless communication standard for high speed voice, data and internet.

Mobile communications' service coverage reception level was confirmed using an Android mobile phone, showing service coverage in decibel milliwatts (dBm).

Figure 1. A view from the high exposure apartment's balcony, this was one of the highest exposure areas in the apartment's main floor with the RF field topping at 5.1 V m^{-1}, base station antenna as 11.87 m away from the fence. RF, radiofrequency.

Figure 2. A view from the high exposure apartment's balcony, a set of mobile phone base station antennas are 6 m away from the fence; this was the highest radiation area, with the RF exposure topping at 6 V m^{-1}, even though the antennas sector was positioned away from the balcony. RF, radiofrequency.

In the present study, measurement data are presented both in tables and visual views. Spatial distribution of the RF levels is presented in a heat map view. Heat map is possible only by a volume of spatially scattered spot measurements. Heat map is also seen by some other authors as a way to communicate the measurements data in a comprehensible way to the public (11).

The measurement data, specifically average spot measurement values, were entered to the contour map software 3DFIELD ver. 4.5.2.0 (by Vladimir Galouchko) and spatial field distribution maps were drawn. Field distribution maps were based on the spot measurements using 1 min time averaging.

Conversion from V m^{-1}, to W/m^2, see also Table I. In most of our earlier studies we have used the EME Spy 200 from Satimo and preferred to show our results in power flux density in W/m^2 and μW/m^2 for RF radiation. In the current measurements the broadband analyzer Narda NBM-520 measures in V m^{-1} and the contour map software 3DFIELD is also constructed for measurements in V m^{-1}.

To convert from electric field strength, E, in V m^{-1} to power flux density in W/m^2, S, use the formula: $S=0.002654 \times E^2$

Figure 3. View to the low exposure apartment (rectangle). Mobile phone base station antennas are visible on the neighboring building (circled); the highest RF exposure level was on the balcony, which had the line of sight to both of the antennas. However due to the elevation difference, the balcony had highest electric field only 0.75 V m^{-1}; based on spot measurements maximum readings over 1-min period; base station antennas' sector include the investigated building. Rectangle, low exposure apartment; circle, neighboring building. RF, radiofrequency.

Statistical methods. The data was analyzed using the spreadsheet software Microsoft Excel 2016, calculating mean, median, minimum and maximum for the measured areas. Mean ($\bar{x}$), median, and minimum values were based on the time averaging function of spot measurements; maximum value was based on the maximum reading registered during the spot measurement. Differences in field level across different areas was compared in a table and illustrated in a box plot and the factors determining the attenuation/elevation of the field pointed out.

Results

The field spatial distribution measurements conducted at the apartments in Stockholm show great variation in the RF field levels.

High exposure apartment. As illustrated in Figs. 4-6, the propagation of the field from the nearby mobile phone base stations' several antennas in the high exposure apartment. Based on the RF broadband analyzer spot measurements, the maximum indoor E-field topped at 3 V m^{-1} at the bedroom on the 7th floor. The maximum outdoor exposure level of 6 V m^{-1} was encountered on the 8th floor balcony, located at the same elevation and only 6.16 m away from the base station antennas. Outdoor areas i.e. balconies have notably higher exposure, indicated by the darker color.

High exposure levels were encountered also on the 6th floor balcony with a direct line of sight to the mobile phone base station antenna at 11.87 m distance. Since the base station antenna was not aimed at the aforementioned area, hence even higher exposure conditions are avoided. The lowest exposure area is in the middle of the apartment (0.30 V m^{-1}), which is still twice as high as the mean exposure level of the low exposure apartment (0.16 V m^{-1}).

Low exposure apartment. RF field in the low exposure apartment is illustrated in Fig. 7. The field distribution in low

Table I. Conversion table from E, Electric field strength in V m^{-1} to S, power flux density in $\mu W/m^2$.

E in V m^{-1}	S in $\mu W/m^2$
3.3	28,902
3.0	23,886
2.7	19,348
2.4	15,287
2.1	11,704
1.8	8,599
1.7	7,670
1.6	6,794
1.5	5,971
1.4	5,202
1.3	4,485
1.2	3,822
1.1	3,211
1.0	2,654
0.9	2,150
0.8	1,698
0.7	1,300
0.6	955
0.5	663
0.4	425
0.3	239
0.2	106
0.1	26

exposure apartment shows much less variation in amplitude, as the field is several times lower when compared to the high exposure apartment. The maximum indoor field (0.52 V m^{-1}) was measured at the corner window, with direct line of sight to the neighboring house with mobile phone base station antennas. Maximum outdoor field of 0.75 V m^{-1} was measured at the balcony facing the same next-door building with mobile phone base station antennas. The minimum field of 0.10 V m^{-1} was registered on the apartment area closest to the center of the building, hence demonstrating the shielding effects of the indoor walls.

Spot measurements resulted both in time averaged and maximum RF field levels. Pearson's product-moment correlation coefficient shows high correlation between the two sets of values: r=0.95 in the high exposure apartment, and r=0.97 in the low exposure apartment. In average, maximum values were 58% higher than time averaged values in the high exposure apartment, and 87% higher in the low exposure apartment.

In Fig. 8 a boxplot is presented comparing the high exposure apartment to the low exposure apartment (indoor areas) based on 1-min spot measurements maximum reading.

Field increase from indoor to outdoor. Comparison was also made in both apartments between the outdoor area (staying at the balcony) and the corresponding adjacent room area, which had access to the balcony. There was about a five-fold difference in mean indoor exposure levels and about a six-fold difference in mean outdoor exposure levels between the apartments. Considering maximum readings, the outdoor exposure difference was eight-fold.

Tables II and III presents the main statistics for the low exposure and high exposure apartment, expressed in V m^{-1} (Table II) and $\mu W/m^2$ (Table III). Tables II and III statistics are based on 1-min averaged spot measurements. The maximum indoor RF field was 0.52 V m^{-1} and 3.00 V m^{-1} respectively. Maximum reading at the balcony outside the low exposure apartment was 0.75 V m^{-1} compared with 6.00 V m^{-1} outside the high exposure apartment.

Discussion

There are a limited number of studies of RF exposure levels in Sweden. A car mounted measurement system was used to map spatially some of rural, urban and city areas published in 2014 (12). Mean power density levels showed highest exposure levels at Stockholm city (6,700 $\mu W/m^2$), followed by city of Solna (3,278 $\mu W/m^2$) and then urban areas as represented by Göteborg, Helsingborg, Jönköping and Ljungby (1,500 $\mu W/m^2$) and rural areas represented by Ryssby and Ekerö (230 $\mu W/m^2$). Their study clearly indicated that higher population density results in higher RF exposure (12). They also noted that power density can vary by >50 dB (100,000 times) over a driving distance of 10 km, which supports the finding in our study-high RF exposure is not required to have a good mobile telephony service reception. Note that these measurements do not represent current RF radiation due to the rapid increase of deployment of the wireless communication (8).

A RF survey in Greece, found median urban electric field (median 1.1 V m^{-1}) to be significantly higher than rural levels (median 0.3 V m^{-1}). As the study utilized temporal measurements with 6-min averaging from 90 installed measurements stations, the data showed large diurnal variation for stations positioned close to mobile phone base stations, with median diurnal variation of 33.8% (13). These measurements are also old.

Baltrėnas *et al* (4) investigated a 10-story building neighboring a mobile phone base station antenna, where the height of the building was 30 m distanced 35 m from the base station. These conditions are similar to our study. The base station antenna was approximately on the same height as floor 6. Consequently, the highest exposed floors were 5-7, with floors where the power density at the balcony was about three times higher at 6th floor as compared to the 3rd floor. The difference was about 15-times when comparing the RF power density at the 1st floor to the 6th floor (4).

The present measurements with means of RF radiation up to 1.3 V/m (4,485 $\mu W/m^2$) at the windows in the bedrooms in the high exposure apartment imply that this exposure may almost be compared to the exposure from a mobile phone in calling mode for many hours per day. A cohort study on Swiss adolescents showed that there was an association between whole body cumulative RF radiation dose from mobile phone talk, internet use and sent SMS and symptoms like headache and exhaustibility and also a decrease in figural memory (14,15).

Many research studies have shown effects from RF radiation exposure on animals below current safety levels with opened blood brain barrier and neuronal damage (16,17), oxidative stress with increased production of reactive oxygen species (18,19),

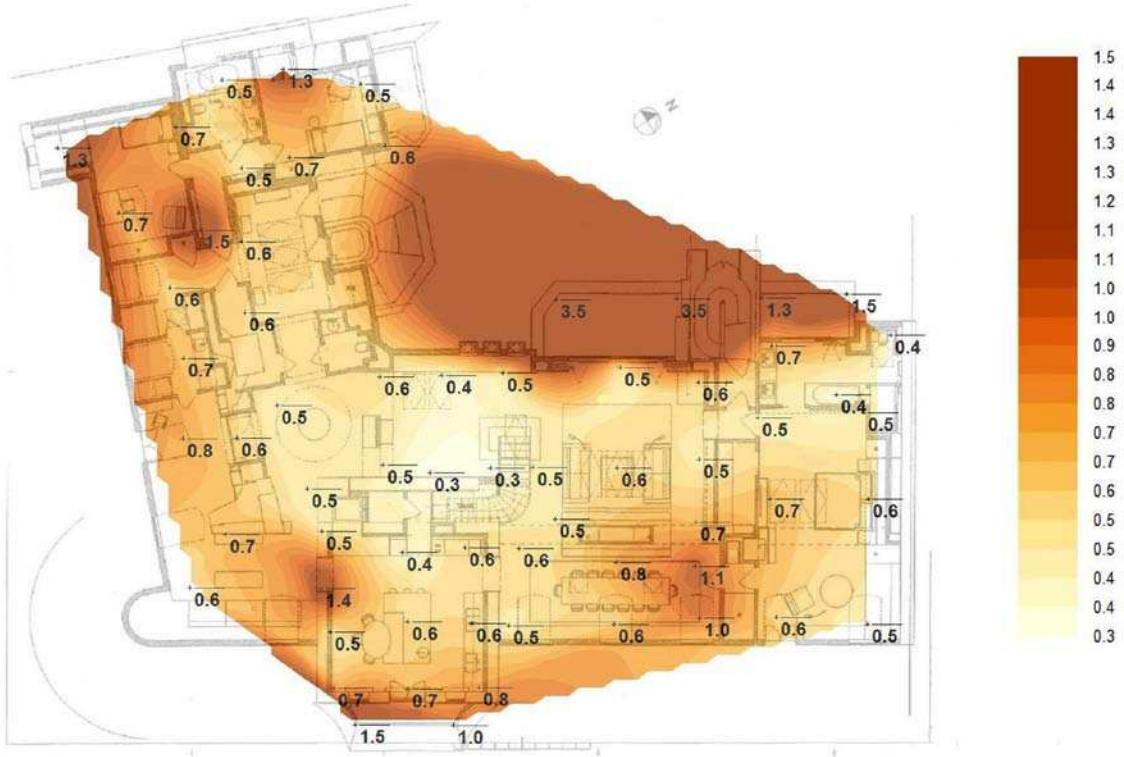

Figure 4. Spatial field distribution map of the high exposure apartment on the 6th floor; time-averaged RF electric field (V m^{-1}). RF, radiofrequency.

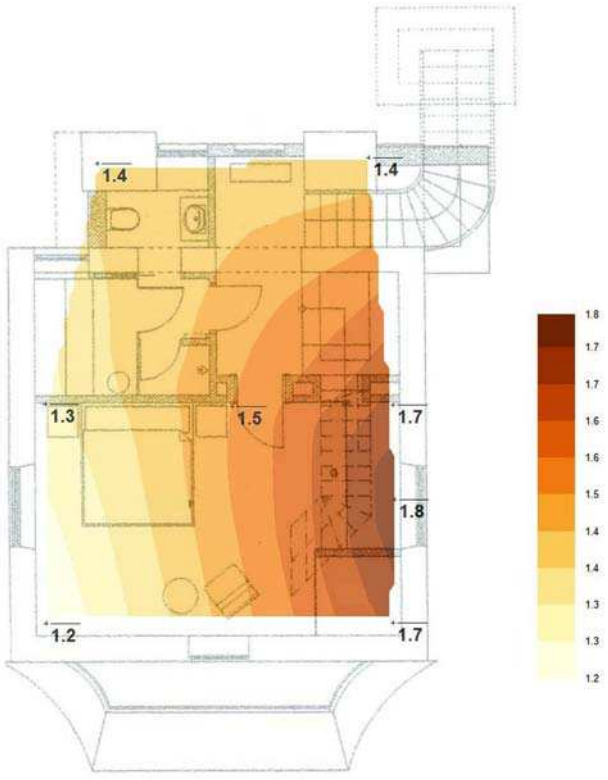

Figure 5. Spatial field distribution map of the high exposure apartment, a bedroom on the 7th floor; time-averaged RF electric field (V m^{-1}). RF, radio-frequency.

DNA-damage especially in the memory center hippocampus in the brain and increase in pro-inflammatory cytokines (20).

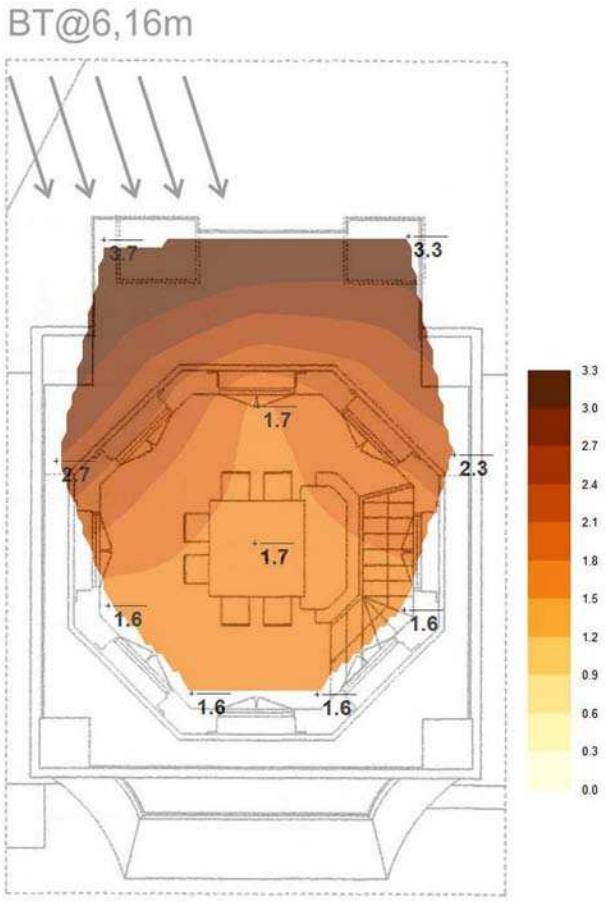

Figure 6. Spatial field distribution map of the high exposure apartment, a room on the 8th floor and on the same elevation with mobile phone base station antennas; RF radiation from base station antennas indicated with arrows; time-averaged RF electric field (V m^{-1}). RF, radiofrequency.

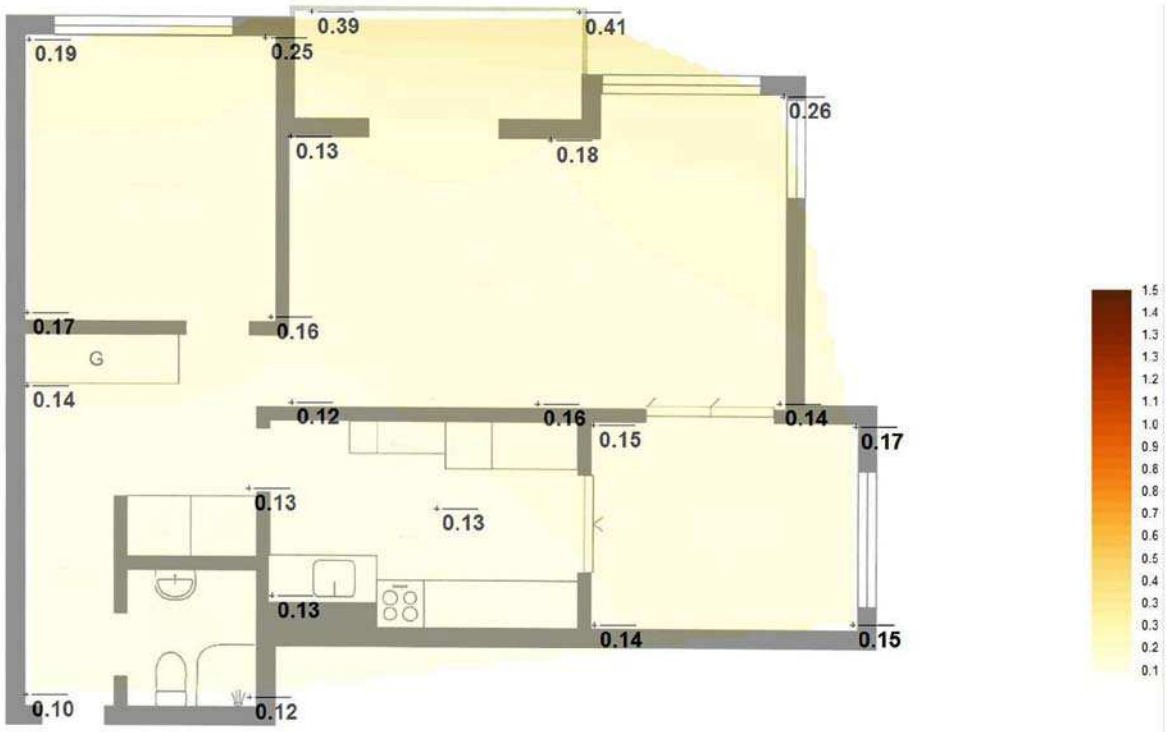

Figure 7. Spatial field distribution map of the low exposure apartment; time-averaged RF electric field (V m^{-1}). RF, radiofrequency.

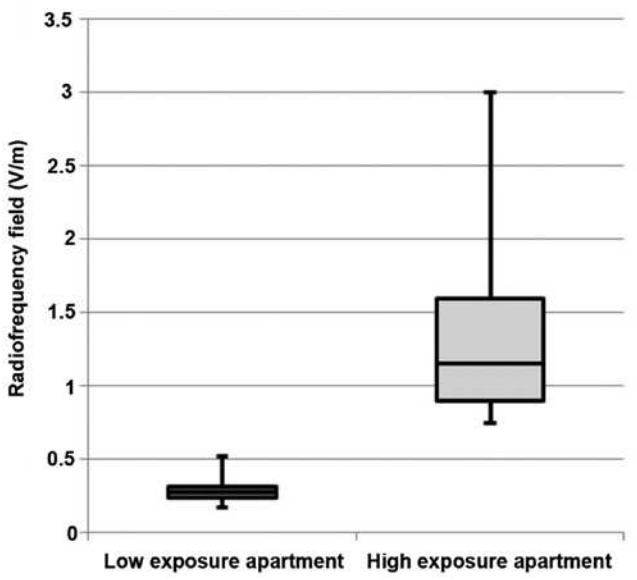

Figure 8. Box plot comparing indoor areas of the low exposure apartment to the high exposure apartment (V m^{-1}). For median RF electric field the difference is more than four times; based on spot measurements maximum readings over 1-min period. Whiskers plot depicts (from bottom up) minimum, first quartile, median, third quartile and maximum of the sample containing all the spot measurement values in the area. RF, radiofrequency.

Apart from animal studies research studies on people living near mobile phone base station show augmented indications on health risks. Adverse effects have been seen on neurotransmitters in the brain (21), on hormones like cortisol, ACTH and from the thyroid, decreased levels of testosterone in men and prolactin in young women and also increase in salivary cortisol (22,23). Other studies have shown lowered antioxidant levels and induced DNA damage in blood lymphocytes (10,24) as well as health complaints. Symptoms like sleep disturbances, headache, fatigue, dizziness, cardiovascular symptoms depression and difficulties with memory and concentration have been reported from people living near mobile phone base stations (25,26).

Human exposure has increased rapidly in recent years and will increase substantially with the introduction of the fifth generation (5G) for wireless communication (www.5gappeal.eu) (27,28) and should now be regarded as an environmental pollutant. Of special concern is that RF radiation in the frequency range 30 kHz to 300 GHz was in 2011 classified as a 'possible human carcinogen' Group 2B by the International Agency for Research on Cancer (IARC) (29,30). The carcinogenic evidence has by now strengthened and RF radiation should be reclassified as a known human carcinogen, Group 1 (27,28). Environmental RF radiation is often involuntary with little possibilities to avoid, especially since mostly nothing has been done to inform and protect people from RF radiation (31,32).

Especially the two bedrooms for the children in this apartment were exposed to high RF radiation, (mean 2,531 and 1,471 $\mu W/m^2$) (9). Children will probably be exposed for a whole lifetime in contrast to the present generation. They also seem to be more sensitive for RF radiation with more immature cells in their growing bodies (33,34).

A study from Taiwan calculated annual power density in watt-year/km^2 to each township from all 71,185 mobile phone base stations in service between 1998-2007. They found a statistically significantly increased risk for all neoplasms in children with higher-than-median exposure of RF radiation from base stations during five years prior to their neoplams (35). The Interphone study group calculated the estimated RF dose from mobile phones in five of the participating countries. The

Table II. Statistics for the low and high exposure apartment. Radiofrequency field (V m^{-1}). Mean, median and minimum values are based on the average of 1 min spot measurements (calculated based on 1 min temporal monitoring sample). The maximum is based on the same spot measurements maximum registered RF level.

Apartment	Area	Number of measured spots	Mean ($\bar{x}$) V m^{-1}	Median (V m^{-1})	Minimum (V m^{-1})	Maximum (V m^{-1})
Low exposure	Indoor area	20	0.16	0.15	0.10	0.52
High exposure	Indoor area	72	0.77	0.60	0.30	3.00
Low exposure	Outdoor area (balcony)	2	0.40	0.40	0.39	0.75
High exposure	Outdoor area (balcony)	10	2.46	2.65	1.00	6.00

RF, radiofrequency.

Table III. Statistics for the low and high exposure apartment. The radiofrequency field in power flow density in $\mu W/m^2$. Mean, median and minimum values are based on the average of 1 min spot measurements (calculated based on 1 min temporal sample monitoring). Maximum is based on the same spot measurements maximum registered RF level.

Apartment	Area	Number of measured spots	Mean ($\bar{x}$) in $\mu W/m^2$	Median ($\mu W/m^2$)	Minimum ($\mu W/m^2$)	Maximum ($\mu W/m^2$)
Low exposure	Indoor area	20	68	60	26	718
High exposure	Indoor area	72	1,573	955	239	23,886
Low exposure	Outdoor area (balcony)	2	424	424	404	1,492
High exposure	Outdoor area (balcony)	10	16,061	18,638	2,654	95,544

RF, radiofrequency.

RF radiation dose was estimated as total cumulative specific energy (TCSE) in J/kg absorbed at the tumor's estimated centre. The risk for a glioma increased with increasing TSCE 7+ years before diagnoses (36). Several studies have shown increasing risks for brain tumors, especially glioblastoma multiforme, with increasing years of mobile phone use, amount of calls and calling time (29,30,37,38).

In comparing two Stockholm apartments, several factors and exposure determinant could be pointed out. Both apartments were located in the vicinity of the mobile phone base station antenna, which allowed good mobile services reception indoor. Measured RF field levels in the low exposure apartment demonstrate that high exposure is not needed to provide good mobile phone reception.

Two mobile phone base stations placed very near, less than 20 m to an apartment may imply health risks for the inhabitants.

The low exposure apartment exposure levels were lower since the mobile phone base station was installed on top of the neighboring building, whereas on the high exposure apartment the base station was on top of the same building.

The high exposure apartment was on the top floor, with the mobile phone base station antennas situated on the roof above. The low exposure apartment was positioned seven floors lower from the roof where the base station antennas were located.

The low exposure apartment was located on the opposite side of the building from the mobile phone base station, hence the building itself provided cover (Fig. 9). Building materials, such as concrete and metal structures provide partial shielding effect against inbound radio waves.

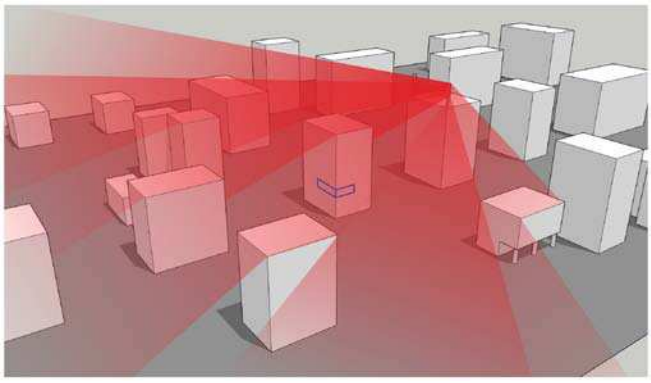

Figure 9. Mobile phone base station antenna neighboring the low exposure apartment; the buildings hinder the propagation of the RF field, especially shielding the sides facing away from the radiofrequency source; mobile phone base station antennas are sector antennas and radiate the microwaves into a direction (sector) these are aimed at. Rectangles, low exposure apartment.

In both apartments, indoor RF levels were several folds lower than outdoor levels of the corresponding room. Lower levels were detected also in the vicinity of the windows. This indicates a notable screening effect by the contemporary heat-reflecting windows.

In both apartments the lowest exposure levels were registered within the center of the building, far away from the windows, shielded by the concrete or brick walls.

Although the tin roof of the high exposure apartment shields it from the majority of the inbound radio waves, countless reflections and diffraction from the structures on the roof and balconies provide the pathway to indoor. The RF field penetrated the building's constructions, including the windows and resulted in notably high exposure levels indoor.

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Availability of data and materials

The datasets generated and/or analyzed during the present study are available from the corresponding author on reasonable request.

Authors' contributions

All authors participated in the conception, design and writing of the manuscript and have read and approved the final version. TK, LH and MA conducted the measurements. MC and TK performed the statistical analysis. MC checked the statistical methods and results. LKH wrote the medical sections, contributed to the conclusions and conversion tables.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Advisory Group recommendations on priorities for the IARC Monographs

An Advisory Group of 29 scientists from 18 countries met in March, 2019, to recommend priorities for the International Agency for Research on Cancer (IARC) Monographs programme during 2020–24. IARC periodically convenes such advisory groups to ensure that the Monographs evaluations reflect the current state of scientific evidence relevant to carcinogenicity.[1] A detailed report of the Advisory Group will be published subsequently.[2]

The Advisory Group assessed the response to a public call for nominations and considered more than 170 unique candidate agents, including the recommended priorities remaining from a similar Advisory Group meeting convened in 2014.[3] The expertise of the Advisory Group covered multiple disciplines, and the members appraised, on an individual nomination basis, the evidence according to human exposure (including any evidence of exposure in low-income and medium-income countries), cancer epidemiology, cancer bioassays in experimental animals, and carcinogen mechanisms, in line with the evaluation methodology recently refined in the Preamble to the IARC Monographs.[1] A complementary approach assessed all nominations using a chemoinformatics, text mining, and chemical similarity analysis workflow;[4] this approach helped to reveal coverage and gaps in the extent of evidence across data streams, supporting decisions on individual agents and groups of chemically related nominations. The Advisory Group deliberated on all nominated agents both by evidence stream (ie, exposure, human cancer, cancer bioassay, and carcinogen mechanisms) and by type of agent (eg, metals, fibres, chemicals, biological agents, and complex mixtures) to inform development of priority recommendations.

The Advisory Group recommended a broad range of agents with high (table 1), medium, or low (table 2) priority for evaluation. Priority was assigned on the basis of evidence

	Rationale
Agents not previously evaluated by IARC Monographs	
Haloacetic acids (and other disinfection byproducts)	Relevant human cancer, bioassay, and mechanistic evidence
Metalworking fluids	Relevant human cancer and bioassay evidence
Cannabis smoking, fertility treatment, glucocorticoids, *Salmonella typhi*, sedentary behaviour*, tetracyclines and other photosensitising drugs	Relevant human cancer and mechanistic evidence
Cupferron, gasoline oxygenated additives, gentian violet, glycidamide, malachite green and leucomalachite green, oxymetholone, pentabromodiphenyl ethers, vinclozolin	Relevant bioassay and mechanistic evidence
Breast implants, dietary salt intake*, neonatal phototherapy*, poor oral hygiene*	Relevant human cancer evidence
Aspartame	Relevant bioassay evidence
Arecoline, carbon disulphide, electronic nicotine delivery systems and nicotine*, human cytomegalovirus, parabens	Relevant mechanistic evidence
Agents previously evaluated by IARC Monographs†	
Automotive gasoline (leaded and unleaded), carbaryl, malaria	New human cancer, bioassay, and mechanistic evidence to warrant re-evaluation of the classification
Acrylamide*, acrylonitrile, some anthracyclines, coal dust, combustion of biomass, domestic talc products, firefighting exposure, metallic nickel, some pyrethroids (ie, permethrin, cypermethrin, deltamethrin)	New human cancer and mechanistic evidence to warrant re-evaluation of the classification
Aniline, acrolein, methyl eugenol and isoeugenol*, multi-walled carbon nanotubes*, non-ionising radiation (radiofrequency)*, some perfluorinated compounds (eg, perfluorooctanoic acid)	New bioassay and mechanistic evidence to warrant re-evaluation of the classification
Ostrogen:oestradiol and oestrogen-progestogens‡, hydrochlorothiazide, Merkel cell polyomavirus, perchloroethylene, very hot foods and beverages	New human cancer evidence to warrant re-evaluation of the classification
1,1,1-trichloroethane, weapons-grade alloy (tungsten, nickel, and cobalt)	New bioassay evidence to warrant re-evaluation of the classification
Acetaldehyde, bisphenol A*, cobalt and cobalt compounds, crotonaldehyde, cyclopeptide cyanotoxins, fumonisin B_1, inorganic lead compounds, isoprene, o-anisidine	New mechanistic evidence to warrant re-evaluation of the classification

Evidence of human exposure was identified for all agents. *Advised to conduct in latter half of 5-year period. †See current International Agency for Research on Cancer (IARC) list of classifications, volumes 1–123. ‡Group 1 carcinogen; new evidence of cancer in humans indicates possible causal associations for additional tumour sites (see Section 3 of Preamble to the IARC Monographs[1]).

Table 1: **Agents recommended for evaluation by the IARC Monographs with high priority**

Published **Online**
April 17, 2019
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For more on the **IARC Monographs** see http://monographs.iarc.fr/

Upcoming meetings
June 4–11, 2019, volume 124: Shift work that involves circadian disruption
Nov 5–11, 2019, volume 125: Some industrial chemicals
March 24–31, 2020, volume 126: Opium

IARC Monographs Advisory Group Members
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Declaration of interests
All advisory group members declare no competing interests

Invited Specialists
None

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Declaration of interests
All representatives declare no competing interests

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Declaration of interests
SB is sponsored by the American Beverage Association

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Declaration of interests
MCT received personal fees from ICF Incorporated, LLC, outside this work. All other secretariat declare no competing interests.

For the **Preamble to the IARC Monographs** see https://monographs.iarc.fr/wp-content/uploads/2019/01/Preamble-2019.pdf

For **IARC declarations of interests** see https://monographs.iarc.fr/wp-content/uploads/2018/07/priorities-doi.pdf

For the **IARC list of classifications, volumes 1–123** see https://monographs.iarc.fr/list-of-classifications-volumes/

	Previous evaluation status
Medium priority agents	
2,3-butanedione (diacetyl), alachlor, biphenyl, chlorinated paraffins, chlorpyrifos, c.i. direct blue 218, diphenylamine, hydrazobenzene, indole-3-carbinol, mancozeb, nanomaterials (eg, titanium dioxide or nanosilica), nitrogen dioxide, *o*-benzyl-p-chlorophenol, ozone, pendimethalin, sleep, styrene-acrylonitrile trimer, terbufos, tris(chloropropyl)phosphate	Agents not previously evaluated by the IARC Monographs
Aflatoxins†, anthracene, antimony trioxide, atrazine, bromate compounds, dimethyl hydrogen phosphite, furan, *N*-methylolacrylamide, *p*-nitrotoluene, *Schistosoma mansoni*, tris(2-chloroethyl) phosphate, tobacco smoking (including second hand)†	Agents previously evaluated by the IARC Monographs*
Low priority agents	
2-hydroxy-4-methoxybenzophenone, aluminium, androstenedione, butyl methacrylate, cinidon ethyl, dysbiotic microbiota, fonofos, furmecyclox, isoflavones, isophorone, laboratory work and occupation as a chemist, methanol, S-ethyl-*N*,*N*,-dipropylthiocarbamate, semiconductor manufacturing, Sucralose	Agents not previously evaluated by the IARC Monographs
1,1-dimethylhydrazine, benzophenone-1, carbon black, catechol, chlordecone, cumene, dichloromethane, hepatitis D virus, human papillomavirus (beta [cutaneous] and some alpha [mucosal] types), *Opisthorchis felineus*, outdoor air pollution†, pyrrolizidine alkaloids, selenium and selenium compounds	Agents previously evaluated by the IARC Monographs*

Evidence of human exposure was identified for all agents. *See current International Agency for Research on Cancer (IARC) list of classifications, volumes 1–123. †Group 1 carcinogen; new evidence of cancer in humans indicates possible causal associations for additional tumour sites (see Section 3 of Preamble to the IARC Monographs[1]).

Table 2: **Agents recommended for evaluation by the IARC Monographs with medium and low priority**

of human exposure and the extent of available evidence for evaluating carcinogenicity (ie, the availability of relevant human cancer, experimental animal bioassay, or mechanistic evidence to support a new or updated evaluation according to the Preamble to the IARC Monographs[1]). Any of the three evidence streams could alone support prioritisation of agents with no previous evaluation. For previously evaluated agents, the Advisory Group considered the basis of the previous classification, as well as the potential impact of the newly available evidence during integration across streams (see table 4 in Preamble to the IARC Monographs[1]). Agents without evidence of human exposure or evidence for evaluating carcinogenicity were not recommended for further consideration. The Advisory Group recognised that agents related to the identified priorities might also warrant evaluation. Furthermore, additional agents might merit consideration if new relevant evidence indicating an emerging carcinogenic hazard (eg, from cancer epidemiology studies, cancer bioassays, or studies on key characteristics of carcinogens) becomes available in the next 5 years. In line with the interim standard operating procedure adopted by the IARC Governing Council,[5] IARC will consider this advice when selecting agents for future Monograph evaluations according to the Preamble to the IARC Monographs.[1]

The views expressed are those of the authors and do not necessarily represent the decisions, policy, or views of their respective institutions.

IARC Monographs Priorities Group
International Agency for Research on Cancer, Lyon, France

All authors declare no competing interests.

1 International Agency for Research on Cancer. Preamble to the IARC Monographs. 2019. https://monographs.iarc.fr/wp-content/uploads/2019/01/Preamble-2019.pdf (accessed April 16, 2019).
2 International Agency for Research on Cancer. Report of the Advisory Group to Recommend Priorities for IARC Monographs during 2020–2024; 25–27 March, 2019. Lyon: Monographs on the Evaluation of Carcinogenic Risks to Humans, in press.
3 Straif K, Loomis D, Guyton K, et al. Future priorities for the IARC Monographs. *Lancet Oncol* 2014; **15:** 683–84.
4 Guha N, Guyton KZ, Loomis D, Barupal DK. Prioritizing chemicals for risk assessment using chemoinformatics: examples from the IARC Monographs on pesticides. *Environ Health Perspect* 2016; **124:** 1823–29.
5 International Agency for Research on Cancer. Sixtieth Session of the IARC Governing Council, GC/60/13. 2018. http://governance.iarc.fr/GC/GC60/En/Docs/GC60_13_CoordinationWHO.pdf (accessed April 12, 2019).

Swiss Re Institute

Swiss Re SONAR

New emerging risk insights

May 2019

What shapes the insurance industry?

Past experience and, importantly, new, changing and not-yet envisioned risks.

We created this SONAR report to inform and inspire conversations about emerging risks, so we can continue to build resilience together, in these uncertain times.

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Overview

Emerging risk themes by potential impact and timeframe

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Most affected business areas

for **Property** for **Life & Health** for **Operations**

for **Casualty** for **Financial markets**

Potential impact

High Medium Low

Foreword

In our annual SONAR report for 2019, we highlight the emerging risks we have identified and assessed through the course of the last year. The clash of new and legacy technologies, the fiscal and monetary imbalance, and the retirement skills gap are just some of the risks we feature in this study.

Many of these risks are entirely new, because they emerge from innovations not seen before, or new developments in society and/or in insurance framework conditions. The expected timeframe for the maturation of the risks into real-life events with significant loss-making potential is often short. However, we also cover "slow-burner" emerging risks, where evolution of the exposure to the point of potential for notable impact on the insurance industry will be long-running.

A classic example is climate change. Swiss Re and the insurance industry at large first flagged climate change as an emerging risk many decades ago. The risk has now "emerged" but associated (and challenging) uncertainties still remain. Such as, for example, the implications of climate change on Life & Health insurance. We devote a special feature chapter to this important topic in this report.

We hope this year's SONAR report provokes new insights for you. We look forward to engaging with you to discuss your thoughts and the spectrum of emerging risks overall.

Patrick Raaflaub
Group Chief Risk Officer


Failure of key infrastructure like power distribution can significantly impact the insurance industry.

Introduction

We define emerging risks as newly developing or changing risks which are difficult to quantify. The loss potential of these risks is currently difficult to estimate, but they may have a major business impact on the insurance industry. Against the backdrop of macro trends identified by Swiss Re, which are synthesized in overarching perspectives (see macro trends environments and overarching topics, page 7), this year's SONAR report features 15 new emerging risk themes and five emerging trend spotlights. It also includes a special feature on the implication of climate change on Life & Health insurance.

To assess and underwrite risks, the insurance industry relies on experience (ie, historical data for identified and insurable risks). However, historical data alone cannot build understanding of the future risk landscape, which is forever changing and presents new and previously unforeseen risks. Here, the insurance industry needs to demonstrate foresight and make use of sound future intelligence. Knowledge sharing through different forms of risk dialogue with all stakeholders can help insurers manage emerging risks more effectively, for industry sustainability and to improve societal resilience. Swiss Re's SONAR report, which has been published annually since 2013, provides a forward-looking perspective, to further promote and engage with such risk dialogue.

Swiss Re identifies emerging risks, first and foremost, through its proprietary SONAR tool, an internal crowdsourcing platform that collects input and feedback from underwriters, client managers, risk experts and others across Swiss Re. The emerging risk themes outlined in this report are based on early signals collected throughout the year. They neither reflect the entire emerging risk landscape of the insurance industry nor that of Swiss Re. They have been categorised according to their estimated impact and potential timeframe to materialise, and with respect to the line of business (see figure page 2) where we expect the biggest exposure will rest.

Per lines of business, the top emerging risk themes identified in this year's edition are:

for **Property:**	Retirement skills gap – accidents waiting to happen
for **Casualty:**	Teaching an old dog new tricks – digital tech meets legacy hardware
for **Life & Health:**	Don't ask, don't tell – genetic testing and adverse selection
for **Financial Markets:**	Limits to tinkering – the fiscal and monetary policy balance at risk
for **Operations:**	Getting the balance right – technology regulation affecting the insurance industry

Some of the emerging risk themes and trends presented in this and previous SONAR reports may never materialise as exposures with loss-making potential. Others likely will. The earlier the re/insurance industry starts adapting to new risks, the better prepared it will be to successfully protect its bottom line, develop new products and write profitable business.

In an appendix, emerging risks with highest impact are listed from past reports dating back to 2015, with additional information about cross-cutting themes (see Appendix A: Key emerging risks from SONAR reports 2015–2019).

The low carbon economy calls for new solutions such as high speed trains or renewable energy installations.

Macro trends

Screening of interdependent macro trends

For today's and tomorrow's risk landscape, Swiss Re has identified and assessed 23 macro trends. This is central to understanding the risk landscape of the future, to make informed decisions and to create solutions for emerging risk pools. Swiss Re assesses these trends and their interdependencies through discussions with experts, in-depth reviews and by undertaking annual surveys.

The list of 23 macro trends for 2019 remains unchanged from the previous year, when "Data as an asset" was added. We deem these trends as having the potential to be decisive elements for the re/insurance industry within the next five to ten years. The trends fall into four, interlinked "environment" categories, namely (1) societal; (2) political; (3) competitive and business, and (4) technological and natural environments.

In this section, we provide insights into the four environments and the respective macro trends. We also highlight three overarching topics that feature strong interdependencies between certain macro trends, are reflected in today's reality, and are expected to shape the future of our industry.

Societal environment

- Growing middle class in HGM
- Longevity & radical medical innovation
- Connected & collaborative society
- Mass migration & urbanisation
- The future of work & talent gaps
- Rising social inequality & unrest

Political environment

- Public sector moving risk to private sector
- Protectionism & fragmented regulation
- Increasing nationalism
- Instability of geopolitical & economic systems
- Low yield environment & risk of inflation

Technological and natural environment

- Climate change & resource scarcity
- Structural change of energy production, distribution & consumption
- Massive expansion of digital & cyber risk
- Data as an asset
- Technology application as efficiency play
- Disruptive digital technologies
- Autonomous transportation & robotics

Competitive and business environment

- Re/insurance value chain disaggregation
- Rise of collateralised reinsurance
- Strategic partnerships with non-insurance companies & institutions
- Regional champions going global
- Increasing digital customer interaction

Societal environment

The economic relevance of the middle class remains all pervasive but increasingly, the onus is shifting to the emerging economies, most notably Asia Pacific. A large number of households in emerging markets have escaped poverty over the past years, and growing household wealth has led to more consumption. Shift and overall growth of insurance markets are to be expected, but they cannot be taken for granted (see page 38 "Risky bets?").

Escape from poverty has seen mass migration from rural areas to cities. The UN Department of Economic and Social Affairs estimates that by 2030, the number of people living in urban settings across the world will reach 5 billion, up from around 2.5 billion today. Over 90% of the increase will occur in high growth markets (HGM), and most in China and India. Migration and urbanisation concentrate risk. For one, large cities are more prone to health hazards, an important consideration for Life and Health (L&H) insurers. And also, with an associated accumulation of economic assets in urban areas, there is more potential for large financial losses in the event of a major natural disaster, an opportunity for property lines of business. Serving as powerhouses to national economies, cities also invite resentment from the rural periphy. A number of recent democratic elections and votes, e.g. Brexit, have shown signs for a growing divide between the populations of metropolitan centres and of rural peripheries (see page 24 "Conflicting interests").

In the mature economies, the post-World War II baby-boomers have mostly reached retirement years (with regard to the skills gap opening and subsequent risk, see page 17 "Retirement skills gap") with sufficient savings and asset gains accumulated from the prosperity of the 20th century. The next generation is still strong in numbers, but is accumulating less wealth and experiencing more pressure from globalisation and automation. The pension systems in many mature markets is becoming increasingly unsustainable for future retirees. This, and a shrinking middle class in some part of the developed world, may deepen social inequality. As more people feel left behind, a pool of dissatisfaction will likely build. In times of social media connectivity and of traditional political parties disrupted by political entrepreneurship, channeled resentment has become more decisive, and social unrest is on the rise. The "gilets jaunes" (yellow vests) movement in France is a case in point.

Currently, this is mostly a mature markets' problem, but it may also provide signals for the fast changing demography of developing countries. For instance, China's is grappling with unintended effects of its population policies. Small family units after decades of one-child policies will struggle to finance the retirement years of the country's already rapidly aging population. Restrictions on mobility are further exacerbating inequality.

Political environment

Globalisation momentum has slowed over the past decade. Relative to world GDP, cross-border investment, trade, bank loans and supply chains have all been shrinking or stagnating. This started well before US Donald Trump's Presidency. His anti-globalisation campaign, which has been a political success for his administration, has since been copied around the world. Multilateralism is on the back foot and global governance of commerce embodied by the World Trade Organization (WTO) is at risk of collapse (see page 12 "Globalisation fragmented" and page 23 "Beggar thy neighbor").

Rules on privacy, data and espionage are splintering. Even accounting and anti-trust regulations are fragmenting. Tax systems are being bent to patriotic ends, including a strong trend of taxing corporates locally based on local revenues. In this regard, the US is pressuring firms to repatriate capital, while Europe is targeting Silicon Valley. Moreover, the US makes frequent use of the power it derives from running the world's dollar-payments system to curb the activities of foreign companies. The old powers cling to what they still command, while broader economic, political and cultural power continues to shift to Asia.

While the low yield environment persists, room for manoeuvre by central banks has become increasingly limited. Long-term accommodative monetary policy combined with weaker global coordination has led to growing inflation risk. Central banks may lack the tools and independence to address inflation, resulting in a new super-cycle dominated by fiscal policy (see page 22 "Limits to tinkering").

Aging populations in richer countries put public finances, including pension and health systems, under strain. As a consequence, public services and assets are being outsourced to the private sector. One example is Haven, a joint venture by Amazon, Berkshire and JP Morgan to improve access to primary care in the US, simplify insurance and make transcription drugs more affordable for employees.[1]

[1] *Amazon, Berkshire, JPMorgan healthcare company to be called Haven,* Reuters, 6 March 2019,

Technological and natural environment

The world is undergoing a shift in terms of who owns data, especially in regards to individuals (see page 27 "The surveillance economy"). As social media and other companies which have "control" over data begin shifting their approach – either pre-actively or in response to regulatory pressure – data may increasingly come back into the hands of the individual. Data has been and will continue to be a key differentiator in business, including for re/insurers (see page 41 "Getting the balance right").

Technologies like Big Data and cloud computing can greatly increase the efficiency of capturing, storing and computing data. The Fourth Industrial Revolution is progressing full steam ahead including, among others, process automation, Internet of Things (IoT) devices and digital analytics capable of rapid analysis of massive amounts of un-/structured data (regarding speed of data transmission through 5G also see page 29 "Off the leash"). The combination of more data and better analytics can lead to better insurability of previously difficult-to-price products, and also to new capabilities in fraud detection. All this, however, raises privacy issues and trust concerns, and thus regulatory involvement. For insurers, specifically, these developments also raise the spectre of adverse selection.

The rise of Artificial Intelligence (AI) will have a major impact on knowledge and human capital-intensive businesses (for the realm of healthcare see page 31 "Wiggle room"), transforming existing employees into an "augmented" workforce. This could lead to an increase in unemployment, initially among lesser-skilled professions, and trigger social unrest.

The frequency and severity of risks resulting from cyber-attacks are expected to grow significantly over the next years. Recent examples have revealed how unprepared companies and government agencies are for such attacks. The need for cyber resilience has become a main focus of attention among corporate clients and insurance companies, triggering insurance demand. Cyber risk presents one of the largest opportunities for the re/insurance industry, while simultaneously also posing one of the biggest challenges. Keeping up with the changes in the cyber and technology space, and developing solutions to cover the resulting and ever-evolving risks is no mean feat.

Competitive and business environment

Primary insurers continue in their struggle to reshape their traditional systems and processes to serve the consumer base with next-generation insurance solutions (also see page 40 “Financial services and the digital revolution”). Reinsurers and intermediaries seek access to distribution channels and risks outside the traditional value chain. As international re/insurers face more restrictions with respect to data privacy and operational issues with legacy systems, innovative business models and digital eco-systems are likely to emerge, starting off in HGMs. These will significantly impact the global re/insurance value chain.

Digital ecosystems present a great opportunity for insurers to interact with potential customers. China is one of the fiercest Insurtech marketplaces in the world, and e-commerce is transforming the way the population interacts with industry. With the emergence of disruptive technology finding application in many industries, strategic partnerships seem to be a key component of the development of innovative insurance solutions. Investments in digital platforms by tech giants and Insurtech start-ups have been on a continual upward path since 2014.

With respect to the growing involvement of the emerging economies in global development, on the re/insurance side, many of the emerging markets remain dominated by national players. Some of these firms, for example from China and Korea, are developing global ambitions, and this will impact the dynamics of international re/insurance business in the years to come.

Low-carbon economy

The low-carbon economy model is rapidly gaining traction, as societies and enterprises explore and develop new solutions. The last decade has seen exponential growth of solar and wind power installations. This was mainly driven by large falls in production costs, initially due to innovation and then economies of scale. Similarly, there has been significant progress in battery storage technology, critical for the anticipated future dominance of the electric mobility. The official policy stance has become more supportive of the low-carbon ideal. For instance in motor, advanced countries' regulatory measures on combustion engine are becoming ever more demanding. And in China, the leadership has decided to clean up its cities. In 2018, the Ministry of Ecology and Environment was established with a mandate to invigorate enforcement and conduct local inspections. Policy innovation in transport means that today, China operates 99% of the world's electric buses.[2] The current policy priority is autonomous electric cars.

From an insurance perspective, the low-carbon economy is characterised by a range of often competing technologies and business models. The lack of performance history on these raises many uncertainties. A number of hypes in recent times turned out to be dead-ends: biofuels for cars and carbon capture for coal-fired power generation, to name a couple of examples. That's not say insurers should not remain alert of new developments. While low-carbon energy generation models contains inherently less property and casualty risk than high temperature/pressure generation, they also entail new risk factors and insurability potential, such as performance risk due to weather dependency. Insurers have already begun to address exposures like this with innovative parametric-based products.

Less optimistic, technological progress and shifts in regulation and in social norms are giving rise to the risk of stranded assets. These are unanticipated asset write-downs, devaluations or conversion to liabilities in high-carbon sectors such as oil and gas (including tar sands), utilities and basic materials. Nevertheless, despite such challenges, opportunities abound. The low-carbon economy is growing broadly and tangibly. Re/insurance can facilitate the introduction of low-carbon technologies by assessing and underwriting new risks as they emerge and by partnering with innovators across different industries, design new risk transfer solutions for the exposures of the future.

Globalisation, fragmented

After eight decades of expansion, trade globalisation is facing a major test. After World War II, the Western powers established an open and stable system of multinational trade – centred on the General Agreement on Tariffs and Trade (GATT), and the Soviets created an alternative trade network. After the end of the Cold War, the Western system prevailed, leading to a "golden age" of globalisation (1990–2007) powered by trade expansion, the rise of emerging markets and differentiation of value chains according to comparative advantage. This was facilitated by a period of (relative) US-led international political stability and embedded in global formats such as the G7, G20, and WTO.

The global financial crisis of 2008–09 brought globalisation momentum to a halt. In the decade before the financial crisis, world goods and services trade volumes doubled. In the decade after, volumes have grown by less than half the pre-crisis rate.[3] Cross-border investment, trade, bank loans and supply chains have all been shrinking or stagnating relative to world GDP. The Trump administration enthusiastically engages in trade wars. Geopolitical rivalry is gripping the tech

[2] *Electric Buses in Cities,* Bloomberg New Energy Finance, March 2018
[3] The International Monetary Fund's (IMF) *World Economic Outlook Database*

industry, which accounts for about 20% of world stock markets. Rules on privacy, data and espionage are splintering. Tax systems are being bent to patriotic ends. The US and EU have new regimes for vetting foreign investment, while China has only started to live up to its commitment to giving foreign firms a level-playing field.

All the while, the globalisation of world commerce continues, but patterns and governance have changed markedly.[4] Global institutions have lost influence relative to assertive nation states, which in turn are catering to the louder and more idiosyncratic demands of their citizens. For multinationals, re/insurers included, this represents a more demanding landscape with less predictable stakeholders.

Demography and health

The world's population is growing. Average life expectancy at birth is expected to rise from 71 years in 2010–2015 to 77 years in 2045–2050.[5] Demographic cohorts differ in age structures, wealth and health. While longer lives are a positive social outcome, they also pose a number of challenges.

First, the aging of societies, which we see in mature markets but also in emerging economies. China, which is now the world's second largest economy, is aging rapidly due to the one-child policy in place from 1979 to 2015. The average age of the Chinese population is increasing much faster than in other markets, the effects being not only a shrinking productive workforce, but also significant shifts in the old age dependency ratio. A resource gap widens quickly for the growing prevalence of age-correlated health issues, chronic diseases, dementia and other problems that require medical attention, caretakers and financial means. The accumulation of resource strain on health systems are common to many economies, but are dealt with quite differently. Immigration can be an important mitigating factor for healthcare labour shortages, but only in societies that are open to foreigners.

Another way to tackle increasing health costs is through technological innovation – from care robots to wearables monitoring health conditions. Face-to-face patient/physician interaction is being increasingly supported (or replaced) by telecommunications and digital means. Consequently, L&H insurance products are increasingly digitalised, from consulting through to claims handling. The number of digitally sold policies is growing exponentially, and the trend to more digital interaction reflects both customer demand and efficiency gains. Advances in diagnosis and testing hold equal promise and risks, including over-diagnosis and subsequent unwarranted treatments, as well as adverse selection risk from genetic tests cheaply available to consumers.

While medical innovation and the increasing awareness around lifestyle impacts has increased longevity and life quality for many, we see significant counter trends. In many emerging markets, with modernisation and changes in diets and physical activity levels, there is increased obesity and diabetes. But even mature markets show unexpected declines in mortality improvement. The longer trends and underlying reasons warrant deeper analysis.[6] In the US opioid crisis, excessive subscriptions for addictive painkillers has fuelled a fire of pre-existing social, demographic and health problems that a struggling health system has failed to cope with. Interconnections between demographics and health will remain an important field to follow.

4 See risk theme "Beggar thy neighbour? - Global trade reordered"
5 *World Population Prospects: The 2017 Revision, Key Findings and Advance Tables,* United Nations Department of Economic and Social Affairs, June 2017
6 *sigma 6/2018 – Mortality improvement: understanding the past and framing the future,* Swiss Re Institute

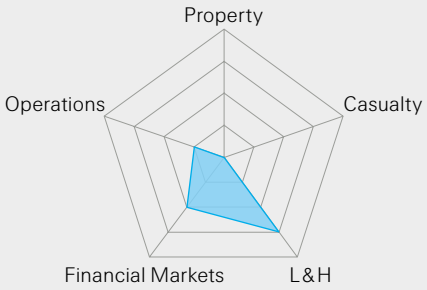
15 Emerging risk themes and 5 Trend spotlights

Societal environment

Impact: medium

Most affected business areas L&H, FM

Time frame (years): 0–3



Vaccination – a shot worth more than politics and profitability

One of the blessings of modern medicine is vaccination. Immunisation is a cost-effective way to keep many transmissible viral and bacterial diseases in check and, according to estimates of the World Health Organization (WHO), prevents 2–3 million deaths every year. But this achievement is under threat, due to questions around the economic viability of vaccine production and distribution and also what we see as weaponisation of vaccination in areas of conflict and with growing impacts of anti-vaccination campaigns. Vaccine shortages and refusal increase the likelihood of infection spread, which can potentially balloon to pandemic proportions, most notably for highly infectious diseases where herd immunity counts. The implications of a pandemic are most severe for life and health insurers. There are indirect implications for broader financial markets also.

For profitability reasons, the large pharmaceutical companies in the West are content to leave vaccine production to companies in new markets such as China. Globally, more vaccines are needed to fight diseases like hepatitis B or influenza and the supply chain has become increasingly based in the East. This results in new dependencies and the possibility of vaccine shortages in some locations, especially during periods of heightened political tension and national economic rivalry. Another danger we see is a lag in the development of new vaccines where the profit potential does not make for an attractive business case for pharmaceutical companies.

The WHO lists "vaccine hesitancy" among the "Ten threats to global health in 2019."[7] This "reluctance or refusal to vaccinate despite the availability of vaccines" is attributable, among others, to complacency, inconvenience in accessing vaccines and lack of confidence, the report says. Whatever the reasons, such attitudes risk the resurgence of otherwise avoidable dangerous diseases. A very recent case in point, in the US, there were 465 confirmed cases of measles across 19 states in the first quarter of 2019, the second largest outbreak since 2000 when measles were said to have been eliminated.[8]

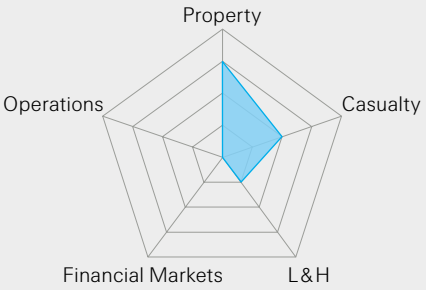
The anti-vaccination movement derives legitimacy from cultural and/or philosophical rejection of vaccines as a force for good. However, in our view the politicisation of the anti-vaccination movement as a means of expressing dissent against domestic authorities and international organisations only heightens the risk of pandemics.

Potential impacts:

- Vaccines shortages and refusals increases the likelihood that infections will spread, increasing morbidity and mortality.
- Life insurers are exposed to higher claims in the case of a severe pandemic.
- A large pandemic can have significant impact on the health system and also mortality, with potential for large scale reduction of regional populations.
- A drop in productivity, due to many factors (eg, closed schools). Trade, travel and tourism will be subdued and economic output will be reduced.[9] This affects financial markets and is therefore directly relevant for re/insurers' balance sheets on both the asset and liability side.

7 *Ten threats to global health in 2019,* World Health Organisation, 2019
8 *Measles Cases and Outbreaks,* Center for Disease Control and Prevention
9 *Epidemics and Economics,* Finance and Development, Vol 55, No. 2, IMF, June 2018

Impact:	medium
Most affected business areas	P&C
Time frame (years):	>3



Retirement skills gap – accidents waiting to happen

Almost one in five workers in the oil and gas industry is a baby boomer. This generation will retire in the next few years, which may lead to an expected shortage of 10 000 petro-technical professionals globally by 2025.[10] Combined with current layoffs and fewer students enrolling in university courses as petro-technical professionals, this situation is threatening the safe operation of hazardous oil and gas installations. Such circumstances are troubling especially given that ageing facilities often run at peak capacity.

The same is true of healthcare. By 2030, there's likely to be a shortage of 15 million healthcare professionals globally.[11] This is worrying since the demand for healthcare services in developed markets will rise as the baby boomers retire. These two sector-specific scenarios can be extrapolated to developed economies as a whole. In all sectors, from manufacturing to engineering to financial services, many people will be retiring in the coming years, taking with them critical know-how and experience that technology can only in part replace.

Research shows that professional experience is a very important element for safety, and that an increase in workplace-related accidents in manufacturing industries can come as a result of outsourcing.[12] The safety of individuals at the workplace and general operational safety are closely related, and workplace safety records will be a sentinel for things to come.

What does this mean for insurance? An uptick in P&C, professional indemnity, medical malpractice as well as healthcare-related claims might be among the consequences.

Potential impacts:

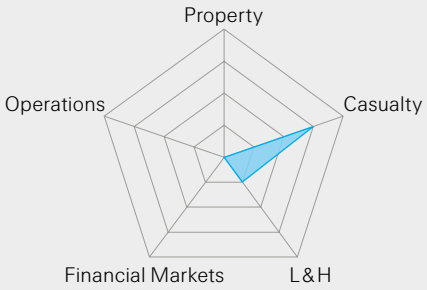
- A growing skills gap might cause more frequent occurrence of major accidents in hazardous industries and incidents in production and construction industry.
- Service industries could be exposed to more professional indemnity claims.
- A shortage of healthcare professionals might increases the risk of the healthcare industry to medical malpractice claims.
- A skills shortage in the healthcare sector will likely lead to increase costs in the provision of healthcare services, something that advances in technology will only in part offset.

[10] *Retirement Wave and Digital Reinvention Prompt Urgent Talent Reassessment,* www.rigzone.com, 29 December 2017, and *The Talent Well Has Run Dry,* Accenture, 2017

[11] *Global Health Workforce Labor Market Projections for 2030,* World Bank Group, August 2016

[12] Sanna Nenonen, *Fatal workplace accidents in outsourced operations in the manufacturing industry,* Safety Science, Vol.49, Issue 10, December 2011

Impact:	low
Most affected business areas	Casualty
Time frame (years):	>3



Concussion injuries in sport – head on to more widespread claims

Each year more than 42 million individuals are diagnosed with concussion, often resulting from sports or recreational activity.[13] Millions more suffer sub-concussive blows, where the intensity of impact is not sufficient to result in a clinical diagnosis of concussion. Repetitive head trauma has been linked to many conditions that have latency periods of years or even decades, such as Parkinson's disease, Amyotrophic Lateral Sclerosis (ALS), dementia and Chronic Traumatic Encephalopathy (CTE), a progressive neurodegenerative disease which currently can only be diagnosed post death. Researchers are getting closer to being able to identify CTE in the living and this is expected to significantly increase the number of diagnoses.[14] There is also increasing evidence that even a single concussion experience may result in an increased risk of Parkinson's disease and dementia, or have lasting impact on cognitive function.[15]

Head trauma is not only a concern for professional athletes. CTE has been discovered in individuals who only played youth or college sports, including athletes and (American) football, soccer, rugby, basketball and baseball players.

The discovery of the long-term risks of head trauma has sparked high-profile litigations in the US, filed by athletes against professional sports organisations such as the National Football League (NFL). That lawsuit was settled for an uncapped amount, estimated to be well over USD 1 billion. Hundreds of other suits remain pending, against collegiate sports governing bodies, helmet manufacturers, youth sports organisations, and dozens of individual colleges and universities.

The number of people potentially affected, the increased attention on head trauma, and the size of the NFL settlement are just a few of the factors that make this a true emerging risk.

Potential impacts:

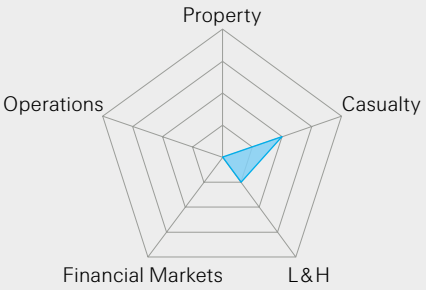
- Broader awareness of the issue will increase concussion diagnoses, as more people seek medical attention. This will trigger liability insurance, but also impact on life and health books.
- Additional litigations are to be expected due to the size of the NFL settlement and rising awareness of long-term risks increases.
- Enactment of youth concussion laws and revised rules/guidelines by sports organisations may increase requirements on standards of care and potential liabilities.

[13] Kathryn L. Van Pelt et al., *A cohort study to identify and evaluate concussion risk factors across multiple injury settings: findings from the CARE Consortium,* Injury Epidemiology, Vol. 6, No.1, 2019

[14] Nadia Kuonang, *A study of NFL players' brains might help diagnose CTE in the living,* CNN, 10 April 2019, and *Study suggests path to detecting CTE in the living,* Associated Press, 11 April 2019

[15] Barlow et al., *Investigation of the changes in oscillatory power during task switching after mild traumatic brain injury,* European Journal of Neuroscience, Vol. 48, No. 12, 2018; and Nina Bai, *With Dangers of Everyday Concussions Revealed, Scientists Race to Find Solutions,* UCSF Research, October 17, 2018

Impact:	low
Most affected business areas	Casualty
Time frame (years):	>3



A celebrity body – the hazards of aesthetic surgery tourism

More people are travelling abroad for medical procedures such as plastic surgery and dentistry. The reasons include lower costs, no coverage for the treatment under home country healthcare plans, lack of access to required treatment in the home country, better quality of treatment and reduced waiting time. Travelling abroad for plastic surgery beauty enhancement treatments not covered by healthcare insurance policies is especially popular.

Experts have put the dramatic growth of plastic surgery down to a rise in the number of celebrities promoting cosmetic procedures, cultivating a consumer-base conditioned to desire new norms in body shapes and looks. Researchers at the Boston Medical Center have found the kinds of facial surgery people are requesting now include nasal and facial symmetry, rhinoplasties, hair transplants and eyelid surgical procedures.[16]

Cosmetic surgery poses pressing problems. A report by the Nuffield Council on Bioethics outline the risks of this lucrative market in the UK.[17] The absence of a coherent regulatory framework means that often non-specialist or underqualified physicians perform aesthetic procedures without adequate infrastructure, sometimes with devastating consequences. When undertaking treatment abroad, the patient and physician may not speak the same language fluently. this increases the risk of misunderstandings, wrong treatments and undesired effects.

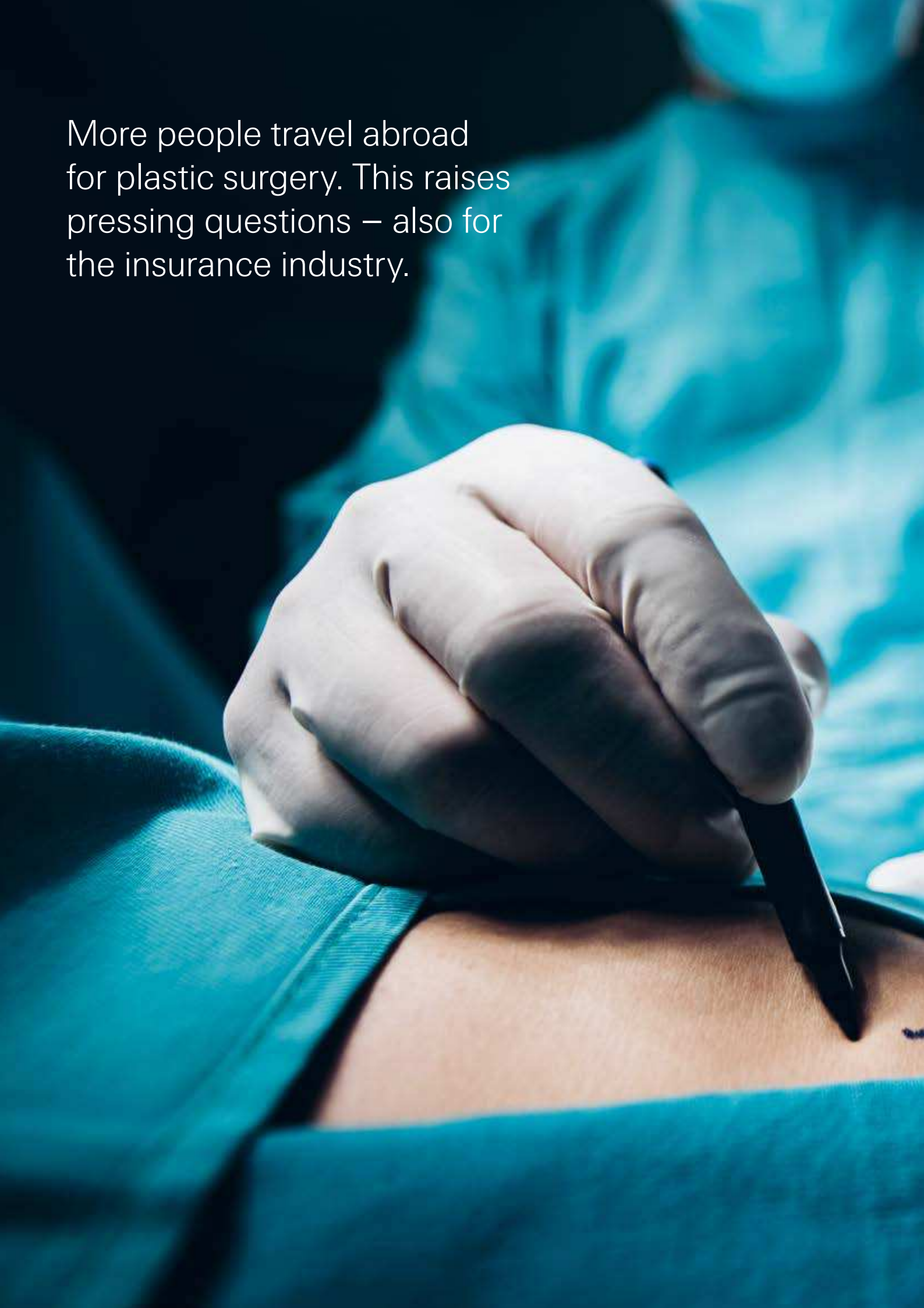
It's not always clear if health insurance in the home country covers the additional costs originating from planned procedures abroad. This relates to rules on medical malpractice and its insurance, for example, regarding whether insurers have to cover costs from unplanned procedures abroad and if these costs can be recovered. Moreover, given difference in regulatory practices by jurisdiction, it can be difficult to assign the weight of responsibility. Given the high growth of cosmetic plastic surgery both at home and abroad, it's likely that in the future answers to these questions will more often come out of the courtroom.

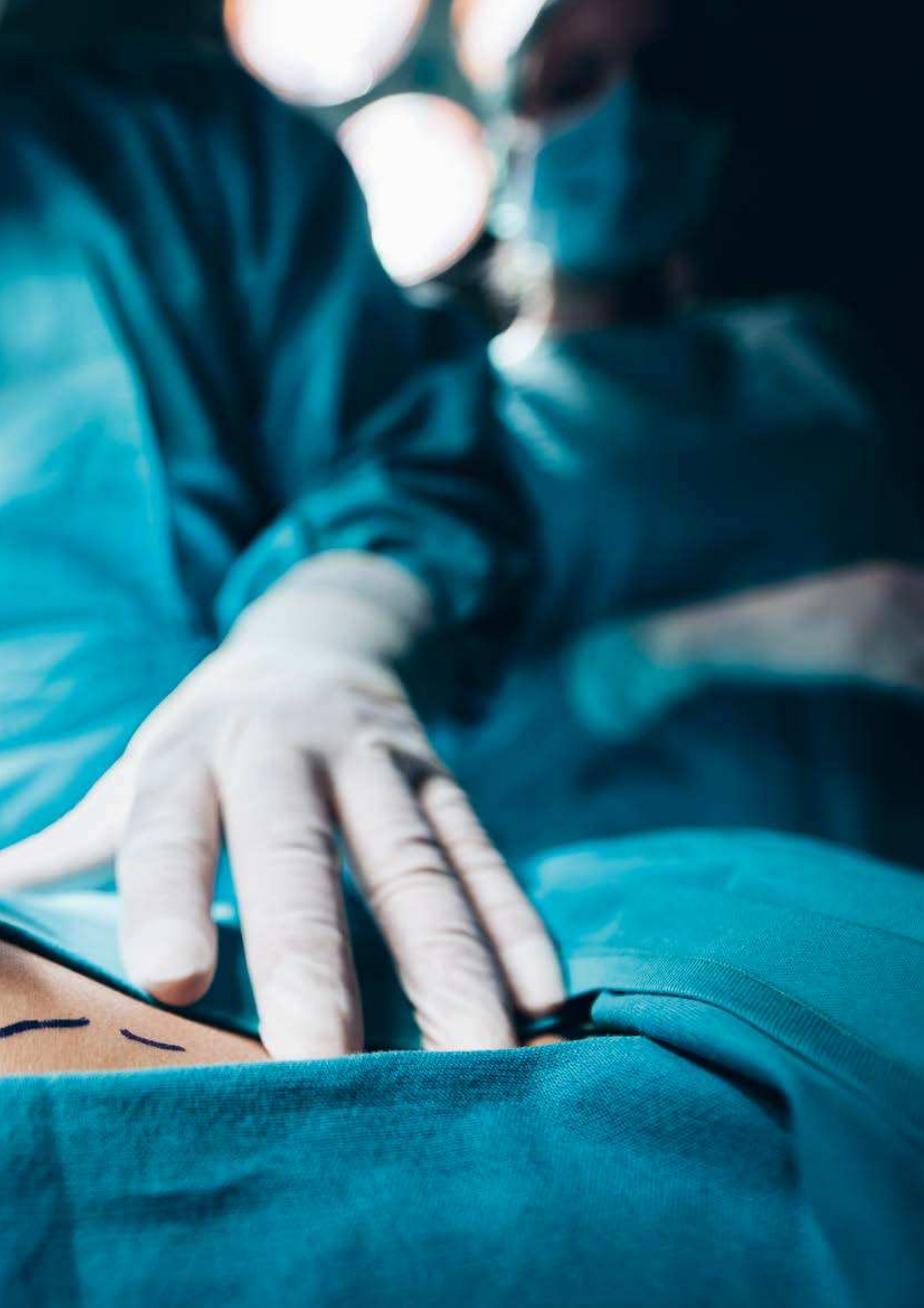
Potential impacts:

- An unclear situation about medical malpractice claims in this area could trigger defense costs and later even the claims themselves.
- Manufacturers of the products and equipment used in cosmetic procedures are competing in a lucrative market, with different approval and oversight processes in different jurisdictions. Product liability claims for related implants could become an issue.
- Medical standards – also for procedures and related care services – may be of poor quality in some countries. If medical travelers do not research their chosen medical centre, they may find themselves with unwanted and potentially dangerous results.
- Antibiotic resistance is a global problem. Resistant bacteria may be picked up in countries with a high prevalence of superbugs in hospitals.
- For operations abroad, flying after surgery can increase the risk of blood clotting.

[16] Susruthi Rajanala, Mayra B.C. Maymone, Neelam A. Vashi, *Selfies — Living in the Era of Filtered Photographs*, JAMA Facial Plastic Surgery, Vol. 20, No. 6, 2018

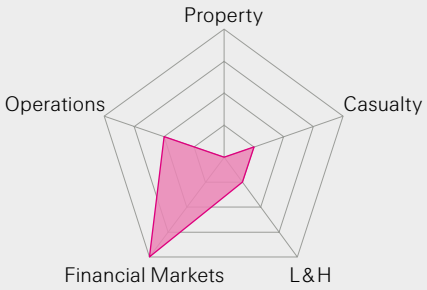
[17] *Cosmetic procedures: ethical issues*, Nuffield Council on Bioethics, 2017


More people travel abroad
for plastic surgery. This raises
pressing questions – also for
the insurance industry.



Political environment

Impact:	high
Most affected business areas	FM
Time frame (years):	>3



Limits to tinkering – the fiscal and monetary policy balance at risk

Since the global financial crisis, the world's major central banks have engaged in extraordinary policy measures resulting in massive expansion of their balance sheets. With central banks running out of tools to stimulate the economy, the growing consensus is that another economic downturn will need a fiscal response. The key question is what form of fiscal activism we might see. One idea – similar to the case of Japan – is to combine increased fiscal spending with more ultra-accommodative monetary policy, such as quantitative easing (QE) or yield curve control.

There are still more radical proposals. One example is outright "helicopter money", where central banks use their balance sheets to absorb the increase in government borrowing. This would be different from QE in that central banks would pledge to keep government bonds on their balance sheets indefinitely. Another idea that has gained traction recently is Modern Monetary Theory (MMT), which some argue is neither modern, monetary nor a theory. Under MMT, fiscal rather than monetary policy acts as the main stabilisation tool for the economy, while low interest rates are used to keep public finances sustainable.

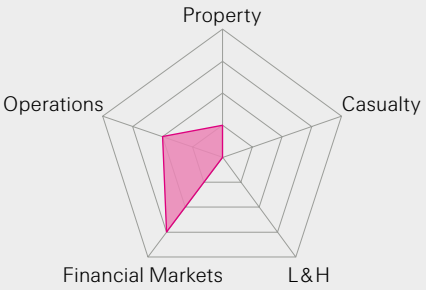
While officially central banks retain their independence, the closer coordination with government raises questions about how true that is. We think outright regime shift towards alternative monetary/fiscal frameworks such as MMT is unlikely in the near-term. That said, we do expect fiscal policy to play a significantly bigger role, this at a time when global leverage is already close to historic highs, both to stimulate economic growth and to reduce income and wealth inequality. The degree and design of fiscal dominance will be important to monitor as it could have significant consequences for the economy and financial markets, including the insurance sector.

In a fairly benign scenario of closer policy coordination amid low inflation, a prolonged period of low interest rates would be the most likely outcome. By contrast, an outright regime change in the fiscal and monetary policy framework, such as MMT, could notably increase uncertainty around the inflation outlook and financial market stability. In the long-run, this could result in much higher inflation and interest rates, with broad repercussions on financial markets.

Potential impacts:

- The economy and financial markets, including the re/insurance industry, could benefit if changes to fiscal and monetary policy stimulate growth and financial stability.
- On the flipside, a policy shift could lead to a notable rise in uncertainty, causing higher financial market volatility and significant declines in asset valuations.
- If central banks (are forced to) keep interest rates low to accommodate increased fiscal spending, the insurance industry would suffer, in particular life insurers.
- Meanwhile, an unexpected and sustained increase in inflation – also a potential consequence of a regime change – would be harmful for the re/insurance industry, in particular for inflation-sensitive liability lines of business. At the same time, however, life insurers would benefit from a potential increase in interest rates as their liabilities typically have a longer duration than their assets

Impact:	medium
Most affected business areas	FM
Time frame (years):	0–3



Beggar thy neighbour? Global trade reordered

As with other multinational businesses, insurers have become used to basic global rules that made global commerce more predictable. But the rules-based trading system is now in crisis. The very existence of the World Trade Organization (WTO) – which governs a thriving global commerce – is being questioned, most notably by its architects. For international businesses, this fundamental challenge is aggravated by regulatory fragmentation and shifts to revenue-based local taxation.

The significance for re/insurers is that regulatory restrictions and capital requirements may threaten business models based on global risk diversification and efficient capital management[18]. The US – architect of the current global trade order – no longer regards trade as the "tide that lifts all boats", but rather as a zero-sum game. The Trump administration questions the WTO's dispute settlement system and favours new trade agreements, based on bilateral relationships. There is now real danger of open trade conflict between the world's major trading blocs: the US, the European Union and China. The named three are all shaping their own trade networks. Further US disengagement from global geopolitical affairs and multilateral institutions for example, as well as the pursuit of trade policy through aggressive use of tariffs, withdrawal from existing agreements, and bilateral renegotiations, will significantly reduce the odds of bilateral deals.[19]

The Chinese approach differs, evolving around sweeping investments of billions of dollars into infrastructure projects in 60 countries under the umbrella of the Belt and Road Initiative. The goal is to create a network of trade routes connecting east and west. It has attracted many cheerleaders but also drawn harsh criticism. The European Commission now considers China both a strategic partner and an economic competitor. It in turn is showing a tendency to join the state mercantilist bandwagon by backing national champions, restricting foreign direct investment in sensitive sectors, and seeking trade deals in its favour.

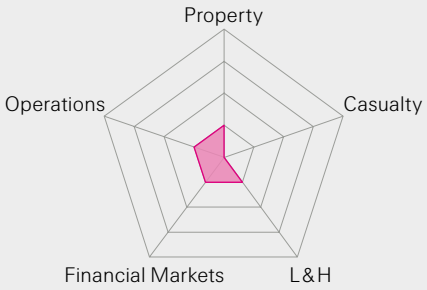
As a result, multilateral trade liberalisation – a significant driver of globalisation and prosperity during the past two decades – has come to a halt. Trade architecture is no longer dominated by a Western alliance, but by competing architects building their own systems and channels. These competing trading blocs negotiate and revise agreements, putting greater emphasis on immediate domestic priorities as well as special interests. Multinational re/insurers will discover new opportunities but must also navigate previously unseen risks.

Potential impacts:

- Multinationals, including re/insurers and banks, risk getting caught between competing spheres of power.
- Regulatory restrictions and capital requirements may reduce market access and threaten business models based on global risk diversification and efficient capital management.
- Local political events may have outsized consequences, including on financial markets, if amplified by geopolitical dynamics (eg, elections in the Maldives becoming a proxy contest between India and China)
- Higher infrastructure project risk resulting from lower standards and less scrutiny due to the absence of respected multilateral agencies
- (Over)-sized projects – partly driven by short-term political interests – increase the risks of unprofitable underwriting of white elephants, for instance railroads without passengers.
- Legal risk of any litigation in unchartered jurisdictions

[18] OECD, *The Contribution of Reinsurance Markets to Managing Catastrophe Risk*, Dec. 2018.
[19] B. Eichengreen, A Mehl and L Chițu, *Mars or Mercury Redux: The Geopolitics of Bilateral Trade Agreements*, ECB Working Paper No. 2246, February 2019

Impact:	low
Most affected business areas	Operations
Time frame (years):	0–3



Conflicting interests – the widening urban-rural divide

The world is urbanising rapidly. More than 50% of world population already resides in cities. While Europe has 35 cities of a million people or more, China will have an estimated 225 by 2025.[20] Cities host many of the world's top research institutions, and the presence of major international business makes them ideal locations to bring together university, government, and private R&D efforts. Capital, knowledge and creativity feed from one other and turn cities into engines of growth, wealth and opportunity.

The flipside is the relative status loss of semi-urban and rural regions. The widening gulf between empowered urban centres and disenfranchised rural peripheries translates into contrasting economic and social realities. In Latvia, for example, about half the country's population lives in the metropolitan area of its capital, Riga, which generates about 70% of the country's economic output. While Riga's standard of living reaches the EU average, in the poorest rural regions, the standard of living is just a third of that average.[21]

Such socio-economic contrast polarises cultural values and political interests. It nurtures resentment and it divides public discourse. The growing political opposition between urban centres and rural periphery is not confined to Latvia. It reflects a broader global development: people who are outward-looking, embrace globalisation and share "progressive" cultural norms tend to live in cities; those rooted in local communities and uphold traditional values tend to live in the rural periphery.

The electoral landscape offers some evidence that a growing cultural divide and segregation between "city" and "countryside" can translate into different political preferences and create unexpected outcomes. Examples include the US presidential election of 2016 and the UK's referendum to leave the EU. In both cases, the peripheral and rural areas outvoted the large cities and metropolitan areas. With persistent division and growing alienation between the poles, more "surprises" may well be forthcoming. For example in the EU, there is evidence that population density is a powerful driver of anti-system voting: regions and localities with lower density are more prone to support anti-European integration parties.[22]

Potential impacts:

- The rapid growth of cities increases economic value concentration. This offers opportunities for the insurance sector, but also accentuates risk accumulation.
- Rural-urban antagonisms could increase market uncertainties, curb investments and endanger functioning supply chains. The cumulative negative impact on financial markets would present challenges for insurers.
- A growing gap between urban centres and the rural periphery could endanger the internal cohesion of territorial states, fan separatist tendencies and make finding policy solutions acceptable to all national socio-economic groups more challenging.
- Economic development could slow if electoral choices and social protests from rural communities prevent policies that enable cities to thrive.
- The (perceived) alienation of geographies and specific regions or localities could create resentments that bring more extremist parties into power, making political and regulatory framework conditions more volatile and detrimental for insurance business.

20 *The Great Migration: Urban Aspirations*, Policy Research Working Paper 6879, The World Bank, 2014.
21 Rudolf Hermann, "Riga und der Rest des Landes", *Neue Zürcher Zeitung*, 29 January 2019.
22 Lewis Dijkstra, Hugo Poelman, and Andrés Rodriguez-Pose, *The Geography of EU Discontent*, European Commission Working Papers, Directorate-General for Regional and Urban Policy, 2018

Emerging trend spotlight

Shifting litigation regimes

Litigation funding and class actions, especially prevalent in the US, are becoming increasingly popular in other parts of the world.

Litigation funding is the upfront payment by a third party of fees associated with a litigation case – such as the plaintiff's own legal costs, or the Adverse Cost Order in case the plaintiff loses – in return for a slice of the compensation received as a result of the lawsuit filed. The ongoing low interest environment makes litigation funding more lucrative to investors and thus drives propensity to sue. In turn, this may lead to more successful – and expensive – insurance claims and ballooning defence costs for the insurer. Well-known in the US, recently the practice has become more prevalent in the APAC region, South Africa and the UK. A landmark decision by the UK court of appeal concerning Mastercard and its fees, greatly lowered the threshold for class actions to proceed. The court also explicitly welcomes third-party funding for litigation. While a further appeal is likely, it is an on-going case example that illustrates that companies in the UK will be needing to spend significant amounts of money to defend against an uptick of class actions. Some of the largest funders, in some cases publicly-traded companies, have significant activities outside the US.

For the insurance industry, the mechanism is a double-edged sword. On one hand, it boosts opportunities to provide products and services to clients facing growing need for liability and legal expenses covers. Stronger consumer protection laws also drive pressure on producers to manage their liability risks by way of product safety and adequate insurance cover. On the other hand, the increased use of third-party funding can result in claims proliferation, longer litigation, more exposure for insurers defending such claims and higher settlement costs. Another worrying trend is the potential of governments to sell its law enforcement authorities to private firms in exchange for a payout. An example is the recent recruitment of external legal counsels by the Office of Attorney General, D.C for climate change litigation. These external counsels will be paid through the contingency fee arrangements in the event of damages awarded by the court.

The other development – class actions – is focused in Europe. Influenced by scandals such as "Dieselgate", the European Commission has proposed a "New Deal for Consumers."[23] This would entail the introduction of a directive on representative actions for the protection of the collective interests of consumers. The core of the proposal is that a qualified entity would be able to bring a representative action before a Member State court or other administrative authority on behalf of classes of consumers. Where Member States do not already allow class actions, they would be required to introduce them. Nevertheless, while the above proposed directive provides progress, a deluge of litigation may not yet be in the inevitable trend due to adverse costs risks, as funders and insurers need to carry out significant due diligence on the cases they may invest in.

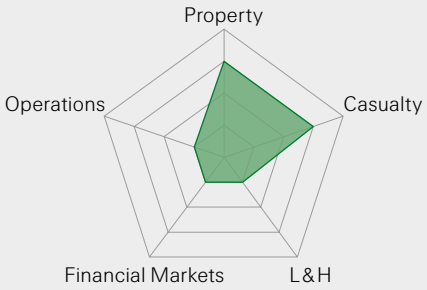
The European Commission considers only litigation-based class or representative actions, but these are not the only mechanisms for collective redress nor the most efficient. Conceivable alternatives include "regulatory redress" with regulatory authority intervention, which would be an agreement or order for redress to be paid. If coupled with an ombudsman scheme, such an alternative dispute resolution (ADR) could present a promising path.[24] The Commission's "New deal" proposal is subject to political negotiations between the European Parliament and the Council. The outcome will have important consequences for the insurance industry.

[23] *Review of EU consumer law – New Deal for Consumers*, European Commission, 2017

[24] Christopher Hodges, *Collective Redress: The Need for New Technologies*, Journal of Consumer Policy, 2018

Technological and natural environment

Impact:	high
Most affected business areas	P&C
Time frame (years):	0–3



Teaching an old dog new tricks – digital tech meets legacy hardware

Digital solutions and old hardware don't always harmonise. A prime and real example happened on 13 July 2017 when thousands of commuters were stranded during peak rush hour as the metro network in Melbourne went into meltdown.[25] The reason was that a 40-year-old interface board being used to connect to the new digitised software was not able to handle the input to the system's tracks and signals, which themselves were also outdated.

This example illustrates the existing challenge. Standard procedure is to seek to improve the operational efficiency of old assets with software enhancements. Technological improvements on software are made on an ongoing basis to keep pace with increasing capacity and complexity of demands. Much hardware, though, is still of yesteryear.

We see an increasing dovetailing of old and new structures, often in areas of critical infrastructure, including smart electric power grids or pipelines, hospitals or cash points. New technology as part of industry 4.0 – a term used to describe technologies like artificial intelligence, quantum computing, 3D printing and IoT in eg production processes – applied to legacy solutions changes the existing risk landscape. While it reduces some old exposures, it also gives rise to new risks. To this end, insurers need to continuously re-evaluate their risks assessments and adapt their underwriting approach to technological innovation as applied in complex, multi-stage, multi-party and sometimes multinational nature construction projects, as well as in legacy infrastructure.[26]

Potential impacts:

- Large infrastructure breakdowns or accidents triggered by new software not working with old hardware can lead to property damage, bodily injury and business interruption claims. There are also new forms of cyber risks to date not priced for.
- Large failures of key infrastructure like power or communications can also impact operations of the insurance industry.
- As technology increases connectivity, insurers face higher risk accumulation and unexpected losses potential from the combination of new software with old hardware.
- While technological innovation can lead to reduced claims frequency in certain areas (eg, advanced driver-assistance systems for vehicles), it may also increase claims severity by introducing new exposures to an existing risk landscape.

[25] Andie Noonan, *Melbourne suffers peak-hour train delays after computer fault*
[26] *sigma 2/2018 – Constructing the future: recent developments in engineering insurance,* Swiss Re Institute

Emerging trend spotlight

The surveillance economy

Many of this publication's readers probably have a social media account. Whether used for professional purposes, to stay in touch with old friends, or exchange views and perspectives, participants benefit from a network of connections which they otherwise would not have access to. And all this for free.

But … just as there's no such thing as a free lunch, there's no such thing as a free internet platform. So how do we pay for these services? We're told that they're funded by the ads that show next to the content we're consuming. We're not that interested in why and how the advertiser chooses to show us that particular commercial at that particular time of day or period in our lives. All the while, however, our online activity – our data – is being used to analyse patterns and predict behaviours, for sale on to the highest bidder. Monetising data is key for corporate profit. According to a recent study, a company is 2.6 times more likely to grow at more than 10% if it monetises data.[27]

The most mundane activities may lead to surprising conclusions. For example, the type of music we listen to online can be analysed for signs of depression, and this information can be then sold for advertising purposes to a medical company. Data on sleep patterns and activity from wearables can be used to confirm the signs of depression gathered from the kind of music we listen to. All this happens without our conscious knowledge and without recourse because our cars, phones, and homes have an increasing number of sensors that link our online and offline lives. Once this knowledge about us has been created, there's no easy way to change it.

For corporates as well as for consumers, today's surveillance economy can generate advantages. Individuals' willingness to "pay" with their data instead of money may translate into competitive advantage and cheaper services. It also helps link consumers with products and services best suited to their needs. On the darker side, however, the surveillance economy can be construed as intrusive and even Orwellian. The constant collection of our data means we are constantly being observed, analysed, compared and rated. And, by knowing what motivates us and understanding our belief systems, corporates can manipulate us, distort the market economy with their asymmetrical knowledge and even the democratic processes by manipulating information flows.

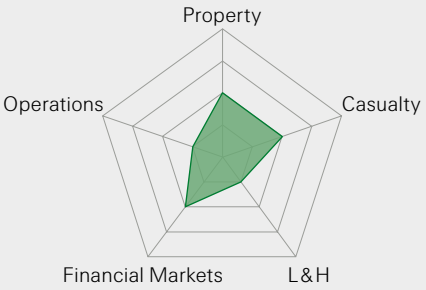
So what are the implications for insurance? According to a recent study on auto insurance, drivers participating in pay-as-you-drive programmes where their driving is monitored and their insurance premiums adjusted based on the data from the car, become 30% safer as a result.[28] This means that monitoring results in safer behaviour, improving the quality of life for the driver and lowering the claims for the insurance company. On the other hand, should an insurance company also receive data from music streaming services about our potential depression, it still may draw inadequate conclusions from correlations, end up reserving excessive amounts for our future medical treatment or face reputational risk and regulatory scrutiny.

[27] *The Dark Side of Data Commercialization*, Forrester Research, 19 April 2018

[28] Yizhou Jin, Shoshana Vasserman, *Buying Data from Consumers – The impact of monitoring programs in US auto insurance*, Harvard Business School, 21 January 2019


5G mobile networks will
enable wireless connectivity
in real time – a prerequisite for
broad use of autonomous cars.

Impact:	high
Most affected business areas	P&C
Time frame (years):	>3



Off the leash – 5G mobile networks

5G – short for fifth generation – is the latest standard for cellular mobile communications. Providing ultrafast broadband connection with higher capacity and lower latency, 5G is not only heaven for your smartphone. It will enable wireless connectivity in real time for any device of the Internet of things (IoT), whether that be autonomous cars or sensor-steered factory. In doing so, it will allow decentralised seamless interconnectivity between devices.

To allow for a functional network coverage and increased capacity overall, more antennas will be needed, including acceptance of higher levels of electromagnetic radiation. In some jurisdictions, the rise of threshold values will require legal adaptation. Existing concerns regarding potential negative health effects from electromagnetic fields (EMF) are only likely to increase. An uptick in liability claims could be a potential long-term consequence.

Other concerns are focused on cyber exposures, which increase with the wider scope of 5G wireless attack surfaces. Traditionally IoT devices have poor security features. Moreover, hackers can also exploit 5G speed and volume, meaning that more data can be stolen much quicker. A large-scale breakthrough of autonomous cars and other IoT applications will mean that security features need to be enhanced at the same pace. Without, interruption and subversion of the 5G platform could trigger catastrophic, cumulative damage. With a change to more automation facilitated by new technology like 5G, we might see a further shift from motor to more general and product liability insurance.

There are also worries about privacy issues (leading to increased litigation risks), security breaches and espionage. The focus is not only on hacking by third parties, but also potential breaches from built-in hard- or software "backdoors." In addition, the market for 5G infrastructure is currently focussed on a couple of firms, and that raises the spectre of concentration risk.

Potential impacts:

- Cyber exposures are significantly increased with 5G, as attacks become faster and higher in volume. This increases the challenge of defence.
- Growing concerns of the health implications of 5G may lead to political friction and delay of implementation, and to liability claims. The introductions of 3G and 4G faced similar challenges.
- Information security and national sovereignty concerns might delay implementation of 5G further, increasing uncertainty for planning authorities, investors, tech companies and insurers.
- Heated international dispute over 5G contractors and potential for espionage or sabotage could affect international cooperation, and impact financial markets negatively.
- As the biological effects of EMF in general and 5G in particular are still being debated, potential claims for health impairments may come with a long latency.

Emerging trend spotlight

Smart construction

The building industry is under construction. Projects are getting more complex, costs are rising and pressure to build quickly is increasing. Driving this trend is growing populations, a global shift towards data-driven solutions, and the increased costs of natural disasters. The last two years were the costliest annual back-to-back period ever in terms of insurance industry losses resulting from natural disasters. The natural catastrophe losses for 2017 and 2018 combined were USD 219 billion.[29]

The above factors are driving change in construction, with greater use of new technologies such as 3D printing, drones, wireless sensors, site automation and prefabricated components. In essence, smart construction is a drive to digitisation. From an insurance perspective, while more data can mean more accurate pricing, there are also challenges regarding data quality and security. Consequently, with increasing use of computer software and automation, associated risks may shift from human error to mechanical malfunction. Risks include cyber vulnerability, data corruption and loss, and inaccurate predictions due to defective modelling.

In 2016, the Dubai government launched its 3D Printing Strategy, with the aim of constructing 25% of buildings using 3D printing technology by 2030. In 2015, a Chinese construction company built the Mini Sky City, a 57-storey skyscraper in central China in 19 working days. The rapid build time was achieved by having 90% of the structure prefabricated in a factory. In Japan, machinery manufacturer Komatsu has been using smart construction technologies since 2015 in over 3 300 sites across the country. This entails using specialised drones to conduct survey work in under one hour, a process that could typically keep many workers busy for several days. An added benefit of such technology could be an increase in worker safety.

All told, traditionally the construction sector overall has been slow to innovate. One factor holding back adoption of new technologies is lack of sufficient data to substantiate the value of offsite manufactured assets across the construction lifecycle. A number of governments, including in the UK, Singapore and Finland, have been making steps towards a more data-driven approach to construction. For instance, they now mandate use of Building Information Modelling (BIM) for public projects to standardise the sharing and exchange of information concerning the construction and post-construction management of a building.[30] But BIM is far from being adopted by all players, especially smaller construction companies. Driving more universal shift to this technology remains a challenge.

As with many other trends in the digital age, to stay competitive insurers must develop new ways to locate, manage and analyse data. At the same time, of paramount importance is to strengthen industry oversight and not overestimate the impacts of innovation. In our view, this will be the only way to protect and advance the principles of sound construction methods.

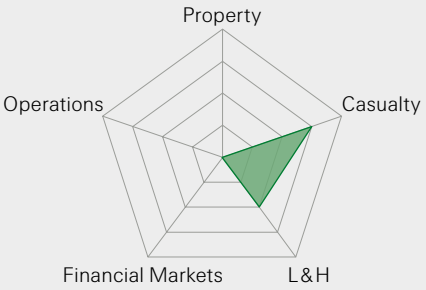
[29] *sigma* 2/2019 op. cit. Read more in *Natural catastrophes and man-made disasters in 2018: "secondary" perils on the frontline,* Swiss Re Institute, 10 April 2019

[30] *sigma* 2/2018, op. cit. Read more in *Digitalisation and its impact on the construction and insurance industries,* Swiss Re Institute, 4 October 2018

Impact: low

Most affected business areas Casualty, L&H

Time frame (years): 0–3



Wiggle room – Artificial Intelligence and healthcare

Medical imaging and related diagnostics are being enhanced by Artificial Intelligence (AI). The US Food and Drug Administration has already approved AI software to support the detection of strokes and fractures based on MRI images.[31] More will follow soon, with many applications beyond imaging pending approval.

Investments in AI healthcare are surging globally. In places where healthcare provision is practically non-existent, affordable AI-driven systems can make treatments more accessible. And in developed countries, a shortage of healthcare workers and instant accessibility and cost efficiency will help build the value proposition and acceptance of AI-driven treatments.

A word of caution, however. Errors in healthcare can have big implications, and longer-term trust in AI-assisted treatments could suffer. Currently, the outcomes generated by AI lack a certain degree of transparency and, thus, accountability. While black-box AI may be fine for shopping, that's unlikely to be the case where AI leads to a wrong decision being made in a healthcare treatment scenario. Patients and insurers alike will need to understand how AI is involved in a doctor's action, and who can be held liable.[32]

Insurers of health-related products should continue to engage with healthcare providers, regulators and customers to ensure proper review of risks of AI in healthcare. Other aims of such dialogue should be to reduce short-term costs and possible liabilities, create standards for explainability and transparency, and shape public perception of AI as being innately beneficial to the provision of healthcare services.

Premature adoption before issues around accuracy, explainability and privacy issues are solved may do more damage than good. A careful, progressive approach, adopting technology that is commensurate with the risks and needs, and that complements human capabilities, is in the longer term interest of patients, healthcare providers and insurers.

Potential impacts:

- In many cases, diagnostics can become more accurate, especially regarding medical imaging. The reverse is true if errors occur leading to catastrophic personal outcomes. In most situations, human interpretation of AI-assisted insights will be a key feature.
- Data used for machine learning may enhance rather than reduce bias, so explainability for ethical and regulatory purposes will be essential, as will be scrutiny of the algorithms used and the decisions taken.
- On the upside. AI in healthcare may enable around-the-clock availability and low cost, thus increasing accessibility to more people.
- Used in conjunction with devices, AI could monitor health 24/7, allowing more rapid identification of problems and better outcomes.
- AI applied to big and combined databases could enhance understanding of health issues, and enhance diagnostics and treatment strategies. However, issues around privacy, compatibility and security need to be overcome first.

31 *FDA permits marketing of artificial intelligence algorithm for aiding providers in detecting wrist fractures*, US Food & Drug Administration, 24 May 2018, and *FDA permits marketing of clinical decision support software for alerting providers of a potential stroke in patients*, US Food & Drug Administration, 13 February 2018

32 "Blame your robot – emerging artificial intelligence legislation", *SONAR*, Swiss Re, 2017

Emerging trend spotlight

The warehouse of the future

The warehouse is the backstage room in retail or department stores, where customers are handed the product they came to the store to buy. If it can't be found in the display, it may still sit in the warehouse. These repositories are the logistical link between increasingly globalised supply chains and consumers on the ground. Warehouses have been around for centuries, and now they are undergoing a revolution.

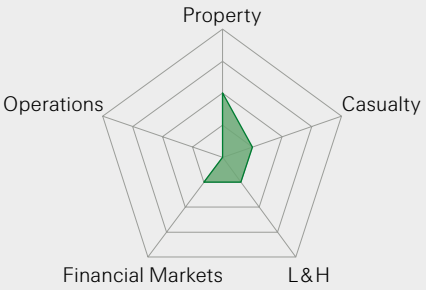
Being relatively closed and controllable systems, warehouses are hotbeds for automisation and robotics. In the warehouse of the future, robotic shelves will move around at high speed. They will feed batches that leave the warehouse, or feed from batches that restock it. Collaboration between humans and robots will continue to be important in the fully automated warehouse. But where humans are involved, they'll only interact from safe islands.

This system is already reality in retail, particularly in pharmacies. A pharmacist feeds the name of a certain drug into the registry system, which is then automatically retrieved from the repository without human involvement. The system also automatically re-orders products, and controls the warehouse according to demand and expiration dates. Informed by former sales, predictive analytics enables optimisation.

On-the-spot 3D printing will gain prominence for certain trades to accommodate demand for customisation and spare parts. Warehouse are set to become much more dynamic, with extended functionalities. Online trade has made front stores redundant: it's no wonder that companies like Amazon are at the forefront of warehouse innovation.

With respect to insurance, we see new risks and opportunities emerging from the more complex, automated and interdependent systems. First, the new warehouses represent a new fire hazard, with high-value concentration risk. If fire protection systems do not work, both the value of the content and the whole supply chain are at risk. With fewer humans and more technology, product liability and professional indemnity losses (for warehouse consultants and engineers), as well as property business interruption losses from flawed hardware and software will gain prominence, while single-loss events from human failure will become more rare. That said, the increasing interconnectivity could also mean a trend towards higher impact loss events, with just one (human or technology) failure having greater consequences.

Impact:	low
Most affected business areas	Property
Time frame (years):	0–3



Resilience at stake – forests' vital functions under threat

Almost a third of our planet's land surfaces is covered by forests. By storing carbon and water, and helping regulate our climate, forests are of enormous value to life.[33] Forests also provide important services like timber, drinking water, food, land slide and storm surge protection, pollinators for agriculture and with their biodiversity genetic resources for pharmacology.

Despite all this, survival of these natural resorts of resilience are coming under ever-increasing pressure. So much so that the destruction of the Amazon rainforest could reach a point where the ecosystem transforms irreversibly into a savannah-like state, with the loss of current benefits accumulating to USD 5 trillion.[34] The situation is just as bad in other parts of the world, such as the Philippines or Indonesia. Key drivers of the destruction of forests are land clearance for agriculture and illegal logging.

Another main concern is that deforestation is in itself a major contributor to climate change, accounting for nearly 20% of global greenhouse gas emissions, more than the whole global mobility sector.[35] This is a self-perpetuating disaster scenario, as under drier and warmer conditions, climate change has also led to increased frequency and intensity of forest fires in Canada, for example. Furthermore, monoculture tree plantations such as for paper are less resilient vis-à-vis natural perils like extreme weather conditions/storms that are occurring more frequently as climates change.[36]

Potential impacts:

- The agriculture sector and associated insurance lines are impacted by water scarcity in deforested areas and by loss of protection from floods, landslides and avalanches.
- Life and health insurance may be impacted via the spread of diseases from cleared forests into cities, driven by increased road access to forests where epidemics can spread from animals to people.[37]
- The biodiversity of forests is deteriorating, putting at risk the cheapest source of clean drinking water, and also undermining the role of forests in protecting us from landslides and storm surges.[38] The related economic activities provide an important basis for forestry and agricultural risk transfer to insurance companies.
- There is a vast potential for insurance as today's commercial forests remain largely uninsured, while an increasing number of commercial forest companies are looking for risk protection/transfer solutions.
- Combined with effective forest management, forestry insurance can strengthen the resilience of forests and reduce emissions from degradation and deforestation.[39]

33 Climate Change 2014: Synthesis Report. Contribution of Working Groups I, II and III to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change, IPCC, 2014

34 Franklin, S and R Pindyck *Tropical forests, tipping points, and the social cost of deforestation,* Ecological Economics, Vol. 153, 2018

35 https://www.ipcc.ch/site/assets/uploads/2018/02/SYR_AR5_FINAL_full.pdf

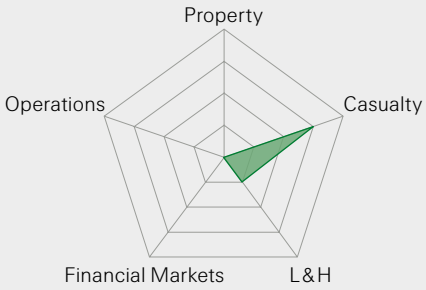
36 Adam Felton et al., *Replacing monocultures with mixed-species stands: Ecosystem service implications of two production forest alternatives in Sweden,* US National Library of Medicine and National Institutes of Health, February 2016

37 Jim Robbins, *How Forest Loss Is Leading To a Rise in Human Disease,* Yale School of Forestry&Environmental Studies, 23 February 2016

38 Winnie Hu, *A Billion-Dollar Investment in New York's Water,* The New York Times, 18 January 2018; Brian Blankespoor, Susmita Dasgupta,Glenn-Marie Lange, *Mangroves as a protection from storm surges in a changing climate,* US National Library of Medicine and National Institutes of Health, May 2017; *The State of the World's Forests,* Food and Agriculture Organisation of the United Nations, 2018

39 *Forestry insurance; a largely untapped potential,* Swiss Re, 8 December 2015

Impact:	low
Most affected business areas	Casualty
Time frame (years):	0–3



Pervasive and toxic – chemicals in our bodies and environment

The chemicals behind Teflon and many other materials used in cookware, food packaging (food wrappers, pizza boxes etc), carpeting, upholstery, fire-fighting foam, apparel, floor wax, textiles and sealants belong to a family of substances known as PFAS (per- and polyfluoroalkyl substances).[40] They have achieved notoriety by showing up in the blood samples of 98% of all Americans, not surprising given their ubiquitous presence in the environment. It is equally not surprising that this finding has invited legal actions. The most notable case currently is a 2018 class action lawsuit brought against major producers on behalf of Americans who have been exposed to PFAS chemicals.[41]

PFAS are just one of a group of chemicals in widespread use and with resulting persistence in the environment where the time between application and first showing of potential negative side effects can be very long. Given the potential dangers that these chemicals pose, regulators across the globe are taking action. In 2007, the EU enacted the REACH legislation to improve the protection of human health and the environment from the risks posed by chemicals.[42] The law requires companies to demonstrate how the substance can be used safely.

China, meanwhile, has introduced a similar law, and the US has overhauled its chemical regulation TSCA.[43] There are differences between how the laws regulate chemicals, but they all require more transparency with respect to ensuring the safety of human life and the environment. Such transparency will help make the world a safer place, but will also generate potential for new lawsuits.

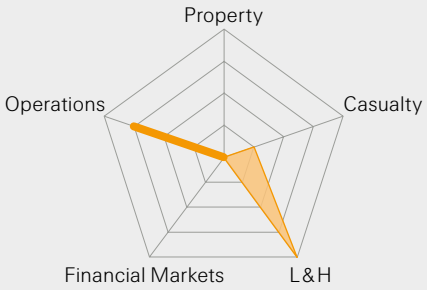
Potential impacts:

- Product liability and recall claims are to be expected in case of known or potentially negative side effects on human health from exposure to chemicals.
- Environmental release on a gradual or accidental basis could impact environmental liability policies, including clean-up costs.
- Employers' liability and workers compensation claims may be triggered where a connection between workplace-related diseases and certain chemicals is established.

40 As according to *United States Environmental Protection Agency*.
41 Sharon Lerner, *Nationwide Class Action Lawsuit Targets Dupont, Chemours, 3M, and Other Makers of PFAS Chemicals*, The Intercept, 6 October 2018
42 REACH stands for Registration, Evaluation, Authorisation and Restriction of Chemicals. See *Understanding REACH*, European Chemicals Agency
43 TSCA stands for Toxic Substances Control Act. See *TSCA Inventory*, US Environmental Protection Agency. Also *China REACH (MEP 7)*, www.chemsafetypro.com, 30 December 2015


An often long time lag between application and the first signs of potential negative side effects of chemicals make protection challenging.

Impact:	high
Most affected business areas	L&H
Time frame (years):	0–3



Don't ask, don't tell – genetic testing and adverse selection

Since publication of our 2017 SONAR report on cancer screening and liquid biopsies, genetic testing has been widely adopted by public health systems and individuals. This has significant implications for life insurers, not least in respect to the regulatory constraints involved.[44]

The major challenge for life insurers is to obtain adequate and risk-relevant information during the underwriting process, since existing regulation was mostly enacted before the widespread distribution of direct-to-consumer (DTC) genetic tests. Generally, regulation disallows the use of genetic information in underwriting life insurance. This raises the prospect of more customers at higher risk of disease or mortality applying for life insurance, leading to adverse selection. Customers in the know may also fear being denied life cover due to some genetic conditions, leading the insured to withhold such information from the insurer.[45]

A new generation of predictive genetic tests based on polygenic risk scores (which attempt to quantify the cumulative effects of a number of genetic variants to display predisposition to a disease[46]) promoted by companies such as 23andMe and YouSurance, is only likely to widen the information gap between insurer and insured. Nevertheless, some insurance groups have been looking at the upside potential.[47]

Regulation that stimulates genetic information asymmetry will significantly impact insurers' ability to offer attractively priced coverage, and may challenge the way in which insurance risk is considered and managed. Insurers must be able to evaluate relevant consumer information when underwriting, and that includes risk-relevant data from genetic tests. Currently, there seem to be three broad regulatory approaches to access and use of genetic data for risk assessment: none/self-regulation, limitations by law, and outright legal ban. This lack of uniform approach shows the need for industry groups and regulators to work together to agree on reasonable self-regulation, one that balances the interests of consumers while maintaining the ability of insurers to underwrite sustainable products.[48]

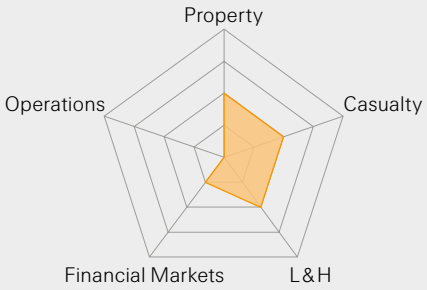
Potential impacts:

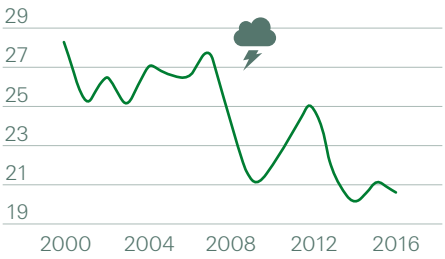
- Loss developments can be worse than expected if those at increased risk buy disproportionate insurance cover, while those not exposed to genetically-triggered diseases stay away.
- As with any new innovation, there will be a challenging transition period in which insurers will need to develop the know-how of capturing and managing the data, design systems to incorporate the data and implement new underwriting approaches.
- As the results yielded by genetic tests become more accurate and their use becomes more widespread, the way insurers traditionally pool risk to differentiate individual risks may no longer be suitable.
- Allowing access to an insured's genetic information would enable more accurate risk assessment.
- In addition, access to genetic information could improve customer engagement and services. New value-added products to cover specific diagnostics, or services tailored to the insured's health goals, could create an active partnership between life insurer and insured, vastly improving customer retention.

44 See *2017 SONAR* report, Swiss Re
45 *Seeing the future? How genetic testing will impact life insurance*, Swiss Re Institute, 2017,
46 *Polygenic risk scores: how useful are they?* Genomics Education Programme, 25 October 2018
47 *The Risk of Anti-Selection in Protection Business from Advances in Statistical Genetics*, Reinsurance Group of America and Kings College London, August 2018
48 *Can life insurance pass the genetic test?* Swiss Re Institute, 2019


Genetic testing can create an
information asymmetry between
customers and insurers.

Impact: medium
Most affected business areas P&C, L&H
Time frame (years): >3





Life insurance has not recovered since the financial crisis (life insurance in trillions USD)

Source: Swiss Re Institute, Life underinsurance in the US; bridging the USD 25 trillion mortality protection gap, September 2018

Risky bets? Insurance demand in an age of shifting markets

Globally, too many people are un- or underinsured and not protected when a catastrophe strikes. In 2018, the estimated worldwide protection gap for global catastrophe risks stood at around USD 80 billion.[49] The mortality protection gap is even larger. In the US alone, the mortality protection gap, a measure of life underinsurance, was close to USD 25 trillion in 2016.[50]

These numbers should be seen against the backdrop of shrinking middle classes in developed markets, the primary target group for many insurance products. The OECD defines middle class income as being between 70% and up to 200% of the median income. While almost 70% of baby boomers were part of middle-income households in their twenties, only 60% of millennials are today.[51] In all OECD countries – except Switzerland, Ireland and Spain – the income portion of the middle class is diminishing. According to McKinsey Global Institute, 98% of all households in 25 developed economies have seen their income rise between 1993 and 2005.[52] The picture is very different for the years 2005 to 2014 when 70% of households experienced flat or even falling income. Moody's paints a similar picture for the US, saying that rising prices and interest rates will erode disposable income. Shrinking disposable incomes may put willingness to pay for insurance coverage to the test. We already witnessed this in the US, after the global financial crisis of 2008–09. There was a significant widening of the mortality protection gap between 2007 and 2010 due to a large increase in joblessness and decrease in household asset values and increase in debt. When people were asked why they didn't buy life insurance even though they knew about the gap, 61% said they have other financial priorities.[53]

From an insurance industry standpoint, the hope – or wishful thinking – is that any shortfalls in the West can be offset by gains in the East, especially China. Indeed, China has already overtaken the US in respect to its global GDP contribution, and is projected to remain the growth engine of the world economy. But growth rates are slowing down. Although insurance potential continues to move East driven by demand from the thriving middle classes there, the risk of growing income inequality there is considerable, especially in the longer run.

Potential impacts:

- China has already overtaken the US in terms of contribution to global GDP and is projected to remain the growth engine of the world economy. Even so, its growth rates are slowing, and that could curb insurance demand.
- Insurance potential continues to move East. The thriving middle classes are to be found in Asia rather than in the West. However, there is risk in the longer run that the middle classes do not grow as expected, which could also lead to rising income inequality.
- Middle income households experiencing relative status loss and financial pressure tend to cut expenses deemed not essential, and insurance often falls into this basket.
- Whether real or just perceived – middle classes in the US and Europe are claiming status loss, which often translates into resentment towards "establishment" and "foreigners." The populist behaviours at the ballot box and social unrest on the street bring more uncertainty and volatility to these market environments.

[49] *sigma* 2/2019 op. cit.
[50] *Life underinsurance in the US: bridging the USD 25 trillion mortality protection gap,* Swiss Re Institute, Expertise publication, Sept 2018
[51] *Under pressure: the squeezed middle class,* OECD, April 2019
[52] *Poorer than their parents? Flat or falling incomes in advanced economies,* McKinsey Global Institute, 2016
[53] *Life underinsurance in the US; bridging the USD 25 trillion mortality protection gap,* Swiss Re Institute, September 2018


Globally, too many people
are un- or underinsured.

Emerging trend spotlight

Financial services and the digital revolution

Digitisation is all the rage in financial services. This has been the case for a long time already and everybody agrees it's happening. Ironic, therefore, that there is little consensus about what it actually means, how it's changing the industry, or what actually needs to be done.

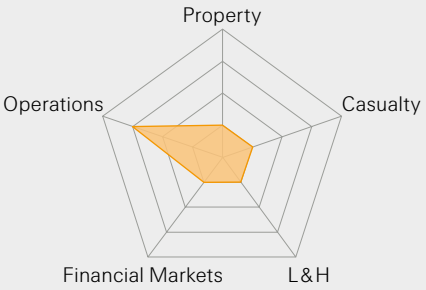
The expected bitcoin upheaval, which started a couple of years ago with calls to bring down the traditional banking system, failed to materialise. On the other hand, smart contracts and the underlying blockchain technology have been adopted by traditional players in banking and insurance, although some critics claim that blockchain itself still isn't living up to its promise.[54]

Nevertheless, nervousness among large financial institutions about innovative start-ups persists, both with regards to potential competition and investment opportunities. There are daily reports about digital disruption and how to survive. The consulting companies in particular are eager to promote digital disruption, offering guidance and a helping hand. Everyone wants to be a winner, but nobody exactly knows what direction to take. Which element of the digital revolution needs to be prioritised? How quickly do we need to adopt new behaviours, and what skills are required? And for re/insurers, what does this all mean?

The problem many institutions face is a clash of culture and hierarchy. Technologically affine youngsters come up against experienced and established caution, meaning their skills are not always deployed most effectively. On the other hand, experience and tradition is not necessarily a bad thing, not where it prevents companies from falling for the wrong technical innovation. Tension between old tradition and new ideas can be healthy. Smart digitisation strategies are not only about how to be ahead of everyone else, but also about the traps and risks to avoid. Too much haste may lead to costly mistakes, institutional memory loss, mispricing, increased cyber vulnerability and a generally bad risk culture. To consider trade-offs, to find the right balance, that's where the challenge lies. Finding a safe pathway through that digital challenge looks set to keep financial services firms busy for a while yet.

[54] *Blockchain's Occam problem*, McKinsey & Company, January 2019

Impact:	medium
Most affected business areas	Operations (incl. regulation)
Time frame (years):	0–3



Getting the balance right – technology regulation affecting the insurance industry

As technological innovation advances, regulation follows on its heels. We are now seeing a first wave of non-insurance tech regulation spilling over into the insurance industry. Much of this is about access to and use of customer data, such as the General Data Protection Regulation (GDPR) and ePrivacy Regulation coming out of the European Union. These have created momentum for similarly broad data protection regulation in other locations, which are both expansive in how businesses collect, store and use customer data, and come with cross-border implications. Even the passive storage of cloud-based data in an overseas jurisdiction entails regulatory, political and business risks. Microsoft's jurisdictional battle with US law enforcement over customer data stored in its Ireland data centre is a case in point.

Excessive data protection requirements like limitations on cross-border data sharing can hinder the ability of insurers to utilise data in meaningful ways, including developing more personalised solutions and more frequent and meaningful interactions with clients. To ensure fair treatment of end users and avoid discrimination, re/insurers have to take a responsible approach. Increasingly in the spotlight are the potential risks arising from the operational aspects of tech innovation such as cloud services, outsourcing and cybersecurity. The consequent general lack of global harmonisation means there's a risk of conflicting laws.

In addition, global fragmentation of cybersecurity laws could increase operational costs and compliance risks. This could impact companies' future use of data-linked technologies. Outsourcing IT infrastructure to the cloud offers access to innovative services in many areas such as robotics, mobility and Big Data. New regulatory requirements that limit the use of cloud computing could hinder the development of a compelling digital service portfolio for a customer base, one that's increasingly asking for digital experience in service delivery.

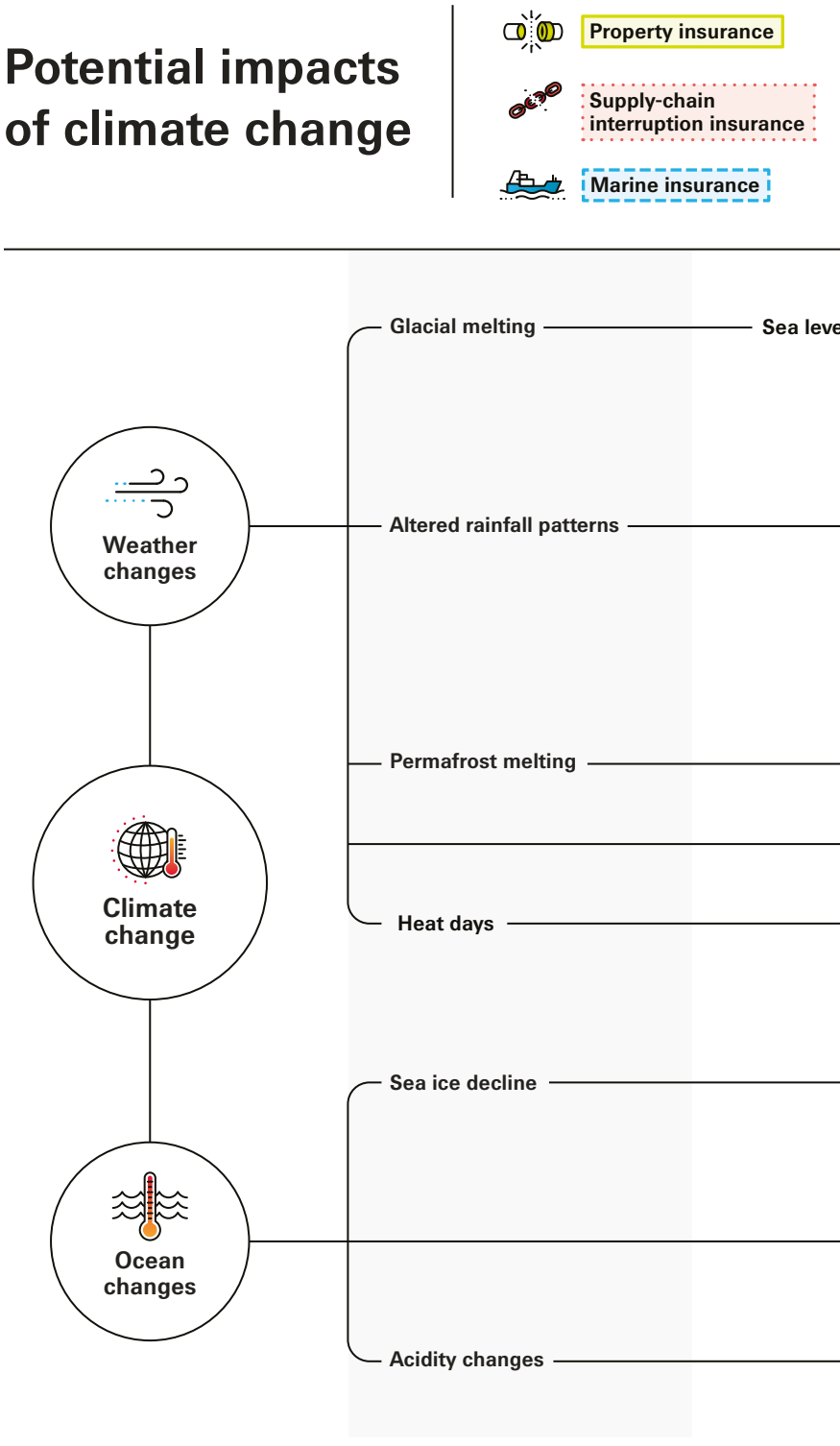
The lack of global harmonisation means risk of conflicting laws. For this reason, it is important that regulator efforts be well coordinated between jurisdictions. Technological development in an increasingly interconnected digital environment underscores the need for collaboration between regulators. Sound international standards are a prerequisite to optimise the insurance value chain from end-to-end.

Potential impacts:

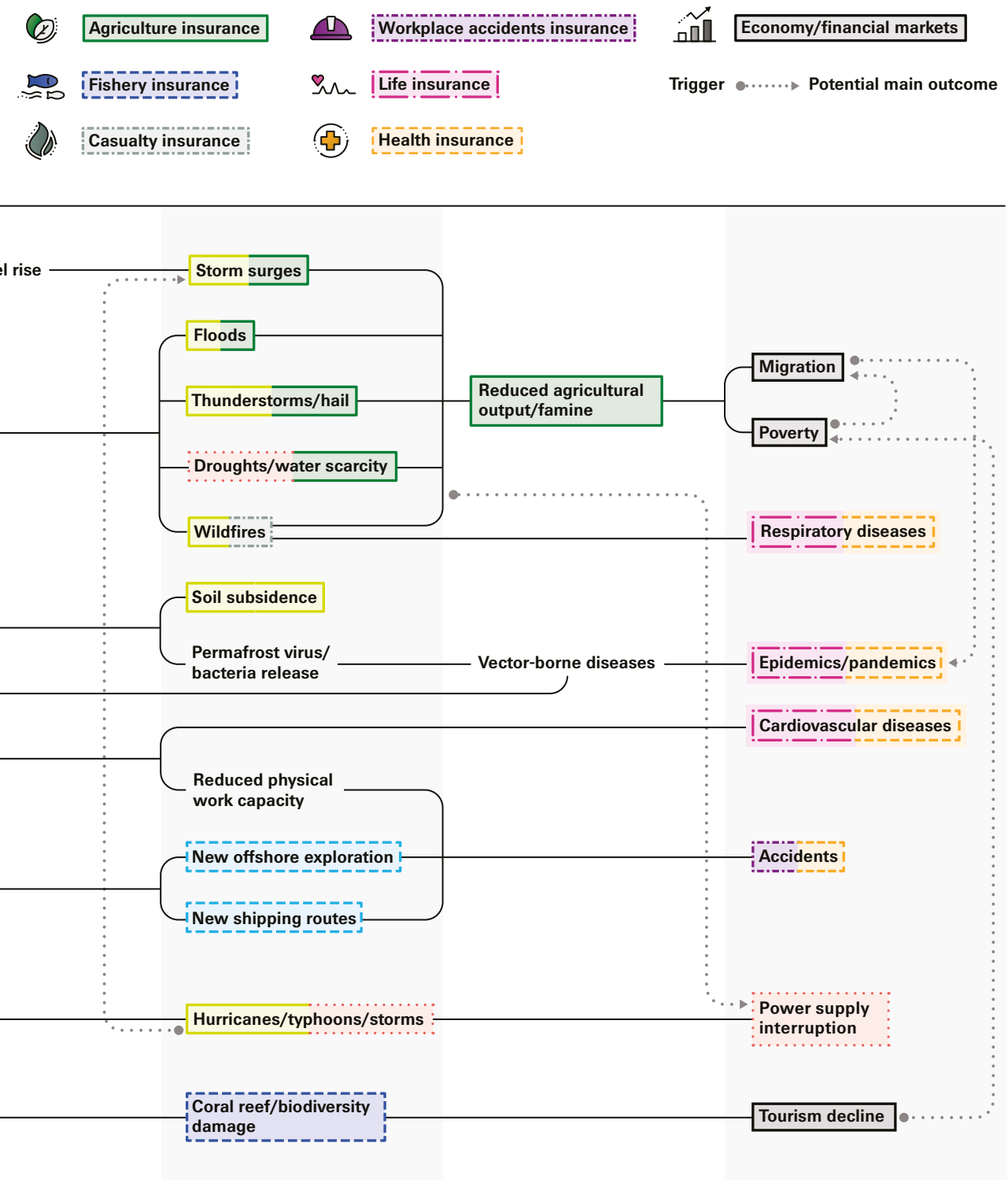
- Laws and regulations relating to technological developments need to be well balanced and internationally coordinated. Otherwise, they could greatly increase a multinational insurers' legal, compliance and regulatory risks.
- Specific insurance regulation and general regulation eg data protection might not be aligned and cause friction.
- Legacy regulation and/or regulatory roadblocks limit timely utilisation of new technologies and hinder meaningful strategic partnerships.
- New data protection regulation might make the use of data for insurance purposes more difficult, thus obstructing fair risk assessment and forcing cross subsidisation.
- Digital transformation is necessary to stay competitive. Regulators should not restrict the use of cloud services but rather ensure that insurers have processes in place to identify, manage and mitigate the risks that cloud usage presents.

Climate change – from emerging risk to real-life danger

Climate change was on Swiss Re's emerging risk agenda in 1989 already, long before the term "emerging risk" was coined. Since then, climate change has fully emerged as a real and present-day problem. First impacts such as the greater frequency and severity of wildfire events are already showing. As the following infographic depicts, however, climate change risks extend to a wide-reaching scope of threats that society, and the insurance industry, are exposed to today. While many areas of climate change have been researched extensively, one risk area that has not, to date, been afforded the same degree of investigation is the impact of climate change on human life and health. We expect the impacts here to also be significant and therefore in the following section, bring the focus to exploring how societies and healthcare systems will need to adapt to a warmer world.



This graphic is intended to foster the risk dialogue and tries to explain complex interactions and interconnections in a simplified way. The graphic does not pretend to be comprehensive or complete. Please also take a look at the interactive version on swissre.com/SONAR2019


Agriculture insurance
Workplace accidents insurance
Economy/financial markets
Fishery insurance
Life insurance
Trigger
Potential main outcome
Casualty insurance
Health insurance
el rise
Storm surges
Floods
Thunderstorms/hail
Droughts/water scarcity
Wildfires
Soil subsidence
Permafrost virus/ bacteria release
Reduced physical work capacity
New offshore exploration
New shipping routes
Hurricanes/typhoons/storms
Coral reef/biodiversity damage
Reduced agricultural output/famine
Vector-borne diseases
Migration
Poverty
Respiratory diseases
Epidemics/pandemics
Cardiovascular diseases
Accidents
Power supply interruption
Tourism decline

It's existential – climate change and life & health

While not all the threats of climate change are fully understood, it fully emerged as a present real life risk. The most pronounced risks from climate change affecting human health stem from heatwaves, floods, droughts, fires and vector-borne diseases. Millions of lives can be saved and the burden on healthcare services reduced if we pay more heed to changing climates. Without action, mortality rates and healthcare costs could soar,[55] and this would have significant consequences for the health, workers' compensation and life insurance lines of business.[56]

Changes in mortality will be driven by several changes occurring simultaneously. First, we expect that heatwaves will become more severe and extend to areas previously not impacted. This includes in the temperate zone, where a large proportion of the world's population lives. The 2003 heat wave in France was a first taste of things to come. It caused 70 000 deaths – mainly among the elderly.[57] With no mitigation, and with rapidly ageing populations in many countries, a future event will have an even bigger impact because the share of vulnerable older populations in the affected regions is rising fast.[58]

Increasing temperatures and high humidity due to climate change is another area of concern, in that this combination enables vector-borne diseases to conquer new ground. The West Nile and Zika epidemics were first warning signs.[59] Climate change will extend the transmission season and geographical range for many infectious diseases.[60] For example Lyme disease, avian influenza, meningitis, dengue fever and tropical bacterial and viral infections are projected to increase with climate warming, including potential shifts in their geographic range.[61] Severe drought conditions can lead to increased wildfires, which in turn lead to air pollution. This is even in areas far away from any conflagration, as the California wildfires of 2018 showed.[62]

Knock-on effects

The secondary impact of climate change will amplify the above. In focus here are migration, urbanisation, food security & nutrition, and water scarcity. Already today, more than 2 billion people live in areas of water stress,[63] areas where access to clean water for drinking, sanitation and personal hygiene – all pre-requisites for public health – is limited.[64] The number of regions affected by water stress will increase as temperatures rise. Public health could be further compromised by the expected increase of, for example, extreme heat, droughts and floods affecting agriculture. This will diminish or destroy the nutritional supply chains that help people withstand health threats.[65] Nor will fishery compensate because rising sea temperatures and ocean acidification will likely mean lower catches.[66]

55 *COP24 Special Report: Health and Climate Change,* World Health Organisation, 2018
56 sigma 6/2018, op. cit.
57 *Death toll exceeded 70 000 in Europe during the summer of 2003,* Comptes Rendus Biologies, Vol. 331, No, 2, February 2018
58 *The 2018 report of the Lancet Countdown on health and climate change: shaping the health of nations for centuries to come,* The Lancet, 8 December 2018
59 *Impacts of 1.5C of Global Warming on Natural and Human Systems,* IPCC, 2018
60 *Human health and adaptation: understanding climate impacts on health and opportunities for action. Synthesis paper by the secretariat,* United Nations, March 2017
61 *Impacts of 1.5C of Global Warming on Natural and Human Systems,* op. cit.
62 *'No fresh air': wildfire smoke sets apocalyptic haze over San Francisco,* The Guardian, 13 November 2018
63 *SDG 6 Synthesis Report 2018 on Water and Sanitation,* United Nations, 28 June 2018
64 *Human health and adaptation: understanding climate impacts on health and opportunities for action. Synthesis paper by the secretariat,* op. cit.
65 *The State of Food Security and Nutrition in the World,* Food and Agriculture Organisation of the United Nations, 2018
66 *Heatwaves take their toll on the high seas,* Nature: international journal of science, 4 March 2019

Extreme weather events will not only impact agriculture. With continued sea level rise,[67] storm surges may reach further inland. With increased value concentration in mega-cities exposed to storm surges, economic and insured losses are likely to increase further. So too will the risk of epidemics. Migration to urban centres is concentrating an ever larger share of the world's population in a small areas. As has happened many times before, storms and floods that destroy infrastructure can trigger significant epidemics. And, once the flood water recedes, toxic mould may remain in buildings, posing yet another threat to public health.

In polar and other regions were permafrost has long been the environment norm, another possible consequence of warmer temperatures could be the release of older bacteria and viruses as ice thaws. Having not been exposed to these strains for thousands of years, the immunity of the world's population to such threats will be low. The building of new harbours to accommodate increasing marine traffic with the opening up of the arctic sea route, will make it more likely viruses or bacteria will be able to travel to far distant locations, triggering an epidemic or pandemic. Such outbreaks could be especially serious if the bacteria prove resistant to antibiotics (see section in the 2017 SONAR report on antibiotic resistance).

Becoming climate resilient in public health

The world community has acknowledged the imperative with the "Paris agreement on climate change" which aims to keep global warming well below 2 degrees Celsius in the long-term. In addition, it outlined in the WHO COP 24 special report on Health & Climate change what to do to save lives, reduce epidemics and make public health climate resilient.[68]

The immediate public health activities necessary to meet the challenge of climate change are to strengthen the prevention of climate-sensitive health risks and to build an adaptive skill set to absorb the changing, increasing risks presented by climate change described before. This also involves related sectors like food security and safety or sanitation. Additionally, however, there are other specific risks the health sector must address to achieve resilience in the face of climate change.

Health care facilities are the operational heart of service delivery, protecting health and treating patients, both during and after weather and climate-related events (such as heat stroke during heatwaves and injuries during cyclones) and in response to other environmental risks to health (such as asthma due to poor air quality). Health care facilities in poor and rich countries alike must be able to deliver in changing climate conditions, such as during extreme weather events. Cooling systems during heat days, flood security, emergency power and water supplies must become standard in health care facilities worldwide.

67 *Impacts of 1.5C of Global Warming on Natural and Human Systems,* op. cit.
68 *COP24 Special Report: Health and Climate Change,* op. cit.


Many workers in the oil and gas industry will retire in the next few years: This will create a skills gap.

Appendix A: High impact emerging risk themes, 2015–2019

Every year Swiss Re publishes new emerging risks in its SONAR report. Occasionally, an emerging risk is reported again in a later report in case new aspects emerge or if the risk persists with increased urgency. Some overarching themes and common aspects are usually reflected in every report, often in the list of emerging risks ranked with highest impacts. For 2015–2019, here we list the top risks of each year and discuss how the overarching concerns regarding market dynamics, cyber risks and climate change have been profiled in specific high impact emerging risk themes over the years.

Market dynamics

As a key sector of the financial industry, the insurance industry is exposed to market dynamics. In 2015, the euro zone crises and expansive monetary policies were in the foreground, as were concerns over domestic populist movements fostering de-globalisation. While uncertainties from central bank quantitative easing continued in 2016, a potential slow-down of emerging market growth was added to the top emerging risks list. International regulatory fragmentation and protective trade barriers where each flagged as top risks in 2017, next to a return of inflation. Then, in 2018 national protectionism and regulatory fragmentation to hamper diversification for international re/insurers were synthesised into a top risk under the theme "A brave new world?" Heightened geopolitical tensions and trade conflicts are further confirming ongoing concern about the volatile global business environment for insurers.

Cyber risks

Cyber risk presents one of the largest opportunities for the re/insurance industry, while simultaneously presenting one of the largest challenges. The frequency and severity of risks resulting from cyber-attacks and their changing nature, are expected to grow significantly over the next years. The need for cyber resilience has become a main focus of attention among corporate clients and insurance companies, triggering insurance demand. The SONAR report has flagged the evolving aspects of technological change and uncertainty in a series of cyber risk themes over the past years. This includes a focus on vulnerabilities from the IoT (2015), the challenges from increased internet fragmentation (2016), accumulation risks from cloud solutions (2017) and lurking cyber risk from end-of-life software and hardware still in use (2018). In this year's report, we focus on exposures from 5G mobile and where software enhancements are applied to existing legacy hardware, particularly in the case of large infrastructure facilities.

Climate change

Swiss Re identified climate change as an emerging risk 30 years ago. Three decades on, we continue to flag the impact of climate change on specific risk factors and pools. For example, in 2015 we highlighted the associated theme of potential super natural catastrophes from "atmospheric rivers" affecting the US West Coast. We then raised a closer connection to climate change in the form of growing water stress and drought potential in our 2017 SONAR report, in our exposé on "The big drying". Today, climate change has become a fully-emerged risk, and in this year's report we have a dedicated section on its impact on life & health insurance.

Appendix A: High impact emerging risk themes, 2015–2019

2015	2016	2017	2018	2019
De-globalisation	The great monetary experiment (cont.)	Bugs on the march – underestimated infectious diseases	Asbestos reloaded – USD 100 billion in losses and counting	Limits to tinkering – the fiscal and monetary policy balance at risk
The great monetary experiment	Internet fragmentation	Reduced market access – protecting your own backyard	A brave new world? – emerging geopolitical risk	Teaching an old dog new tricks – digital tech meets legacy hardware
Super nat cats	Emerging market crises 2.0	The perfect storm – cloud risk accumulation	Algorithms are only human too – opaque, biased, misled	Off the leash – 5G mobile networks
Challenges of the Internet of Things		The big drying – growing water stress	Coming back to bite us – lurking cyber risks	It's existential – climate change and life & health (Special feature)
		The return of inflation – the effect on insurance business	A slow poison – the erosion of risk diversification	Don't ask, don't tell – genetic testing and adverse selection
		Island solutions – regulatory fragmentation		

Societal environment

Political environment

Technological and natural environment

Competitive and business environment

Listed above are our risk themes from the SONAR reports 2015–2019 with high impact potential. Risks with medium and low impact potentials are not listed. These high impact risks are colour-coded according to their respective macro trend environments. Together with the following descriptions per risk, the synopsis reflects a "high priority portfolio" of emerging risk, as it develops over time. It also allows "to keep track" of older emerging risks and reflect that some of the risks are reported once only, others reappear, and some overarching concerns (see page 47) are monitored throughout the years.

Summary of high impact emerging risk themes (2015–2019)

2015

De-globalisation: Political conflicts have been intensifying over the last few years in many regions including Eastern Europe, the Middle East and East Asia. Sanctions and other interventionist policy tools have led to economic distress, driving an upsurge in populist and nationalist sentiment. In Europe, this could trigger territorial separatism (eg, Scotland or Catalonia) and eventually undermine integration projects such as the European Union.

The great monetary experiment: The euro area debt crisis lingers on, with only modest growth, high unemployment and unsustainable debt levels in some countries. Traditional policy tools, including expansionary fiscal policy and monetary easing, are no longer feasible. Nevertheless, extremely accommodative monetary policies continue and even intensify. Short- to mid-term consequences include extremely low interest rates, distortions of risk-return profiles, potential asset price bubbles, and increasing economic inequality. Longer-term consequences include potential for higher inflation and reputational damage for central banks.

Super nat cats: The US Geological Service published a study on a winter storm scenario, looking at the impact of an "atmospheric river" event with a return period of 1 000 years. Findings indicate that flooding would overwhelm flood protections in many areas, resulting in the evacuation of more than a million residents, direct property damage of nearly USD 400 billion and business interruption costs of about USD 325 billion. The risk of volcanic eruptions might also be underestimated as no large eruption has occurred since 1815. However, eruptions can have a devastating impact if they occur close to population centres. They can also impact global travel, as illustrated by the eruption of Iceland's Eyjafjallajökull volcano in 2010.

Challenges of the Internet of Things: The IoT will revolutionise the digital world. Increased connectivity and reliance on digital processes raises questions about network and data security, resilience and long-term maintenance and software updates. Losses could occur from system malfunction and malicious attacks from hackers and criminals. There may also be legal and compliance risks due to the lack of consistent regulatory standards across countries.

2016

The great monetary experiment (cont.): Quantitative easing continues, resulting in a low to negative interest rate environment. Economic growth and inflation remains tepid in the euro area and Japan, triggering discussions about additional monetary policy stimulus. Negative interest rates will further undermine the conventional insurance business model, particularly for life insurers and pension funds.

Internet fragmentation: Cyber crime and espionage have grown strongly, making the internet less safe. Governments urge more effective protection of online assets and consider isolating critical IT infrastructure from global networks. Disconnected national/regional nets will become more common. Technology companies risk disruption to their business model and might face liability suits if no longer able to access data stored on cross-border servers.

Emerging market crises 2.0 – Amid rising US interest rates, economic growth in China has continued to slow, with knock-on impact on commodity prices leading to net capital outflows from emerging markets. Emerging market turmoil could hurt insurers' balance sheets and may trigger detrimental regulatory consequences.

2017

Bugs on the march – underestimated infectious diseases: The risk factors associated with infectious diseases, even known ones, are changing (eg, climate change, animal husbandry, land use, and poor health in areas connected with the world economy). These factors could facilitate outbreak and proliferation of infectious diseases.

Reduced market access – protecting your own backyard: Free trade, open markets and globalisation are coming under increasing pressure, with governments favouring local markets and national champions. Protectionism is no longer an emerging market phenomenon.

The perfect storm – cloud risk accumulation: Ever more widespread use of cloud and cloud-of-clouds solutions comes with a variety of risks: cyber attack, technical failure, prolonged outage and data inaccessibility. The data volumes involved and service interruption potential pose significant and catastrophic risk to system resilience.

The big drying – growing water stress: Farming, industrial use and household consumption are exacerbating water shortages in a growing number of regions (eg, California, US mid-West, southern Europe, the Mediterranean, South Africa). Severe water shortages also have an adverse impact on food production and can undercut oil and gas production.

The return of inflation – the effect on insurance business: Inflation is picking up in US and UK (not yet Europe and Japan). A sudden increase in inflation can adversely impact insurer profits. The long-term effect of accommodative monetary policy of recent years remain unclear.

Island solutions – regulatory fragmentation: International regulatory coordination activities among G20 are increasingly stalling, diminishing the chance for international standards and norms, and leaving an uneven playing field. Regulatory island solutions increase coordination and operational costs, and compliance burden to multinational insurers.

2018

Asbestos reloaded – USD 100 billion in losses and counting: Millions of metric tons of asbestos are still being processed in many countries. A UN report showed that over 300 million people in Europe and Central Asia are potentially exposed. Latin America and other regions are at risk also.

A brave new world? – emerging geopolitical risk: The global political and economic balance has become multi-polar. Global institutions lack mitigating power in circumstances of conflict. Aggressive propaganda, cyber-attacks and other means of "hybrid war" between nation states increase uncertainty.

Algorithms are only human too – opaque, biased, misled: Algorithms are susceptible to discriminatory bias. Black-boxed workings of algorithmic calculations can conceal and perpetuate mistakes. What's lacking is governance around development and application of algorithms.

Coming back to bite us – lurking cyber risks: Flaws and vulnerabilities in hardware (chips) and software may remain undetected for a long time (eg, "sleeper" cyber risk played out in recent WannaCry-attack). The risk is mispricing in cyber-covers, which may in turn impact operations.

A slow poison – the erosion of risk diversification: Re/insurance provides financial protection from risks by deploying capital across borders and lines of business. National protectionism and regulatory fragmentation are jeopardizing the benefits of international diversification.

2019

Limits to tinkering – the fiscal and monetary policy balance at risk: There is a growing consensus that another economic downturn will need a fiscal response. Potential responses include quantitative easing, "helicopter money" or modern monetary theory. The re/insurance industry could benefit if changes to policy bring growth and financial stability. The possible flipside is a rise in uncertainty, causing higher financial market volatility and declines in asset valuations.

Teaching an old dog new tricks – digital tech meets legacy hardware: Technological improvements are ongoing. Hardware in areas of critical infrastructure, including smart electric power grids or pipelines, hospitals or cash points, however, is often out dated. As a consequence, insurers face higher risk accumulation unexpected loss potential in the areas of property damage, bodily injury, business interruption and cyber risk.

Off the leash – 5G mobile networks: 5G will enable wireless connectivity in real time for any device of the IoT, such as autonomous cars or sensor-steered factory. Current concerns regarding potential negative health effects from electromagnetic fields are likely to increase. Hackers can also exploit 5G speed and volume to acquire (or steal) more data faster. Major concerns are possible privacy and security breaches, and espionage.

It's existential – climate change and life & health: The most pronounced risks from climate change affecting human health stem from heatwaves, floods, droughts, fires and vector-borne diseases. Millions of lives and healthcare services could be at risk. Without action, mortality rates and healthcare costs could soar, and this would have significant consequences for the health, workers' compensation and life insurance lines of business.

Don't ask, don't tell – genetic testing and adverse selection: Over the past years, the cost of genetic testing has declined significantly and, with direct-to-consumer (DTC) testing kits, genetic tests are now available and affordable for individual use. It has been widely adopted by public health systems and individuals. This has significant implications for life insurers, not least in respect to the regulatory constraints involved.

Appendix B: Terms and definitions

What is SONAR?

SONAR stands for Systematic Observation of Notions Associated with Risk. It is Swiss Re's tool for identifying, assessing and managing emerging risks. Experts across the company use a web-based platform to collect early signals of emerging risks. All signals are assessed and prioritised by an emerging risk management team which closely interacts with topic experts from Swiss Re's business areas. The team serves as a catalyst for risk identification and assessment to define and implement recommendations in collaboration with the business. The findings are regularly shared internally and summarised for external audiences here.

What are emerging risks?

We define emerging risks as newly developing or changing risks that are difficult to quantify and could have a major impact on society and industry.

What are emerging risk themes?

Emerging risk themes illustrate potential new or changing risk developments for the insurance industry. They are mainly derived from SONAR but also draw on other sources. All themes have been assessed and edited by Swiss Re's emerging risk management experts. This report only features new emerging risk themes (ie, topics covered in previous editions are not listed again). You can retrieve prior reports from our webpage: www.swissre.com/sonar

What is meant by overall impact?

The overall impact is an indicator of the potential financial, reputational and/or regulatory impact associated with an emerging risk topic. It is assessed on a scale from high to low:

HIGH	Potentially high financial, reputational and/or regulatory impact or significant stakeholder concern
MEDIUM	Potentially medium financial, reputational and/or regulatory impact or moderate stakeholder concern
LOW	Potentially low financial, reputational and/or regulatory impact, or low stakeholder concern

What is meant by time frame?

We divide themes into those likely to occur in less than 3 years and those likely to occur over a longer time horizon. This assessment should not be used as an indicator of when action is needed, as some themes likely to occur in the more distant future may, nonetheless, require immediate action to prepare.

What is meant by impact per business area?

Spider graph indicating the potential impact on major insurance business areas on a scale from 0 (= no impact) to 4 (=significant impact).

What are trend spotlights?

Boxes throughout the text provide selective spotlights on emerging trends which could become relevant for the re/insurance industry and its clients. The selection of topics is non-exhaustive, and descriptions are intended as food for thought and discussion starters rather than comprehensive reviews.

What are macro trends?

Swiss Re has identified a set of macro trends assumed to have a high impact on the re/insurance industry within the next 5 to ten years. The macro trends featured in this report have been selected independently through expert discussions and surveys. They provide context to the emerging risk insights from the SONAR process.

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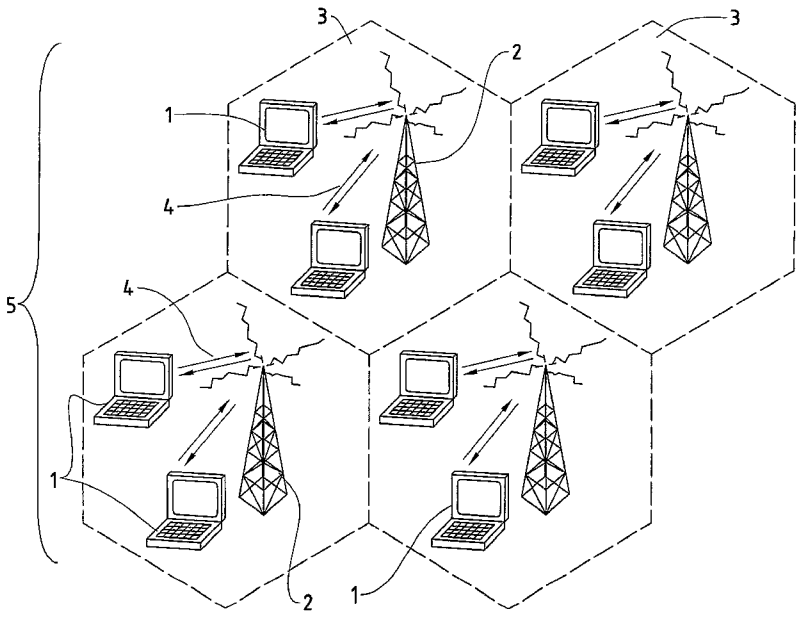
(54) Title: REDUCTION OF ELECTROSMOG IN WIRELESS LOCAL NETWORKS



(57) Abstract: A method and system for reduction of electrosmog in wireless local networks, one or more mobile network units (1) communicating with a base station (2) of a wireless local network (5). After a predefinable time interval without connecting signal, the base station (2) changes over from the normal transmitting-receiving mode into a sleep mode, in which sleep mode no beacon signals and/or other radio frequency signals are transmitted from the base station (2). If a mobile network unit (1) requires a network connection, it transmits an alert signal, and, upon receiving the alert signal of the mobile network unit (1), the base station transmits beacon signals to the mobile network unit (1) and changes over into the normal transmitting-receiving mode.

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Reduction of Electrosmog in Wireless Local Networks

This invention relates to a method and system for reduction of electrosmog in wireless local area networks (WLAN), one or more mobile network units communicating with a base station by means of radio frequency signals in a wireless local area network, which base station amplifies the radio frequency signals of the mobile network unit and/or connects the wireless local area network to a wired fixed network by means of bridge functions. In particular, the invention relates to a method and system in which a WLAN comprises a plurality of access points with differing transmission cells.

The influence of electrosmog on the human body is a known problem. The health risk from mobile radio transmitters, handys and DECT telephones has been an explosive subject among the general public at least since the enormous breakthrough in mobile radio technology in the 1990s. To meet the concerns of science from the legislative side, the permissible limit values have thus been lowered several times, and technology has been increasingly focused on this problem. The risk of damage to health through electrosmog has also become better understood as a result of more recent and improved studies. When, for example, human blood cells are irradiated with electromagnetic fields, clear damage to hereditary material has been demonstrated and there have been indications of an increased cancer risk (Mashevich M., Folkman D., Kesar A., Barbul A., Korenstein R., Jerby E., Avivi L., Department of Human Genetics and Molecular Medicine, Tel-Aviv University, Tel-Aviv, Israel, "Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability," *Bioelectromagnetics,* 2003 Feb., 24(2): 82-90). In this study, for example, human peripheral lymphocytes were exposed to continuous electromagnetic fields of 830 MHz in order to examine whether this leads to losses or gains in chromosomes (aneuploidy). Bigger changes lead to instability of the genome (= the totality of all genes of a germinal cell) and thereby to cancer. The human peripheral blood lymphocytes (PBL) were irradiated at different average specific absorption rates (SAR) of 1.6 to 8.8 W/kg over a time period of 72 hours in an exposure system based on a parallel plate resonator in a temperature range of 34.5 to 37.5 °C. The average absorption rate (SAR) and

its distribution in the exposed tissue culture flask were determined by combining the measurement results with a numerical analysis based on a finite element simulation code. A linear increase in the chromosome No. 17 -- an aneuploidy (=numerical chromosome aberration) -- was observed as a function of the SAR, demonstrating that this radiation has a genotoxic effect. The SAR-dependent aneuploidy was accompanied by an abnormal mode of replication of the chromosome 17 region engaged in segregation (repetitive DNA arrays associated with the centromere), suggesting that epigenetic alterations are involved in the SAR dependent genetic toxicity. Control experiments (i.e. without any radio frequency radiation) carried out in the temperature range of 34.5 to 38.5 °C showed that elevated temperature is not associated with either the genetic or epigenetic alterations observed following RF radiation, these alterations being the increased levels of aneuploidy and the modification in replication of the centromeric DNA arrays. These findings indicate that the genotoxic effect of electromagnetic radiation is elicited via a non-thermal pathway. Moreover aneuploidy is to be considered as a known phenomenon in the increase of cancer risk.

Thus it has been possible to show that mobile radio radiation can cause damage to genetic material, in particular in human white blood cells, whereby both the DNA itself is damaged and the number of chromosomes changed. This mutation can consequently lead to increased cancer risk. In particular, it could also be shown that this destruction is not dependent upon temperature increases, i.e. is non-thermal. Based on the scientific studies in the field, and owing to increasing pressure from the public, especially in the industrialized countries, epidemiological studies have been systematized by the World Health Organization (WHO) in the last few years, such as e.g. the currently running WHO Interphone Project, in order to be able to assess more precisely the health risks from electrosmog and work out corresponding guidelines.

Local networks (LAN: Local Area Network) usually consist of so-called nodes which are connected via physical media, such as e.g. coaxial cable, twisted pair or optical fiber cable. These LANs are also referred to as wired LANs (wired fixed networks). In the last few years wireless LANs have

also become more and more popular (e.g. through developments such as the AirPort System of Apple Computer, Inc.). Wireless LANs -- also referred to as WLANs -- are especially suitable for integrating mobile units (nodes), such as e.g. laptops, notebooks, PDAs (Personal Digital Assistants) or mobile radio devices, in particular mobile radio telephones, with a corresponding interface, into a local computer network. The mobile nodes have an adaptor comprising a transceiver as well as a control card (such as e.g. infrared (IR) adaptor or a low frequency radio wave adaptor). The advantage of such mobile nodes is that they can be moved freely within the range of the wireless LANs. The mobile nodes communicate either directly with one another (peer-to-peer wireless LAN) or send their signal to a base station which amplifies the signal and/or passes it on. The base stations can likewise comprise bridge functions. Via such base stations with bridge functions, so-called access points (AP), the mobile nodes can access the wireless LAN on a wired LAN. Typical network functions of an access point comprise the transmission of messages of one mobile node to another, the sending of messages from the wired LAN to a mobile node and the transmission of messages of a mobile node to the wired LAN.

There exist many different access methods for WLAN in the state of the art which make it possible for a user of a mobile network device to access a wireless local network. One of these access methods, such as e.g. Carrier Sense Multiple Access/Collision Detection (CSMA/CD) or token passing have proved to be highly successful in their industrial application. Today the use of local or wide area networks usually does not have any clearly defined, predetermined characteristics anymore. With the growth of heterogeneous multimedia data exchange (e.g. video data streams, etc.) via WLANs, the Quality of Service (QoS) parameter for a particular type of data exchange (or application) has become more and more important. Such parameters comprise, for example, the highest possible bandwidth, lowest possible delay, etc. For such accesses, new access methods in the asynchronous or synchronous networks have been developed and can be found in the state of the art.

Together with the growth of the WLAN and the standardization of the access methods and the physical layer specifications for WLANS, such as e.g.

the 802.X physical layer protocols and non- 802.X protocols (e.g. ATM: Asynchronous Transfer Mode Protocol), the security needs of users and service providers of such networks have also become greater and greater. Unambiguous network recognition as well as user identification and/or authentication thereby complement one another. Within a WLAN, an AP transmits so-called Service Set IDentifier (SSID) when a mobile network unit tries to integrate itself in the wireless network. An SSID is an unambiguous identification, 32 characters long, which is assigned to the header of data messages sent over the network, and serves as a password for the mobile network units. The SSID differs from one WLAN to another. That means that all APs and mobile network units of a particular WLAN must use the same SSID. A network unit which cannot support the unambiguous SSID will not be granted any network access via a base station or respectively an AP. As mentioned, in the 802.X network technology, such as e.g. the 802.11 network technology, the network units normally communicate via an access point (AP). In the infrastructure mode, mobile network units can either communicate with one another or with network components of a wired network. An AP with bridge functions, which is connected to a wired network and one or more other access points, is referred to as the Basic Service Set (BSS). Designated as the Extended Service Set (ESS) are a plurality of BSS, which form in each case a sub-network. WLANs are usually operated in the infrastructure mode in order to provide access to other services, such as e.g. file server, printer services and/or the worldwide backbone network (Internet). In the 802.X technology, an SSID concerns in each case a Basic Service Set. Thus a mobile unit can only have network access to a BSS if it supports the corresponding SSID. SSIDs are sometimes referred to as network names since the SSIDs unambiguously designate or identify a network.

The physical range of an AP is called the Basic Service Area (BSA). If a mobile node is located within the BSA of an AP, it can communicate with this AP if the AP is likewise within the signal range (Dynamic Service Area (DSA)) of the mobile node. Mobile nodes typically have a signal strength of 100 mwatt to one watt. To connect the wireless LAN to the wired LAN, it is important for the AP to determine whether a particular message (information frame) on the network is intended for a node which lies within the wired LAN or within the

wireless LAN, and to pass on this information, if necessary, to the corresponding node. For this purpose APs have so-called bridge functions, e.g. corresponding to the standard IEEE Std 802.1D-1990 "Media Access Control Bridge" (31-74 ff). With such bridge functions, a new mobile node is registered in the wireless LAN, typically in a FDB (Filtering Database) of the AP in whose range the node is located. With each information frame on the LAN, the AP compares the destination address with the addresses (MAC addresses (Media Access Control Addresses)) which it has stored in the FDB, and sends, rejects or passes on the frame to the wired LAN or to the wireless LAN. The range of a wireless LAN is limited by factors such as e.g. wavelength of the signal, signal strength, impediments, etc. The radio frequency parameters cannot be selected freely, however. In most countries there are regulations, more or less strict, as mentioned further above, as concerns the low frequency transmission for wireless LANs (e.g. USA (FCC), Switzerland (BAKOM), etc.). This applies in particular to the USA, for example. In the USA the regulations are issued by the United States Federal Communications Commission (FCC) (D 15, Title 47, Code of Federal Regulations 1985). Three bandwidths are permitted: 902-928 MHz, 2400-2483.5 MHz and 5725-5850 MHz. Many applications today use the 900 MHz band. The quantity of data which can be transmitted over the 900 MHz band is limited, however, by the narrow frequency bandwidth in this band. Therefore more and more applications are using the frequency band around 2400 MHz. Future applications will presumably also use the band around 5800 MHz in order to meet the growing demand for high data throughput.

Despite increasingly strict national guidelines with respect to legally specified limits, the impact of electrosmog in WLANs on the human body can be considerable. Moreover it is to be expected that this impact will continue to increase in the future for many people. Two factors in particular are playing a role in this: First, more and more applications require additional, usually higher-energy frequency bands in order to be able to meet the growing need with respect to transmission rate. Second, the need for WLAN expansion in the private sphere as well as in the public sphere, e.g. in airports, railway stations, trains, restaurants, exhibition halls, etc., has by far not yet reached its peak. With the state of the art as a basis, there has been a lot of effort put into providing evidence for the detrimental effects of electrosmog and setting

corresponding limits. Limits and guidelines alone will not suffice, however, to further contain the electrosmog in WLANs since the development in WLANs runs in exactly the opposite direction, as mentioned above. WLANs even represent zones in which people usually spend longer periods of time (place of work, Internet, network games, etc.) and are therefore to be considered as particularly problematic with respect to radiation impact. WLANs in the state of the art moreover send base stations, such as access points, so-called beacon signals periodically so that mobile units can recognize the network and authenticate themselves with an access point. These beacon signals comprise recognition signals, such as e.g. SSIDs and/or other radio frequency signals with control parameters. Even if no mobile units are located in the WLAN, the beacon signals continue to be transmitted periodically to the APs. This means that even when the WLAN is not being used at all, an underlying stress from electromagnetic radiation remains for persons in the Basic Service Area of an access point of the WLAN. For example, in the case of WLANs at places of employment, such as offices, etc., there exists therefore permanent stress from electrosmog from the WLAN on the employees of the company or organization. In the state of the art there exists only the possibility of further reducing the limits for electromagnetic radiation.

It is an object of this invention to propose a new method and system for reducing electrosmog in wireless local networks which do not have the drawbacks described above. In particular a solution should be proposed which can be managed without any disruptive software and/or hardware adaptations and is thus easily achievable for existing WLAN technologies.

These objects are achieved, according to the present invention, in particular through the elements of the independent claims. Further preferred embodiments follow moreover from the dependent claims and from the description.

In particular, these objects are achieved through the invention in that, for reducing electrosmog in wireless local area networks (WLANs), one or more mobile network units communicate with a base station in a wireless local network by means of radio frequency signals, which base station amplifies the

radio frequency signals of the mobile network unit and/or connects the wireless local area network to a wired fixed network by means of bridge functions, the base station changes over from the normal transmitting-receiving mode into a sleep mode after a predefinable time interval without connecting signal to a mobile network unit, in the sleep mode no recognition signals and/or other radio frequency signals being transmitted from the base station, the base station being ready to receive radio frequency signals, however, when needing a network connection, a mobile network unit transmits an alert signal to the base station, and upon receiving the alert signal of the mobile network unit, the base station transmits to the mobile network unit the recognition signals necessary for the connection and changes over into the normal transmitting and receiving mode. The invention as described above has the advantage that electrosmog in WLANs can be greatly reduced during times when there is no network activity. At the same time energy consumption is also reduced since in sleep mode no beacon signals or other radio frequency signals are transmitted from the base stations. The whole method and system is achievable in particular without any hardware changes of any kind in the mobile network unit being necessary on the user side, nor on the side of the base stations, and it is therefore simpler and less expensive to achieve compared with other solutions. This means that not only are the costs for new hardware saved, but also the costs for installing it. It must also be pointed out that in mobile network units weight and space considerations often play a role too. The present invention requires neither additional hardware space, nor does it result in increased weight of the mobile terminal (network unit). For company-internal WLANs, for example, it also further increases security, making it more difficult for the WLAN to be used by unauthorized persons e.g. outside of business hours since no periodic beacon signal is sent anymore by the base station or base stations if they are in sleep mode.

In an embodiment variant, when in need of a network connection, the mobile network unit transmits an alert signal only if it does not receive any recognition signal from a base station. This embodiment variant has the advantage, among other things, that no unnecessary alert signal has to be transmitted if the base station is already in normal transmitting-receiving mode.

This likewise results in a further reduction of electrosmog and at the same time energy saving in the mobile network units.

In another embodiment variant, only the base station in whose basic service area (BSA) the mobile network unit is located changes over into the normal transmitting and receiving mode, the other base stations of the wireless local network remaining in their previous operating mode. This embodiment variant has the advantage, among other things, that the electrosmog can be further reduced since for mobile units which are at times stationary, such as e.g. when working with a laptop at one's place of employment, only the needed base station goes back into the normal transmitting-receiving mode.

In still another embodiment variant, the base stations of the basic service areas (BSAs) bordering on the basic service area (BSA) of the base station in whose BSA the mobile network unit is located likewise change over automatically into the normal transmitting-receiving mode if they were previously in the sleep mode. This embodiment has, among other things, the same advantages as the preceding one, but during a shift of the mobile network unit from one BSA to the next, the base station of the bordering BSA is already in the normal transmitting and receiving mode.

In an embodiment variant, the base station of the wireless local network changes over from sleep mode into the normal transmitting-receiving mode only if a network-specific recognition signal of the alert signal corresponds to a stored recognition signal of the wireless local network. This embodiment has the advantage, among other things, that the user as well as the service provider of the WLAN is given additional security. Through the additional authentication by means of a network-specific recognition signal, an unauthorized person, such as someone outside the company in the case of company WLANs, cannot even activate the normal transmitting and receiving mode of the WLAN or respectively of the base station.

In an embodiment variant, at least parts of the network-specific recognition signal, such as e.g. supplementary information data, are definable for the wireless local network by a user of the mobile unit and/or by an operator.

This embodiment variant has, among other things, the same advantages as the preceding embodiment variant. The security can be further increased however through the addition of supplementary information data determinable by the user or operator. In an embodiment variant, these data can even be supplementary information data freely chosen by the user, whereby, as a borderline case, the supplementary information data could even be empty. As further embodiment variants, an unambiguous identification code of the user can be used as the supplementary information data. For example, this can be an IMSI (International Mobile Subscriber Identification) and/or a MSISDN (Mobile Subscriber ISDN) which is stored on a SIM (Subscriber Identification Module) card of the mobile network unit. This has the advantage, among other things, that a particular user can be identified by means of the MSISDN, and, if required, can be correspondingly authenticated, e.g. with a log-in password, etc., without the user having to be registered beforehand in the system, e.g. in a database. As an additional embodiment, it is even conceivable for the MSISDN of a mobile radio device of the user to be used as the MSISDN, for example, the mobile radio device being one from which an access request was previously sent to a central unit.

In a further embodiment variant, the alert signal is transmitted from the mobile unit in a network-independent way for each wireless local network. This embodiment variant has the advantage, among other things, that any mobile network unit can activate possibly available WLANs in a standard way, independently of a specific recognition signal, or at least can receive a beacon signal or similar signal of the network.

In another embodiment variant, the wireless local network is set up based on the 802.X network technology, the recognition signals containing the corresponding Service Set Identifiers (SSID). This embodiment variant has the advantage, among other things, that a standardized access method and standardized physical layer specifications with the 802.X layer protocols can be used for the WLANs. This allows a cost-effective implementation without it being necessary to depart from the standard methods. At the present time the standards of the Institute of Electrical and Electronics Engineers (IEEE) have taken hold worldwide in the WLAN area. Among the IEEE standards which

have gained acceptance are in particular the IEEE 802 standards for LAN (Local Area Network) technologies.

In another embodiment variant, the wireless local network is set up based on Bluetooth technology. Among other things, this embodiment variant has the same advantages as the preceding one. In particular, Bluetooth is supported by a wide range of well-known hardware and software producers, such as e.g. Ericsson, IBM, Intel, Nokia, Toshiba, etc., which are themselves members of the Bluetooth Special Interest Group, which defines the Bluetooth standard.

Embodiment variants of the present invention will be described in the following with reference to examples. The examples of the embodiments are illustrated by the following attached figures:

Figure 1 shows a block diagram illustrating schematically the architecture of an embodiment variant of a method and/or system according to the invention for reducing electrosmog in wireless local networks 5, one or more mobile network units 1 communicating by means of radio frequency signals 4 with a base station 2 of a wireless local network 5, which base station 2 amplifies the radio frequency signals 4 of the mobile network unit 1 and/or connects the wireless local network 5 to a wired fixed network by means of bridge functions.

Figure 2 shows a flow chart presenting schematically the architecture of a method and/or system in a wireless local network 5, whereby a beacon signal is constantly being transmitted from the base stations 2 in order to make a potential user aware of the availability of a WLAN 5.

Figure 3 shows a flow chart presenting schematically the architecture of a method and/or system according to the invention in a wireless local network 5, the WLAN 5 having two different operating modes, such as a normal transmitting - receiving mode and a sleep mode. The figure shows in particular the course of switchover from the sleep mode into the normal transmitting -

receiving mode when a mobile network unit 1 would like to use the wireless local network 5.

Figure 1 illustrates an architecture which can be used to achieve the invention. In this embodiment example, one or more mobile network units 1 communicate by means of radio frequency signals 4 with a base station 2, or respectively an access point, of a wireless local network 5. Wireless local networks 5 are also referred to as WLANs (Wireless Local Area Networks). A WLAN can be composed of one or more such base stations or respectively access points. The base station 2 amplifies the radio frequency signals 4 of the mobile network unit 1 and/or connects the wireless local network 5 by means of bridge functions to a wired fixed network. Base stations 5, or respectively access points, of a WLAN 5 can be connected e.g. via physical media such as, for instance, coaxial cable, twisted pair or fiber optic cable to assigned radius servers. The connection can comprise communication networks, such as, for example, mobile radio networks, such as a terrestrial mobile radio network, e.g. a GSM or UMTS network, or a satellite-based mobile radio network and/or one or more fixed networks, for instance the public switched telephone network (PSTN) and/or ISDN (Integrated Services Digital Network) or a suitable LAN (Local Area Network) or WAN (Wide Area Network). During log on of a mobile network unit 1 of a user in a WLAN 5, an identification code of the user is transmitted for authentication of the user together with supplementary information data, which can be determined by the user, via one of the APs 2 of the WLAN 5 to a central unit and/or radius server. The communication between the central unit and the access points 2 can take place e.g. via a TCP/IP interface and/or CORBA interface, an ATM module, a SMS and/or USSD gateway by means of special short messages, for example SMS (Short Message Services), USSD (Unstructured Supplementary Services Data) messages, or other techniques such as MExE (Mobile Execution Environment), via protocols such as GPRS (Generalized Packet Radio Service), WAP (Wireless Application Protocol) or another user information channel. The data transfer between the central unit and the access points 2 is initiated and carried out e.g. via transfer modules, implemented through software or hardware, of the central unit as well as of the access points. The mobile network units 1 or so-called mobile nodes can be e.g. laptops, notebooks, PDAs (Personal Digital

Assistants) or mobile radio devices, in particular mobile radio telephones. The mobile nodes are equipped through hardware and software with a corresponding interface in order to integrate them in a local wireless computer network (WLAN). They communicate by means of radio frequency signals with the access points 2 of the WLAN 5. The mobile nodes 1 can comprise e.g. an adaptor, which includes a transceiver as well as a control card (such as e.g. an infrared (IR) adaptor or a low frequency radio wave adaptor). The mobile nodes 1 are thereby able to move freely within the range of the wireless LAN 5. The access points 2 of the WLAN 5 can e.g. amplify the radio frequency signals of the mobile node 1 as well as comprise bridge functions which make it possible to access nodes 1 of a wired LAN from the wireless local network 5 and vice-versa. For transmission of the radio frequency signals, the access points 2 comprise at least one antenna. The antenna can be e.g. a dipole antenna, a loop radiator such as a folded dipole, a Marconi aerial or a ground plane antenna, a directional antenna such as e.g. a yagi aerial, a turnstile antenna or a parabolic aerial, an omnidirectional antenna or a fractal antenna system. The radio frequency signals lie typically in the frequency bands reserved for wireless LAN between 800 MHz and 6000 MHz, such as e.g. three frequency bands set by the United States Federal Communication Commission (FCC) in the USA: 902-928 MHz, 2400-2483.5 MHz and 5725-5850 MHz (D 15 of Title 47, Code of Federal Regulations). They can also be in the range of 400 MHz, for example, as is common e.g. with electronic, wireless garage openers, or at the WLL (Wireless Local Loop) frequencies auctioned a year ago in Germany and Switzerland, e.g. 26 GHz for wireless local loop methods. It is to be pointed out, however, that other frequencies are also possible, without affecting the scope of the invention. Thus, in principle, infrared signals can also be used for the invention such as e.g. IrDA, IR-LAN, etc. The bridge functions of the base station 2 can be achieved e.g. according to IEEE standard 802.1D-1990 "Media Access Control Bridges" pp. 31-47. In the WLAN network recognition and user identification and/or authentication complement one another. For network recognition, an AP periodically transmits so-called beacon signals within a WLAN, which signals comprise e.g. Service Set IDentifiers (SSID) and/or other control parameters for integrating a mobile network unit 1 into a wireless network. This applies in particular to the 802.X, such as e.g. the 802.11 network technologies, but also to Bluetooth and other network technologies. Beacon

signals are thus transmitted all the time to make potential users or respectively their mobile network units 1 aware of available WLANs 5. In the present invention, however, after a predefined time interval without a connection signal to a mobile network unit 1, the base station 2 switches over from normal transmitting and receiving mode to sleep mode. Understood by "normal transmitting and receiving mode" is the normal operating mode of the AP during which mobile network units 1 can access the APs or not.

In a flow chart, Figure 2 illustrates how a mobile network unit 1 recognizes the WLAN and connects thereto before the user can authenticate himself e.g. with the central unit and/or radius server. As mentioned, the base station in normal transmitting and receiving mode transmits beacon signals periodically 11. Even when no mobile network units are located in the WLAN, the beacon signals continue to be periodically transmitted from the APs. The SSID can be an unambiguous identification symbol, 32 characters long, which is assigned to the header of data messages sent over the network and which serves as a password for the mobile network units. The SSID differs from one WLAN to another. That means that all APs and mobile network units of a particular WLAN must use the same SSID. A network unit which cannot support an unambiguous SSID will normally not be granted any network access via a base station or respectively an AP. In the secure access mode (802.X) of the APs, the SSID from base station 2 and mobile network unit 1 must agree. In the non-secure access mode, a mobile network unit 1 can log on with the configured SSID, a blank SSID, or with the SSID set on "any." The beacon signals can be transmitted encrypted or unencrypted. The 802.11 network standard uses for encryption purposes WEP (Wired Equivalent Privacy), for example. WEP operates in three modes: no encryption, 40-bit encryption and 128-bit encryption. The 802.11 standard encrypts only the data packets, however, and not the management packets. The SSID is part of the beacon and probe management signal and is not encrypted when WEP is activated. A mobile network unit 1 receives the beacon signal 13, and recognizes the WLAN 5 from the beacon. Default SSIDs of WLANs are e.g. “tsunami” - Cisco, “101” – 3Com, “RoamAbout Default Network Name” - Lucent/Cabletron, “Default SSID", “Compaq” - Compaq, “WLAN” – Addtron (a popular AP), “intel” - Intel, “linksys” – Linksys, “Wireless”. Thus if a mobile network unit 1 receives a

beacon signal 13, it logs on with the corresponding AP, and carries out the authentication 14 of the user, if necessary, e.g. with the central unit, before it has access to the WLAN 5. If the mobile node 1 does not receive any beacon signal, but nevertheless needs a WLAN connection, it continues to scan for beacon signals 15 until it has found an available WLAN. This applies to the normal transmitting and receiving mode. In the normal transmitting and receiving mode the AP automatically transmits a further beacon signal after a predefined time interval 12. In the case that a base station 2 switches over into sleep mode, no recognition signals and/or other radio frequency signals are transmitted anymore from the base station 2, i.e. also no beacon signals, but the base station 2 nevertheless remains ready to receive radio frequency signals 4 also in sleep mode.

Figure 3 illustrates the method according to the invention on the side of the AP 2 when the base station 2 is in sleep mode. If a mobile network unit 1 needs a network connection, it transmits an alert signal which is received by the base station 2. If, in the normal transmitting and receiving mode, the base station does not receive any connection signal from a mobile network unit 1, the AP 2 waits for a predefinable period of time 24, if thereafter it still does not receive any connection signal 25, the base station 2 switches over into sleep mode 26, and waits 27 for a connection signal from a mobile node 1. Upon receiving an alert signal from a mobile network unit 1, the base station 2 transmits 22 the recognition signals necessary for the connection and/or beacon signals to the mobile network unit 1 (e.g. beacon signal), and, as described under Figure 2, carries out the authentication of the user of the mobile network unit 1. All base stations 2 of a WLAN 5 can always switch together from sleep mode into the normal transmitting and receiving mode, or only those base stations 2 in whose basic service areas 3 the mobile network unit 1 is located, the other base stations 2 of the wireless local network 5 remaining in their previous operating mode. It can make sense in addition for the base stations 2 of basic service areas 3 bordering on the basic service areas 3 of the base station 2 in whose BSA the mobile node 1 is located to automatically switch over into the normal transmitting and receiving mode if they were previously in sleep mode. In an embodiment variant, the mobile network unit 1, when needing a network connection, can transmit an alert signal

only when no recognition signal is received from a base station 2, or automatically every time it needs a WLAN, for example. It is furthermore possible for the base station 2 of the wireless local network 5 to switch over from sleep mode into the normal transmitting-receiving mode only when a network-specific recognition of the alert signal corresponds with a stored recognition signal of the wireless local network 5. This results in additional protection against unauthorized use of the WLAN. The security of the WLAN 5 can be further increased in that at least parts of the network-specific recognition signal are definable for the wireless local network 5 by the user of the mobile unit 1 and/or by an operator. As a special embodiment variant, the MSISDN and/or IMSI of a mobile radio device of the user of the mobile network unit 1 can be used as the supplementary information data. Moreover this can be stored on a SIM (Subscriber Identification Module) card of the mobile network unit. For other embodiments it can be important, however, that the alert signal is transmitted from the mobile network unit 1 in a network-independent way. This could be advantageous in particular for WLANs in public buildings, airports, etc. It is important to point out that the method or respectively system according to the invention can be achieved without modification of existing hardware on the side of the base stations 1 and on the side of the mobile network units 1, requiring only modification of the corresponding software components. Of course it is also possible to achieve the method and system according to the invention through addition of corresponding hardware modules.

Claims

1. A method for reducing electrosmog in wireless local networks, one or more mobile network units (1) communicating with a base station (2) of a wireless local network (5) by means of radio frequency signals (4), which base station (2) amplifies the radio frequency signals (4) of the mobile network unit (1) and/or connects the wireless local network (5) to a wired fixed network by means of bridge functions, wherein

the base station (2) changes over from the normal transmitting-receiving mode into a sleep mode after a predefinable time interval without connecting signal to a mobile network unit (1), in the sleep mode no recognition signals and/or other radio frequency signals being transmitted from the base station (2), the base station being ready to receive radio frequency signals (4), however,

when needing a network connection, a mobile network unit (1) transmits an alert signal to the base station,

upon receiving the alert signal of the mobile network unit (1), the base station (2) transmits to the mobile network unit (1) the recognition signals necessary for the connection and changes over into transmitting and receiving mode.

2. The method according to claim 1, wherein, when in need of a network connection, the mobile network unit (1) transmits an alert signal only if it does not receive any recognition signal from a base station (2).

3. The method according to one of the claims 1 or 2, wherein only the base station in whose basic service area the mobile network unit (1) is located changes over into the normal transmitting and receiving mode, the other base stations (2) of the wireless local network (5) remaining in their previous operating mode.

4. The method according to claim 3, wherein the base stations (2) of the basic service areas (3) bordering on the basic service area (3) of the base station (2) in whose basic service area the mobile network unit (1) is located likewise change over automatically into the normal transmitting-receiving mode if they were previously in the sleep mode.

5. The method according to one of the claims 1 to 4, wherein the base station (2) of the wireless local network (5) changes over from sleep mode into the normal transmitting-receiving mode only if a network-specific recognition signal of the alert signal corresponds to a stored recognition signal of the wireless local network (5).

6. The method according to claim 5, wherein at least parts of the network-specific recognition signal are definable for the wireless local network (5) by a user of the mobile unit (1) and/or by an operator.

7. The method according to one of the claims 1 to 6, wherein the alert signal from the mobile network unit (1) is transmitted in a network independent way for every wireless local network (5).

8. The method according to one of the claims 1 to 7, wherein the wireless local network (5) is set up based on the 802.X network technology, the recognition signals containing the respective Service Set Identifier (SSID).

9. The method according to one of the claims 1 to 7, wherein the wireless local network (5) is set up based on Bluetooth technology.

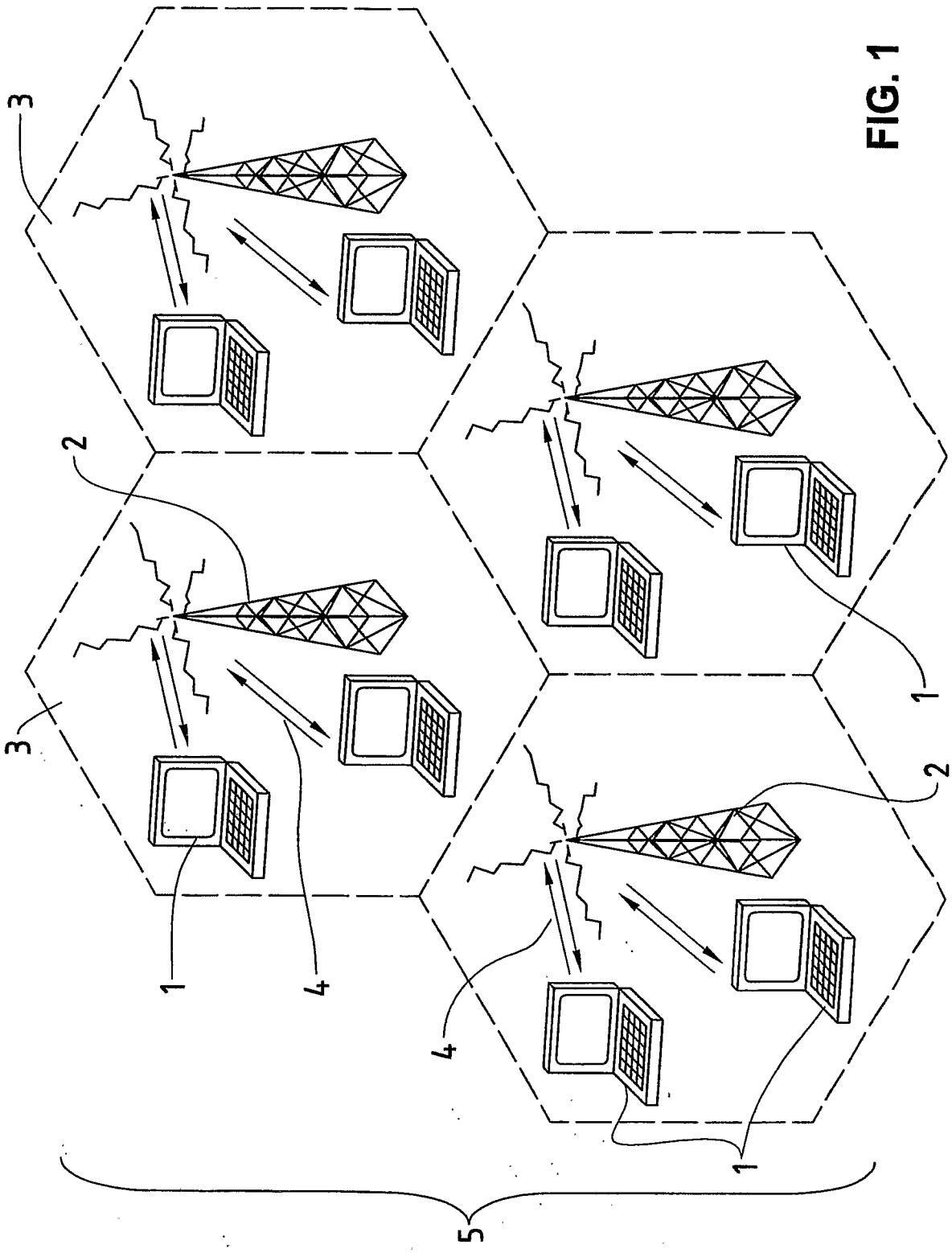

FIG. 1

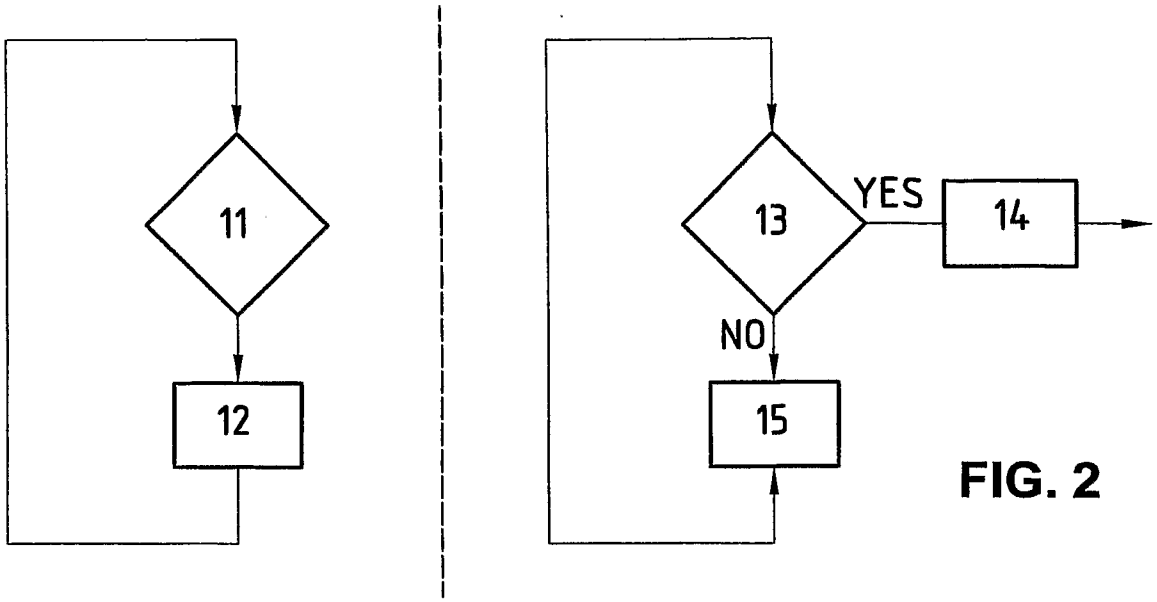

FIG. 2

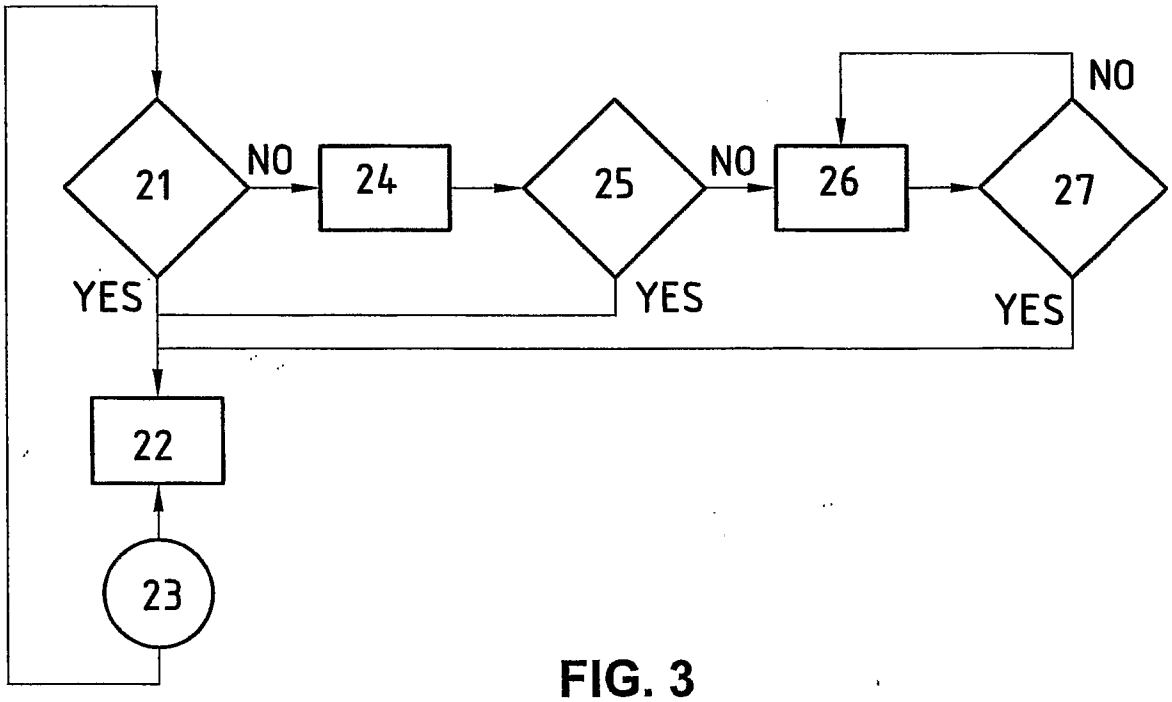

FIG. 3

INTERNATIONAL SEARCH REPORT

International Application No
PCT/CH 03/00138

A. CLASSIFICATION OF SUBJECT MATTER
IPC 7 H04Q7/32 H04Q7/30

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)
IPC 7 H04Q H04B

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practical, search terms used)
EPO-Internal, WPI Data

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category °	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
A	US 5 884 196 A (LEKVEN ERIC J ET AL) 16 March 1999 (1999-03-16) abstract figure 2 column 6, line 11 - line 31	1-9
A	WO 02 093778 A (QUALCOMM INC) 21 November 2002 (2002-11-21) abstract paragraph '0009! - paragraph '0010! claim 1	1-9
A	US 6 339 694 B1 (NUCKOLS JEFFREY R ET AL) 15 January 2002 (2002-01-15) column 3, line 43 - line 60 abstract	1-9

-/--

[X] Further documents are listed in the continuation of box C.

[X] Patent family members are listed in annex.

° Special categories of cited documents :

"A" document defining the general state of the art which is not considered to be of particular relevance

"E" earlier document but published on or after the international filing date

"L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)

"O" document referring to an oral disclosure, use, exhibition or other means

"P" document published prior to the international filing date but later than the priority date claimed

"T" later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention

"X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone

"Y" document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art.

"&" document member of the same patent family

Date of the actual completion of the international search	Date of mailing of the international search report
14 October 2003	22/10/2003

Name and mailing address of the ISA	Authorized officer
European Patent Office, P.B. 5818 Patentlaan 2 NL – 2280 HV Rijswijk Tel. (+31–70) 340–2040, Tx. 31 651 epo nl, Fax: (+31–70) 340–3016	Dionisi, M

Form PCT/ISA/210 (second sheet) (July 1992)

INTERNATIONAL SEARCH REPORT

International Application No
PCT/CH 03/00138

C.(Continuation) DOCUMENTS CONSIDERED TO BE RELEVANT

Category °	Citation of document, with indication,where appropriate, of the relevant passages	Relevant to claim No.
A	WO 02 07464 A (ERICSSON TELEFON AB L M) 24 January 2002 (2002-01-24) page 2, line 5 - line 23 page 15, line 11 - line 15	1-9

INTERNATIONAL SEARCH REPORT

Information on patent family members

International Application No
PCT/CH 03/00138

Patent document cited in search report	Publication date	Patent family member(s)	Publication date
US 5884196 A	16-03-1999	AU 717244 B2	23-03-2000
		AU 3569397 A	05-01-1998
		BR 9709555 A	11-01-2000
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		WO 9747149 A2	11-12-1997
WO 02093778 A	21-11-2002	US 2002177461 A1	28-11-2002
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		WO 02093954 A1	21-11-2002
		WO 02093948 A1	21-11-2002
		WO 02093812 A2	21-11-2002
		WO 02093778 A1	21-11-2002
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WO 0207464 A	24-01-2002	US 6584330 B1	24-06-2003
		AU 7121601 A	30-01-2002
		WO 0207464 A1	24-01-2002

Toprådgiver i Holland advarer mod ukritisk 5G-udrulning

Author: Tabt Tråd

NYHED / UDLAND: Formanden for strålingskomitéen i Hollands nationale sundhedsråd, Hans Kromhout, advarer nu landets beslutningstagere mod ukritisk 5G-udrulning.

Den nederlandske professor mener, at alle herlighederne ved 5G skal holdes nøje op imod de risici for folkesundheden, som samfundet løber.

Det skriver det nederlandske dagblad, De Telegraaf.

Hans Kromhout mener ikke, at forsigtigheden med mobilstråling lever op til de standarder, man anvender til at beskytte befolkningen mod andre risici som kemikalier, pesticider og medicin, hvor kravene ifølge Hans Kromhout er strenge.

Hans Kromhout kritiserer især, at autoriteterne på strålingsområdet ignorerer klare forskningsresultater som det amerikanske NTP-projekt, der varede i 19 år og som sluttede med en klar konklusion af kræftfund i forsøgsrotter.

"Den amerikanske NTP-undersøgelse, som leverede klare beviser for sammenhæng mellem kræft og eksponering for EMR (red. elektromagnetisk stråling) – var godt udført og repræsenterede et stort gennembrud. Alligevel forsøger visse grupper at

bortforklare", siger han til avisen.

Mistillid til ICNIRP

Hans Kromhout kritiserer, at en lang række lande ret ukritisk adopterer deres grænseværdier fra det private forskerselskab ICNIRP og kalder det "ganske usædvanligt", at et selskab som ICNIRP kan opnå så stor en indflydelse.

"Det er lidt af en uigennemsigtig klub, hvor det er uklart, hvordan medlemmerne vælges. Kald det som selvbestaltet. I den forstand kan man ikke tillægge organisationen en uafhængig status", siger han til avisen.

To af verdens nøgleposter på området er besat af hollændere. I ICNIRP er nederlandske Eric van Rongen formand, indtil han træder et skridt tilbage og bliver næstformand i maj 2020.

Siden 2006 har nederlandske Emilie van Deventer været leder af WHO-kontoret The International EMF Project i Geneve, som siden oprettelsen i 1996 har arbejdet meget tæt med ICNIRP.

ICNIRP's hovedstifter og første formand, Michael Repacholi, forlod ICNIRP i 1996 for at blive leder i det nyoprettede WHO-kontor, som i betydelig grad har fungeret for teleindustriens sponsormidler.

REPUBBLICA ITALIANA

IN NOME DEL POPOLO ITALIANO

LA CORTE D'APPELLO DI TORINO

SEZIONE LAVORO

Composta da:

Dott.ssa Rita MANCUSO	PRESIDENTE
Dott.ssa Caterina BAISI	CONSIGLIERE
Dott.ssa Silvia CASARINO	CONSIGLIERE Rel.

ha pronunciato la seguente

S E N T E N Z A

nella causa di lavoro iscritta al n. **721/2017** R.G.L. promossa da:

ISTITUTO NAZIONALE PER L'ASSICURAZIONE CONTRO GLI INFORTUNI SUL LAVORO – I.N.A.I.L. -, con sede in Roma, Via IV Novembre n. 144, in persona del Direttore Regionale pro-tempore del Piemonte, rappresentato e difeso per procura generale alle liti Notaio Romano di Chivasso del 07.08.2013 rep. N. 55082 Raccolta 16699 dagli Avv.ti Loretta Clerico ed Elia Pagliarulo, ed elettivamente domiciliato in Torino, Corso Galileo Ferraris n. 1 presso l'Avvocatura Regionale INAIL

APPELLANTE

CONTRO

ROMEO ROBERTO, residente a Leinì (TO), Via Lamarmora

n. 11, rappresentato e difeso per procura in calce al ricorso introduttivo del giudizio di primo grado, congiuntamente e disgiuntamente, dagli Avv.ti Renato Ambrosio, Stefano Bertone e Chiara Ghibaudo, ed elettivamente domiciliato presso il loro studio in Torino, Via Bertola n. 2

APPELLATO

oggetto: malattia professionale

CONCLUSIONI

Per l'appellante:

come da ricorso depositato in data 31.8.2017

Per l'appellato:

come da memoria difensiva depositata in data 22.10.2018

FATTI DI CAUSA

Il sig. Roberto Romeo ha chiamato l'INAIL davanti al Tribunale di Ivrea, deducendo la natura professionale del neurinoma dell'acustico destro di cui è affetto, in quanto patologia contratta per l'uso abnorme di telefoni cellulari nel periodo 1995-2010, in cui ha lavorato alle dipendenze di Telecom s.p.a., e chiedendo quindi la condanna dell'Istituto convenuto a pagargli la prestazione dovuta per legge, commisurata alla percentuale di invalidità, indicata in misura pari ad almeno il 37%.

L'INAIL ha contestato la domanda attorea e ne ha chiesto il rigetto.

Istruita la causa mediante escussione di alcuni testimoni e con due c.t.u. medico-legali (una sul nesso causale e l'altra sulla quantificazione dei postumi permanenti), con sentenza n.

96/2017 pubblicata il 21.4.2017, il Tribunale, in accoglimento del ricorso, ha condannato l'INAIL a corrispondere al ricorrente la prestazione spettante con riferimento alla percentuale di invalidità del 23%, con condanna a rimborsare al ricorrente le spese di lite e a pagare le spese di c.t.u..

Propone appello l'INAIL; resiste l'appellato.

Disposta nuova c.t.u. medico-legale (affidata congiuntamente alla dott.ssa Carolina Marino e al dott. Angelo D'Errico, specialisti rispettivamente la prima in medicina-legale e il secondo in medicina del lavoro, dirigente medico del Servizio Sovrazonale di Edipemiologia ASL TO3), all'udienza del 3.12.2019, all'esito della discussione, la Corte ha deciso la causa come da separato dispositivo.

RAGIONI DELLA DECISIONE

Il Tribunale ha accolto il ricorso osservando che:

-il ricorrente, quale referente/coordinatore di altri dipendenti Telecom, ha utilizzato in maniera abnorme telefoni cellulari nel periodo 1995-2010, come dimostrato dall'istruttoria testimoniale (testi Musso, Nani, Bilucaglia);

-in base ad essa si deve infatti ritenere che il ricorrente, coordinando una quindicina di colleghi, nell'ipotesi più prudente utilizzasse con loro il telefono per almeno due ore e mezza al giorno (2 telefonate x 5 minuti x 15 colleghi), e che, nell'ipotesi maggiore, le ore al telefono diventassero oltre sette (3 telefonate x 10 minuti x 15 colleghi), a cui si aggiunge il tempo trascorso al telefono per riferire ai propri superiori e per coordinarsi con il

direttore dei lavori degli enti e con le imprese esterne che collaboravano nei lavori, nonché durante il fine settimana, come confermato dal teste Romeo, figlio del ricorrente;

-inoltre, all'epoca non esistevano strumenti per attenuare l'esposizione alle radiofrequenze e questa era aggravata dal tipo di tecnologia utilizzata per i primi telefoni cellulari (tecnologia ETACS), e dal fatto che spesso l'utilizzo avveniva all'interno dell'abitacolo di un'autovettura;

-la letteratura scientifica è divisa in merito alle conseguenze nocive dell'uso dei telefoni cellulari: da una parte l'Agenzia Internazionale per la Ricerca sul Cancro (IARC), facente parte dell'Organizzazione Mondiale della Sanità (ente imparziale ed autorevole a livello mondiale) il 31.5.2011 ha reso nota una valutazione dell'esposizione a campi elettromagnetici ad alta frequenza, definendoli come "cancerogeni possibili per l'uomo" (categoria 2B); dall'altra lo studio Interphone individua un rischio del 40% superiore per i glioma (famiglia di tumori cui appartiene anche quello che ha colpito il ricorrente) negli individui che abbiano usato il cellulare molto a lungo e per molto tempo; gli unici studiosi che con fermezza escludono qualsiasi nesso causale tra utilizzo di cellulari e tumori encefalici sono i proff. Ahlbom e Repacholi, ma detti autori si trovano in posizione di conflitto di interessi, essendo il primo consulente di gestori di telefonia cellulare ed il secondo di industrie elettriche;

-ai risultati a cui sono pervenuti gli studi finanziati dalle aziende produttrici di telefoni cellulari non può essere attribuita

particolare attendibilità in considerazione della posizione di conflitto di interessi degli autori, come ritenuto dalla S.C. nella sentenza n. 17438/2012 in un caso relativo ad altro tumore encefalico (neurinoma del ganglio di Gasser);

-la c.t.u. ha accertato la sussistenza del nesso causale;

-pertanto, e considerate le peculiarità del caso concreto (associazione tra tumore raro ed esposizione rara per durata ed intensità; periodo di latenza congruo con i valori relativi ai tumori non epiteliali; il fatto che la patologia sia insorta nella parte destra del capo del ricorrente, soggetto destrimane; mancanza di altra plausibile spiegazione della malattia), deve ritenersi provato un nesso causale, o quantomeno concausale, tra tecnopatia ed esposizione, sulla base della regola del "più probabile che non";

-i postumi permanenti debbono essere riconosciuti nella misura del 23%, come da conclusioni del c.t.u., non contestate da alcuna delle parti.

Con il primo motivo di gravame l'INAIL lamenta che il Tribunale abbia omesso di pronunciarsi sull'eccezione di inammissibilità del ricorso, ai sensi dell'art. 152 disp. att. c.p.c., per mancanza della dichiarazione di valore della prestazione richiesta.

Il motivo è infondato, avendo la Corte Costituzionale, con sentenza 20.11.2017 n. 241, dichiarato l'incostituzionalità di detta norma.

Con il secondo motivo, l'Istituto sostiene che il Tribunale abbia erroneamente ritenuto provato un uso abnorme per 15 anni del telefono cellulare per esigenze lavorative, essendo le testimonianze sul punto contraddittorie: in particolare, secondo la deposizione del teste Bilucaglia la durata delle telefonate (e quindi l'esposizione alle radiofrequenze) era di un'ora e quaranta minuti al giorno, mentre secondo quanto riferito dal teste Musso essa arrivava fino a 10 ore, durata inverosimile in quanto superiore alla stessa durata della giornata lavorativa. Inoltre, secondo quanto emerso dall'istruttoria testimoniale, le telefonate tra l'appellato e i colleghi avvenivano anche mediante telefono fisso, e, d'altra parte, il figlio dell'appellato non è stato in grado di quantificare le telefonate ricevute dal padre fuori dell'orario di lavoro quando egli era reperibile. Né in base alle deposizioni dei testimoni è possibile determinare la quantità e la durata delle telefonate all'interno dell'abitacolo dell'autovettura.

Pur non potendosi ritenere, diversamente da quanto sostenuto dall'appellato, che le circostanze storiche relative all'esposizione siano provate per non essere state contestate dall'INAIL ex artt. 115 e 416 comma 3 c.p.c., trattandosi di fatti non noti all'Istituto e che quindi esso non è in grado di contestare o meno, il motivo è comunque infondato.

L'istruttoria testimoniale ha infatti confermato la notevolissima esposizione del sig. Romeo alle radiofrequenze per l'uso del telefono cellulare nel periodo 1995-2010.

Infatti, il teste Musso, collega dell'appellato dal 1990 al 2010, ha riferito che l'appellato coordinava la sua attività e quella degli altri tecnici esterni (di cui l'appellato era superiore gerarchico), pari complessivamente a 15-20 persone; il teste ha dichiarato che si sentiva con l'appellato quotidianamente più volte al giorno, circa 2-3 volte al giorno o anche di più, con chiamate della durata di 5-10 minuti ciascuna.

Il teste Nani, collega dell'appellato dal 2000 al 2011, ha dichiarato di essersi sentito con lui molto spesso, anche un paio di volte all'ora, e che le telefonate duravano 5 minuti, ma anche di meno.

Il teste Bilucaglia, che ha lavorato con l'appellato dai primi anni '90 al 1996, ha dichiarato che quest'ultimo coordinava circa 10-12 colleghi; e di avere contattato l'appellato almeno 2-3 volte in un giorno, con telefonate di circa 5-10 minuti ciascuna.

Come rilevato dal Tribunale, le telefonate dell'appellato intercorrevano anche con il direttore dei lavori, con le imprese esterne e con i superiori (v. testi Musso e Bilucaglia).

Escludendo quindi i valori massimi (che si ottengono considerando il numero più elevato di telefonate effettuate dai tecnici all'appellato e la durata massima di esse, come indicati dai testi) e prendendo perciò in considerazione il numero minimo e il numero medio di telefonate di ciascun tecnico (rispettivamente 2 e 2,5) per il numero di essi (l5-20 secondo Musso, 10-12 secondo Bilucaglia), si ottiene un'esposizione, secondo le testimonianze di Musso e Nani, da un minimo di 3,30

ore al giorno (200 minuti) a un medio di 5 ore al giorno (300 minuti), e, secondo la testimonianza di Bilucaglia, da un minimo di 1 ore e 40 minuti (100 minuti) a un medio di 3 ore e 50 minuti (230 minuti).

Pertanto, pur con il grado di precisione compatibile con il fatto di riferirsi a circostanze che, anche a distanza notevole di tempo, si ripetono durante un periodo lungo, anche con un inevitabile grado di variabilità, il quadro istruttorio consente, a parere della Corte, di ritenere provata un'esposizione a radiofrequenze molto elevata, che, in via del tutto prudenziale, va quantificata in circa 4 ore al giorno per tutto il periodo dedotto nel ricorso.

All'epoca non esistevano strumenti che consentissero di evitare il contatto diretto del telefono cellulare con il viso, come cuffiette o auricolari (v. teste Musso, e v. teste Nani, secondo cui le cuffiette, peraltro acquistate personalmente dai tecnici Telecom, avevano iniziato ad essere utilizzate a partire dall'inizio del 2000, e, nello stesso senso, v. teste Bilucaglia).

E' vero, come osservato dall'INAIL, che l'appellato disponeva di un ufficio dotato di un telefono fisso (v. teste Musso), ma i testi hanno riferito che lo contattavano sul telefono cellulare in quanto era più facile reperirlo, considerato che sovente si spostava fuori dell'ufficio e che era meno agevole rintracciarlo sul telefono fisso, in quanto in tal caso occorreva passare per il centralino (v. testi Musso, Nani, Bilucaglia).

E' poi emerso che la tecnologia ETACS (che, come si dirà più oltre con riferimento alla c.t.u. svolta nel presente grado,

emetteva radiofrequenze molto più potenti di quelle utilizzate attualmente dai telefoni cellulari) è durata circa 7 anni (teste Musso, v. anche teste Nani, che ha dichiarato che a partire dal 2000 prevaleva la tecnologia GSM; nello stesso senso, v. teste Bilucaglia).

Queste circostanze hanno reso l'esposizione, già di per sé prolungata, particolarmente intensa.

Il figlio dell'appellato, sentito come teste, ha poi confermato che il padre è destrimane.

Con il terzo motivo di gravame, l'INAIL deduce l'erroneità della conclusione del Tribunale in ordine all'esistenza del nesso eziologico tra la patologia e l'esposizione lavorativa a radiofrequenze.

In particolare:

-osserva in primo luogo che il neurinoma del nervo acustico non è una malattia tabellata, sicché l'onere di provare la natura professionale della patologia incombe sul ricorrente;

-critica la c.t.u. disposta dal Tribunale, evidenziando gli errori materiali ivi contenuti e sostenendo che essa perviene a conclusioni errate, poiché non suffragate da una legge scientifica generale di copertura o quantomeno da una legge scientifica che abbia un preponderante consenso;

-deduce che la c.t.u., le cui conclusioni sono state recepite dal Tribunale, si è basata sulla classificazione IARC del 2013, senza dare adeguatamente conto di studi successivi, e non ha correttamente valutato il significato della classificazione delle

radiofrequenze in relazione all'evidenza cancerogena, ossia come categoria 2B ("possibilmente cancerogeno per l'uomo"), e quindi la più debole tra quelle utilizzate dall'Agenzia per classificare agenti che presentino evidenze positive di cancerogenicità (a fronte della categoria 2A, "probabilmente cancerogeno per l'uomo" e della categoria 1, "cancerogeno per l'uomo");

-sostiene che lo studio Interphone deve ritenersi attendibile, in quanto studio caso-controllo indipendente, pur a fronte di un solo parziale finanziamento da parte di industrie di telefoni cellulari e operatori di telefonia mobile, come pure devono ritenersi attendibili gli studi di Hardell; detti studi e gli ulteriori, pur con i limiti evidenziati dalla relazione del dott. Grandi (ricercatore del Dipartimento di Medicina, Epidemiologia, Igiene del Lavoro e Ambientale INAIL), prodotta nel presente grado, non supportano l'associazione tra utilizzo del telefono cellulare e l'insorgenza del tumore;

-deduce che, diversamente da quanto sostenuto dal c.t.u. (e condiviso dal Tribunale), non sono conosciuti i meccanismi di azione delle radiofrequenze;

-sostiene che non è provato che l'appellato (soggetto destrimane) usasse il telefono cellulare appoggiandolo sempre all'orecchio destro;

-deduce inoltre che non è corretto, come ha fatto il Tribunale, inferire dalla coesistenza di due fenomeni rari (nel caso di

specie, tumore raro ed esposizione rara a radiofrequenze) un nesso di causa-effetto tra di essi;

-sostiene infine che erroneamente è stato ritenuto un periodo di latenza del tumore (secondo la dottrina scientifica, almeno 10 anni) compatibile con l'esposizione a radiofrequenze sin dal 1995, considerato che il tumore (a lentissima crescita), si è manifestato già nel dicembre 2009, e, pertanto, non risulta applicabile il rischio individuale pari a 1,44, riportato invece dal c.t.u..

Alla luce della c.t.u. disposta nel presente grado anche questo motivo di gravame è infondato.

I Consulenti d'Ufficio si sono correttamente attenuti al quesito formulato dalla Corte con ordinanza del 16.1.2019, in cui era richiesto di svolgere gli accertamenti peritali basandosi su un'esposizione pari a 4 ore al giorno (come dimostrata dall'istruttoria testimoniale di cui si è già detto), seppure per mero errore, nel verbale di conferimento incarico del 19.3.2019, si sia fatto riferimento al quesito formulato nel primo grado, che non precisava la durata dell'esposizione. Pertanto, in conformità ai tempi di esposizione indicati nel quesito conferito, è stato stimato un tempo di utilizzo lavorativo del telefono cellulare pari a 840 ore/anno (4 ore x 210 giorni lavorativi), con un tempo stimato complessivo di utilizzo nell'intervallo di 15 anni intercorso tra il 1995 ed il 2010 pari a 12.600 ore (840 ore/anno x 15 anni) (v. pag. 51 c.t.u.).

I periti hanno inoltre considerato che, come emerso dall'istruttoria, i telefoni cellulari utilizzati dall'appellato sino alla fine del 1999 erano analogici (utilizzavano la tecnologia ETACS) e quindi, dal 2000, erano digitali (utilizzavano la tecnologia GSM), evidenziando che "*I telefoni analogici e quelli digitali basati su tecnologia GSM 2G erano caratterizzati da emissioni di radiofrequenze (RF) molto superiori rispetto a quelli digitali attuali 3G e 4G, con livelli di intensità di emissioni di RF di quasi due ordini di grandezza superiori (IARC, 2013), ovvero quasi 100 volte superiori"* (v. pagg. 51-52 c.t.u., affermazione tratta dalla Monografia IARC (2013) sulle radiofrequenze, come precisato dai Consulenti d'Ufficio a pag. 121 della relazione).

Premesso che il neurinoma acustico (o schwannoma vestibolare, indicato per brevità nella c.t.u. come "NA"), tumore cerebrale benigno, raro e a crescita lenta, è caratterizzato da un periodo di latenza dall'inizio dell'esposizione ad un fattore di rischio fino al momento della diagnosi di malattia pari a non meno di 10-15 anni (v. pag. 54 e segg.), i Consulenti d'Ufficio hanno citato i numerosi studi sulla materia, dando atto che la maggior parte di essi sono studi caso-controllo che sono stati condotti dal gruppo di lavoro Interphone e dal gruppo di ricerca dell'Università di Orebro, Svezia, guidato dal prof. Hardell, evidenziandone le caratteristiche e le metodologie, nonché i limiti e le critiche svolte su di essi dalla letteratura scientifica (v. pag. 58 e segg.).

Dopo lo studio Interphone pubblicato nel 2010 sulla relazione tra esposizione a TC (telefono cellulare) e gliomi e meningiomi (tra cui non era quindi incluso il NA), "*Nel 2011 il gruppo di studio INTERPHONE pubblicava, in un altro articolo, i risultati dello studio internazionale caso-controllo su uso di telefoni cellulari e rischio di sviluppare neurinomi dell'acustico, che comprendeva più di 1.000 casi e oltre 2.000 controlli arruolati tra il 2000 e il 2004 (**INTERPHONE, 2011**).*

Questo studio non ha riscontrato differenze nell'esposizione pregressa a TC in casi e controlli per "utilizzo regolare" definito sulla base di almeno una chiamata alla settimana.

*Al contrario, ha osservato un **eccesso di rischio statisticamente significativo di sviluppare NA** (di quasi 3 volte nei soggetti esposti, rispetto ai non esposti), **nei soggetti** classificati nella classe più alta di esposizione, corrispondente ad un **utilizzo complessivo di TC superiore a 1.640 ore** (traducibili in durate medie di esposizione di 1 ora al giorno per 4 anni, o di 2 ore al giorno per 2 anni, o di mezz'ora al giorno per 8 anni)*", evidenziando inoltre che i risultati dello studio mostravano nella classe con più alta esposizione cumulativa (utilizzo complessivo di telefono cellulare maggiore o uguale a 1640 ore) un'associazione statisticamente significativa del NA solo con l'uso ipsilaterale di telefono cellulare (OR, o *Odds Ratio* = 3.74), sicchè "*Dal momento che, come anche osservato da Cardis (**Cardis, 2008**), le radiofrequenze (RF)/emissioni elettromagnetiche emesse dai telefoni portatili vengono*

assorbite soprattutto dal lato del capo al quale viene accostato l'apparecchio telefonico durante l'utilizzo (c.d. ***utilizzo ipsilaterale****) e che con l'aumentare della distanza del telefono dal capo la dose di radiazioni elettromagnetiche assorbita dai tessuti diminuisce bruscamente, il riscontro di un'associazione statisticamente significativa del NA solo con l'uso ipsilaterale di TC supporta l'ipotesi che le RF emesse dai TC svolgano un ruolo causale nell'induzione/sviluppo di NA*".

Con riferimento ad una delle osservazioni dell'appellante sopra riportate, rileva la Corte che, non contestato e confermato dalla testimonianza del figlio dell'appellato che quest'ultimo è destrimane, il fatto che si tenda ad usare il telefono, esclusivamente o quasi, appoggiandolo all'orecchio del lato del corpo "dominante", rientra nel fatto notorio essendo usualmente riscontrabile nell'esperienza comune.

I Consulenti d'Ufficio hanno poi citato la classificazione dello IARC (Agenzia Internazionale per la Ricerca sul Cancro) del 2011, secondo cui le radiofrequenze sono "possibilmente cancerogene per l'uomo", valutazione confermata nella monografia del 2013 sulle radiazioni non ionizzanti, evidenziando che nell'aprile 2019 un Advisory Group della IARC, composto da 29 ricercatori provenienti da 19 paesi, ha inserito le radiofrequenze tra gli agenti per cui è ritenuta prioritaria una rivalutazione di cancerogenicità da parte della IARC nel periodo 2020-2024 (IARC Monographs Priorities

Group, 2019). Hanno quindi menzionato gli studi successivi (v. pagg. 68-69).

Nella tabella redatta dai Consulenti d'Ufficio alle pagg. 70 e 71 della perizia sono riportate le caratteristiche e i risultati degli studi epidemiologici pubblicati sull'associazione tra utilizzo di TC e NA, relativi al rischio di NA stimato per i soggetti con la più alta esposizione cumulativa in ciascuno studio, in termini di durata dell'esposizione, di durata cumulativa del tempo di esposizione o della durata dell'abbonamento telefonico, divisi anche per utilizzo ipsilaterale e controlaterale rispetto all'insorgenza del tumore.

Come rilevato dai Consulenti d'Ufficio, dall'esame della tabella emerge che la maggioranza degli studi mostra eccessi di rischio associati ad elevata durata di utilizzo o esposizione cumulativa a TC, che in vari studi sono statisticamente significativi, con più alti rischi associati all'utilizzo ipsilaterale di TC.

Nella perizia è evidenziato "*il fatto che negli studi in cui il rischio di NA è stimato sulla base del numero di ore cumulative di utilizzo, la categoria con la più alta esposizione cumulativa stimata (che trova il monte ore più alto di 1640 ore nello studio INTERPHONE 2011) ha un limite che è almeno circa 8 volte più basso del numero di ore (12.600 ore circa) di utilizzo di TC stimato nel caso del Sig. Romeo*" (v. pag. 69 c.t.u.).

I Consulenti d'Ufficio hanno poi esaminato le evidenze da studi sperimentali su animali, pubblicati successivamente alla monografia IARC del 2013, di cui uno condotto dall'Istituto

Ramazzini e l'altro dal National Toxicology Program (NTP) statunitense: il primo ha osservato un incremento statisticamente significativo di Schwannoma delle cellule cardiache di Schwann a carico dei ratti maschi, anche se stimato su un numero limitato di casi (3 casi nel gruppo a più alta esposizione vs. 0 casi nel gruppo non esposto), ed un incremento non statisticamente significativo di iperplasia delle cellule cardiache di Schwann, che costituisce una lesione pre-tumorale, in entrambi i sessi (Falcioni et al., 2018); e anche il secondo ha mostrato, per i ratti maschi, un incrementato numero di casi di Schwannoma cardiaco, rispetto ai ratti maschi non esposti, che era statisticamente significativo sia per esposizione a radiofrequenze CDMA (3 casi nel gruppo con esposizione intermedia, 6 casi nel gruppo con la più alta esposizione e 0 casi tra i non esposti) che per esposizione a quelle da GSM (5 casi nel gruppo più esposto e 0 casi tra i non esposti) (NTP, 2018).

I Consulenti d'Ufficio hanno precisato che "g*li Schwannomi cardiaci sono dello stesso tipo istologico dei neurinomi del nervo acustico (che, infatti, sono denominati anche Schwannomi vestibolari), cosa che supporta una relazione causale tra esposizione a radiofrequenze e incidenza di NA"* (v. c.t.u. pag. 76).

In base a tutti questi elementi, i Consulenti d'Ufficio hanno concluso che "*Nel caso concreto specifico in esame, il rischio derivante dall'utilizzo professionale di telefono cellulare risulta decisamente aggravato in relazione principalmente al lungo*

periodo di esposizione (15 anni) ed all'elevata intensità dell'esposizione stessa, quest'ultima dovuta sia alla tipologia di apparecchi telefonici cellulari utilizzati (ETACS e quindi GSM 2G, con livelli di emissione quasi 100 volte superiori rispetto ai più moderni telefoni cellulari), che all'elevato numero di ore di utilizzo dell'apparecchio telefonico stesso (con un'esposizione media di 840 ore/anno, con conseguente esposizione complessiva in 15 anni stimata nell'ordine di 12.600 ore).

Pertanto, anche alla luce delle risultanze dei più recenti studi sugli animali condotti da NTP e dall'Istituto Ramazzini (che mostrano eccessi di tumori dello stesso tipo istologico del NA, anche se in altra sede) e dalle recenti indicazioni dell'Advisory Group della IARC sulla necessità di una prioritaria rivalutazione da parte della IARC della cancerogenicità delle radiofrequenze, considerando le risultanze degli studi epidemiologici disponibili che, per quanto non del tutto concordanti, mostrano comunque frequentemente un eccesso di casi di NA in presenza di prolungata esposizione o di esposizioni intense, ***è dato ritenere che, nello specifico caso in esame, con criterio di elevata probabilità logica, si possa ammettere un nesso eziologico tra la prolungata e cospicua esposizione lavorativa a radiofrequenze emesse da telefono cellulare e la malattia denunciata dal periziato all'INAIL (neurinoma dell'ottavo nervo cranico destro)***" (v. conclusioni preliminari a pagg. 77-78, ribadite a pagg. 123-124 nelle conclusioni e risposte ai quesiti).

Le conclusioni sono fondate su un accurato ed aggiornatissimo esame delle fonti della letteratura scientifica, applicata alle peculiarità del caso concreto (per quantità e durata dell'esposizione), in assenza di fattori alternativi di rischio, secondo standard di certezza probabilistica ("più probabile che non").

Rispetto alle conclusioni del Consulenti d'Ufficio, i Consulenti INAIL hanno svolto articolate osservazioni (riportate a pagg. 79-84 della relazione), mentre i difensori dell'appellato hanno sottolineato la posizione di conflitto di interesse di alcuni autori di studi che hanno negato la cancerogenicità delle radiofrequenze (v. pagg. 84-97 c.t.u.), in particolare nell'ambito della letteratura citata dall'INAIL (v. pagg. 94-95).

Ritiene la Corte che i Consulenti d'Ufficio abbiano fornito esaustive risposte in merito alle osservazioni dei Consulenti di parte appellante.

In particolare:

1) i dati relativi all'esposizione su cui si sono basati i Consulenti d'Ufficio non sono, come sostenuto dai Consulenti INAIL, tratti "*sostanzialmente dalle informazioni anamnestiche riferite dall'assicurato"*, bensì, come già osservato, oggetto del quesito formulato dal Collegio avuto riguardo alle circostanze comprovate all'istruttoria testimoniale già sopra descritta;

2) con riferimento alle critiche sull'attendibilità degli studi secondo cui sussiste un nesso eziologico tra esposizione a

radiofrequenze e il neurinoma dell'acustico, i Consulenti d'Ufficio hanno svolto le seguenti articolate repliche:

a) quanto alle possibili distorsioni ("*bias*"), i Consulenti d'Ufficio hanno illustrato le differenze tra gli studi caso-controllo e gli studi di coorte, precisando che nella materia in esame la letteratura è quasi interamente costituita da studi caso-controllo. In questo tipo di studio (a differenza degli studi di coorte, da cui si ricava il rapporto tra l'incidenza della malattia nella popolazione esposta al fattore di rischio e l'incidenza della stessa malattia nella popolazione non esposta), il rischio relativo (RR) è approssimato da un altro indicatore di rischio, ovvero l'Odds Ratio (OR), che viene calcolato sulla base del rapporto tra la frequenza di esposizione al fattore di rischio tra i casi (malati) rispetto alla frequenza di esposizione al fattore di rischio tra i controlli (non malati).

Ciò rende possibili misclassificazioni non differenziali (che interessano sia i casi che i controlli nella stessa misura), le quali, come evidenziato dai Consulenti d'Ufficio, determinano sempre una sottostima del rischio rispetto al rischio reale, e misclassificazioni differenziali dell'esposizione (errori di classificazione che interessano in diversa entità i casi rispetto ai controlli), le quali possono condurre sia ad una sovrastima che ad una sottostima del reale rischio di malattia dovuto all'esposizione, e la più seria minaccia alla validità dei risultati è costituita da una forma di misclassificazione differenziale dell'esposizione denominata "*recall bias*", dovuta alla possibilità

che i soggetti che risultano affetti da malattia tumorale ricerchino nella propria memoria dei dati relativi alla propria pregressa esposizione a possibili fattori di rischio per la salute che possano avere determinato tale malattia.

Tuttavia i risultati degli studi disponibili (lo studio di Vrijheid et al., 2009, lo studio di Aydin et al., 2011, e lo studio di Petterson et al., 2015) indicano che è improbabile che gli studi su esposizione a TC e rischio di NA siano stati affetti da una misclassificazione differenziale dell'esposizione a RF da TC, tale da determinare una sovrastima dell'esposizione tra i casi rispetto ai controlli e, pertanto, una conseguente sovrastima del rischio di NA associato all'esposizione a RF da TC; al contrario, sia i risultati di detti studi, che quelli di altri studi che hanno valutato, in soggetti sani, la validità dell'esposizione a TC "autoriferita" (ovvero riferita dagli stessi soggetti inclusi nello studio e rilevata per mezzo di questionario o intervista ad essi somministrati), indicano la presenza di una forte misclassificazione non differenziale dell'esposizione (Samkange-Zeeb et al., 2004; Toledano et al., 2014; Vanden Abeele et al., 2013), con conseguente sottostima della forza dell'associazione tra esposizione a TC e rischio di NA, rispetto al rischio reale, sicché le stime di rischio (O.R.) ottenute nei diversi studi sarebbero fortemente sottostimate e il rischio reale di sviluppare NA sarebbe molto più alto di quello osservato negli studi stessi (v. pagg 99-103 c.t.u.);

b) anche quanto alla ipsilateralità dell'utilizzo del telefono cellulare rispetto al lato di comparsa del tumore gli studi disponibili (Shimizu e Yamaguchi, 2012) evidenziano la possibilità di una forte misclassificazione non differenziale, con conseguente sottostima (v. pag. 103 c.t.u.);

c) a differenza di quanto sostenuto dai Consulenti di parte INAIL, un effetto dose-risposta, cioè un significativo aumento del rischio di sviluppare la malattia tumorale (NA) all'aumentare della dose cumulativa di esposizione a RF da TC, è presente nei risultati della pooled analysis di Hardell et al. (2013), come da tabella riportata a pag. 104 della relazione, che mostra un rischio di NA associato all'uso di telefoni wireless progressivamente crescente all'aumentare della dose cumulativa di esposizione a TC (calcolata in base alle ore di utilizzo di TC): v. pagg. 103-105 c.t.u.;

d) un possibile motivo della mancanza di un effetto dose-risposta nello studio Interphone (2011) e in altri studi è che le categorie di esposizione cumulativa utilizzate fossero troppo basse: per esempio, nello studio Interphone il limite inferiore per la categoria di esposizione cumulativa più alta era posto a sole 1.640 ore di utilizzo di TC, corrispondenti a meno di mezz'ora al giorno per 10 anni. Come osservato nella relazione peritale, una dose di esposizione al di sotto di questo limite potrebbe essere non sufficiente a determinare lo sviluppo di NA (v. pag. 105 c.t.u.).

Si tratta peraltro di una dose di esposizione, come emerge dalla perizia, assolutamente non confrontabile con la massiccia e prolungata esposizione a radiofrequenze subita dall'appellato per ben 15 anni;

e) l'affermazione dei Consulenti INAIL secondo cui soggetti audiolesi protesizzati, che possiedono sussidi uditivi che utilizzano quotidianamente per l'intera giornata con annessa funzione bluetooth, non hanno mai fatto riscontrare casi di neurinomi dell'acustico, non è supportata da alcun riferimento bibliografico (v. pag. 107 c.t.u.);

f) diversamente da quanto sostenuto dai Consulenti INAIL, il trend della patologia per cui è causa (schwannoma dell'VIII nervo cranico) mostra un aumento, in coincidenza con la diffusione della telefonia cellulare, di detta malattia nel corso degli ultimi decenni. I Consulenti d'Ufficio hanno indicato, nelle pagg. 55-57 della relazione, i diversi studi sulla questione, rilevando che, secondo alcuni di essi, l'aumento di incidenza della malattia sarebbe attribuibile al miglioramento delle tecniche strumentali - basata sulla diffusione di nuove tecnologie, ad esempio TAC e RMN - utilizzate per pervenire alla diagnosi di tale tumore; ma osservando tuttavia che studi basati sui dati più recenti mostrano un ulteriore incremento di incidenza di NA, anche riferito a periodi in cui la diffusione dei migliori strumenti di diagnostica di questi tumori era già avvenuto (Kleijwegt et al., 2016: aumento nella regione di Leyden dell'incidenza di NA di oltre 3 volte in un arco

temporale di 11 anni intercorrente tra il 2001 al 2012; Marinelli et al., 2018: aumento dell'incidenza di NA in Minnesota, USA, di oltre 2 volte in un arco temporale di 11 anni intercorrente tra il 1995 al 2016; sempre negli USA, il Central Brain Tumor Registry, CBTRUS, ha pubblicato report annuali dal 2007 al 2016 con dati registrati dal 2004 al 2013 che evidenziano un raddoppio dell'incidenza annuale di NA: da 0,88 a 1,73 x 100.000); a pag. 108 della relazione hanno richiamato i dati del registro tumori danese che evidenziano un incremento nell'incidenza di tumori cerebrali, con un aumento del 40% tra gli uomini e del 29% tra le donne tra il 2001 e il 2010 (Sundhedsstyrelsen, 2010).

E' quindi condivisibile la conclusione dei Consulenti d'Ufficio secondo cui è improbabile che l'incremento di incidenza di NA sia attribuibile unicamente alla possibilità, derivante dell'affinamento delle metodiche diagnostiche di tale tumore o anche da una maggiore accessibilità della popolazione alle strutture sanitarie, di ottenere più diagnosi di NA.

3) Con riferimento agli studi di NTP e dell'Istituto Ramazzini, alle osservazioni critiche dei Consulenti INAIL sulla loro validità scientifica, anche mediante richiamo al recentissimo articolo pubblicato dall'International Commission on Non Ionizing Radiation Protection (ICNIRP) su Health Physics, i Consulenti d'Ufficio (v. pagg. 108-113 della relazione) hanno esaustivamente replicato che:

- si tratta dei più grandi studi sperimentali su animali condotti finora e sono caratterizzati da elevata standardizzazione dei protocolli di ricerca e da alta qualità dei metodi utilizzati;
- lo scopo principale degli studi sperimentali sui tumori condotti sugli animali è quello di valutare se l'esposizione ad un sospetto agente cancerogeno provochi o meno eccessi di tumori nei gruppi di animali esposti. Pertanto il fatto che, per gli animali oggetto di studio, possano essere previsti tempi e modalità di esposizione differenti rispetto a quelli degli esseri umani (per i roditori, a differenza che per l'uomo, "*total body"* e per l'intera vita), non rende i risultati degli studi meno validi.

Inoltre, con riferimento all'osservazione della difesa dell'INAIL, nel corso della discussione orale, circa l'inattendibilità di questi studi in quanto non effettuati sull'uomo, la Corte ritiene esaustiva e condivisibile la replica dei Consulenti d'Ufficio (anche mediante richiamo a fonti di letteratura scientifica sullo studio del NTP) secondo cui il criterio razionale per condurre studi di cancerogenicità in modelli animali *"si basa su dati sperimentali che mostrano che ogni agente noto come cancerogeno nell'uomo, quando adeguatamente testato, ha mostrato di essere cancerogeno negli animali (IARC, 2006) e che quasi un terzo dei cancerogeni umani sono stati identificati dopo che effetti cancerogeni sono stati trovati in studi ben condotti sugli animali (Huff, 1993). Non c'è ragione di credere che un agente fisico come le radiofrequenze possa danneggiare i tessuti animali, ma non i tessuti umani*" (Melnick, 2019, citato

alle pagg. 76-77 e 109 della relazione). Le sperimentazioni sulla cancerogenicità di agenti o sostanze vengono usualmente eseguite su animali, quali i roditori, che presentano elementi di similitudine con gli uomini, sicché non si può negare pregiudizialmente valore scientifico ai risultati di detti studi;

- il fatto che l'eccesso di tumore sia stato riscontrato soltanto nei ratti (e quasi esclusivamente di sesso maschile) non inficia la validità dello studio, considerato che lo schwannoma cardiaco insorge in diverse varietà di ceppi di ratti (e con maggior frequenza nei maschi), ma non è mai stato osservato nei topi;

- nonostante, nello studio dell'Istituto Ramazzini, l'esposizione dei ratti sia avvenuta alla dose massima testata, il tasso di assorbimento specifico conseguente all'esposizione era di poco superiore al limite massimo per irradiazione al corpo intero per l'uomo; mentre, quanto allo studio del NTP, pur essendo la dose di esposizione molto superiore al limite massimo di esposizione ammissibile per irradiazione al corpo intero per l'uomo, la dose assorbita a livello locale è solo una piccola parte della dose somministrata a tutto il corpo, e, in particolare, per il cervello, la dose assorbita è stata stimata in circa il 10% della dose totale somministrata a tutto il corpo;

- il numero di casi di tumore riscontrato negli animali è statisticamente significativo: nello studio di NTP, 6 casi nel gruppo a più alta esposizione a RF da CDMA e 5 casi in quello con più alta esposizione a RF da GSM, mentre nessun caso si è verificato nel gruppo non esposto; nello studio dell'Istituto

Ramazzini, 3 casi osservati nel gruppo a più alta esposizione e nessuno nel gruppo non esposto;

- in merito alla diversa localizzazione degli schwannomi riscontrati nei ratti esposti negli studi dell'NTP e dell'Istituto Ramazzini (localizzazione a livello cardiaco invece che a livello cerebrale), appare probabile che la modalità di irradiazione degli animali abbia influito nel determinare questo risultato, in quanto la somministrazione di RF è stata indirizzata a tutto il corpo e non concentrata solo sulla testa degli animali da esperimento, come invece avviene per l'esposizione a RF negli utilizzatori di TC;

- gli schwannomi cardiaci sono dello stesso tipo istologico dei neurinomi del nervo acustico (che, infatti, sono denominati anche schwannomi vestibolari), cosa che supporta una relazione causale tra esposizione a radiofrequenze e incidenza di NA. Pertanto, il fatto che i NA siano tumori benigni, al contrario degli schwannomi cardiaci maligni osservati nei ratti negli studi del NTP e dell'Istituto Ramazzini, appare irrilevante, considerato che questi studi dimostrano che l'esposizione a RF può determinare una trasformazione neoplastica delle cellule di Schwann, processo che sia i tumori benigni che i tumori maligni hanno in comune;

- lo studio del NTP ha concluso affermando che i risultati dimostrano una chiara evidenza di attività cancerogena delle RF (NTP, 2018);

- l'effettuazione di confronti multipli nelle analisi condotte nei due studi del NTP e dell'Istituto Ramazzini ha sicuramente aumentato il rischio che si verificassero associazioni spurie in questi due studi, ma la probabilità che tre analisi indipendenti abbiano trovato solo per caso un incremento significativo di sviluppare tumori dello stesso tipo istologico e nella stessa sede anatomica è bassissima, anche considerando i molti confronti effettuati in analisi, ciò che supporta in maniera inequivocabile l'effetto cancerogeno delle RF;
- la presenza di un effetto cancerogeno è supportata anche dall'osservazione di un significativo aumento del danno al DNA, valutato per mezzo della presenza di rotture del DNA con la metodica Comet assay, in vari organi, tra cui soprattutto il cervello, sia in ratti che in topi (Wyde, 2016);
- diversamente da quanto sostenuto dai Consulenti INAIL, le analisi sono state condotte "in cieco" (v. articolo di Melnick del 2019, in risposta alle critiche dell'INCIRP riguardo allo studio del NTP);
4) In merito alla motivazione per la quale l'Advisory Group della IARC ha inserito le radiofrequenze tra gli agenti per cui è ritenuta prioritaria una rivalutazione di cancerogenicità da parte della IARC nel periodo 2020-2024 (secondo i Consulenti INAIL non per motivi di particolare allarme, ma in quanto rivalutazione rientrante nelle normali procedure di aggiornamento periodico delle valutazioni di evidenza cancerogena promosse dall'Agenzia), nella relazione peritale è trascritta la tabella

riportata nell'articolo, dalla quale si ricava che le radiazioni non ionizzanti (radiofrequenze) sono tra gli agenti per i quali è raccomandata una rivalutazione urgente ("*high priority*") della cancerogenicità per l'uomo, indicazione, specificata nella tabella stessa, motivata dal fatto che le nuove evidenze derivanti da test biologici e meccanicistici "*richiedono una rivalutazione della classificazione*". Nell'articolo dell'Advisory Group è inoltre specificato che la priorità per la rivalutazione è stata assegnata sulla base di evidenze sull'esposizione umana e in base al grado di evidenza disponibile per valutare la cancerogenicità (v. pagg. 113-115 c.t.u.);

5) Quanto alle osservazioni dei Consulenti INAIL circa l'incompatibilità dell'evoluzione della patologia dell'appellato (essendo il tumore, già nel 2010, di dimensioni pari a 2,6 cm, a fronte di un ritmo di crescita di circa 1,5 mm all'anno) e i periodi di latenza della stessa (oltre 15-20 anni, non meno di 10-15 anni), i Consulenti d'Ufficio hanno osservato che, secondo l'autore citato dai Consulenti INAIL (Dott. P. Ferroli, Istituto Besta di Milano), il ritmo di crescita del tumore, di circa 1,5 mm all'anno, si riferisce a circa il 75% dei neurinomi dell'acustico, mentre un quarto di essi ha tendenza a crescere più rapidamente e in maniera più aggressiva (v. pag. 116 c.t.u.). Inoltre, i Consulenti d'Ufficio, alle pagg. 116-117 della relazione, hanno citato ampia letteratura scientifica da cui risultano tassi di crescita del neurinoma dell'acustico piuttosto variabili. In particolare, in caso di NA caratterizzati da fenomeni cistici ed

emorragici (come quello dell'appellato), sono stati osservati tassi di crescita di oltre 4 mm/anno (Paldor et al., 2016), e nella revisione di Paldor vengono citati anche alcuni case reports nei quali sono stati descritti casi di NA con tassi di crescita fino a 25 mm/anno (Fayad et al, 2014).

Appare dunque condivisibile la conclusione sul punto dei Consulenti d'Ufficio secondo cui "*I tassi di crescita del NA osservati nella letteratura scientifica, la presenza nel caso in esame di fenomeni cistico-necrotici (anche citati dai CTP INAIL) e il lungo periodo intercorso tra la prima esposizione e la diagnosi di NA (15 anni), rappresentano elementi certamente non idonei a giustificare una esclusione del nesso causale tra esposizione a RF da TC e insorgenza di NA, così come sostenuto dai CTP INAIL.*

Al contrario, tali dati rappresentano elementi assolutamente compatibili con la sussistenza, nel caso in esame, del riscontro di un NA delle dimensioni di 2.6 cm al momento della diagnosi, in soggetto esposto da 15 anni a RF da TC" (v. pag. 117).

6) Pertanto, considerato il periodo di esposizione dell'appellato alle radiofrequenze (dal 1995 al 2010, anno in cui gli è stato diagnosticato il NA), il tempo intercorso tra l'inizio dell'esposizione e la comparsa del tumore (pari a15 anni, e non a 4 anni come sostenuto dai Consulenti INAIL) è assolutamente compatibile con l'induzione e lo sviluppo del NA sulla base dei dati di letteratura, anche considerando 5 anni per l'iniziazione del tumore e 10 anni per il suo sviluppo.

Inoltre, diversamente da quanto sostenuto dalla difesa dell'appellante nel corso della discussione orale, non vi è contraddizione tra quanto argomentato dai Consulenti d'Ufficio alle pagg. 115-118 in merito alla latenza della malattia, al suo sviluppo e alle dimensioni del tumore al momento della diagnosi nel 2010 (2,6 cm), e quanto scritto alle pagg. 57-58 della relazione sul periodo di latenza riconosciuto nella letteratura scientifica (almeno 10-15 anni), avendo i Consulenti d'Ufficio motivato sulla compatibilità tra il periodo di latenza della malattia e le dimensioni del tumore, menzionando (a differenza dei Consulenti INAIL) copiosa letteratura scientifica sull'estrema variabilità della crescita media del tumore, che ha registrato anche casi di valori massimi pari a 17 mm/anno e addirittura fino a 25 mm/anno (v. pagg. 116-117 c.t.u.).

7) Non vi è contraddizione tra l'affermazione dei Consulenti d'Ufficio (v. nota 25 a pag. 70 della relazione) secondo cui «*Appare quindi improbabile che si possano vedere gli eventuali effetti dell'uso di TC sull'incidenza dei NA, almeno sui dati fino al 2010, data la diffusione relativamente recente dei TC e il lungo periodo di induzione di questi tumori*» e l'affermazione dell'esistenza del nesso eziologico nel caso di specie, poiché la frase di cui sopra è evidentemente riferita al fatto che appare improbabile che negli studi epidemiologici si potessero vedere eventuali effetti dell'uso di telefono cellulare, in quanto nelle popolazioni esaminate da tali studi l'inizio dell'esposizione, per la gran parte dei soggetti, era troppo recente, mentre, nel caso

concreto in esame, l'esposizione dell'appellato ha avuto inizio nel 1995, ovvero 15 anni prima della diagnosi del tumore (NA) ed in un periodo storico in cui i TC erano ancora poco diffusi nella maggior parte dei paesi europei (v. pagg. 118-119 c.t.u.).

I Consulenti d'Ufficio hanno pertanto ravvisato il nesso causale tenendo correttamente in considerazione la concreta esposizione dell'appellato alle radiofrequenze, che, per le sue peculiarità (durata ed intensità conseguente all'uso abnorme del telefono cellulare), presenta caratteristiche del tutto diverse da quelle medie riscontrate in generale dalla popolazione nel periodo per cui è causa;

8) con riferimento alle conclusioni dei Consulenti INAIL, che, al fine di escludere il nesso causale, richiamano il documento dell'ISS, rapporto ISTISAN 19/11, i Consulenti d'Ufficio hanno esaustivamente replicato che: "*il rapporto ISTISAN su RF e tumori è stato criticato dall'associazione Medici per l'Ambiente (ISDE, acronimo di International Society of Doctors for Environment) per varie ragioni (Di Ciaula, 2019), tra cui: la selezione degli studi inclusi nelle meta-analisi presentate; l'interpretazione delle associazioni osservate tra RF e tumori intracranici; l'uso inappropriato dei dati sull'andamento dell'incidenza dei tumori cerebrali per confutare l'associazione tra RF e tumori cerebrali; il non aver tenuto conto nella loro valutazione dei risultati di recenti studi sperimentali su animali, …, che hanno mostrato effetti cancerogeni su ratti (NTP, 2018; Falcioni et al., 2018) e, soprattutto, per non avere fatto*

conseguire alla dichiarata incertezza sugli effetti associati ad un uso intenso e prolungato di TC raccomandazioni più stringenti sui limiti di esposizione a RF, in particolare per i bambini e gli adolescenti, che potrebbero essere maggiormente suscettibili a tali effetti (Di Ciaula, 2019)" (v. pag. 119 c.t.u.).

I Consulenti d'Ufficio hanno poi menzionato il rapporto della ANSES (Agenzia Nazionale Francese per la Sicurezza Sanitaria per Alimentazione Ambiente e Lavoro) sugli effetti delle onde emesse dai telefoni mobili sulla salute, che conclude segnalando che gli studi scientifici pubblicati sino ad oggi non permettono di escludere la comparsa di effetti biologici per l'uomo oltre certe soglie di esposizione a RF da TC, evidenziando inoltre che il 76% dei telefoni cellulari esaminati emette radiofrequenze superiori al limite massimo raccomandato dall'ICNIRP per esposizione di testa e tronco (v. pagg. 119-121 c.t.u.).

I Consulenti d'Ufficio, a parere della Corte, hanno replicato punto per punto alle osservazioni dei Consulenti INAIL, menzionando copiosa letteratura scientifica a supporto delle proprie argomentazioni, e fornendo, in conclusione, solidi elementi per affermare un ruolo causale tra l'esposizione dell'appellato alle radiofrequenze da telefono cellulare e la patologia per cui è causa.

I dati epidemiologici, i risultati delle sperimentazioni sugli animali (non contraddetti, allo stato, da altre sperimentazioni dello stesso tipo), la durata e l'intensità dell'esposizione (assolutamente peculiari per la loro abnormità) che assumono

particolare rilievo considerata l'accertata – a livello scientifico – relazione dose-risposta tra esposizione a radiofrequenze da telefono cellulare e rischio di neurinoma dell'acustico, unitamente alla mancanza di un altro fattore che possa avere cagionato la patologia, complessivamente valutati, consentono di ritenere che, caso specifico, sussista una legge scientifica di copertura che supporta l'affermazione del nesso causale secondo criteri probabilistici ("più probabile che non").

In effetti, buona parte della letteratura scientifica che esclude la cancerogenicità dell'esposizione a radiofrequenze, o che quantomeno sostiene che le ricerche giunte ad opposte conclusioni non possano essere considerate conclusive, come evidenziato anche dai Consulenti d'Ufficio a commento delle osservazioni della difesa dell'appellato (riportate alle pagg. 84-97 della relazione), versa in posizione di conflitto di interessi, peraltro non sempre dichiarato: si veda in particolare, a pag. 94 della relazione, l'osservazione della difesa dell'appellato (in alcun modo contestata dalla controparte) secondo cui gli autori degli studi indicati dall'INAIL, nominativamente elencati, sono membri di ICNIRP e/o di SCENIHR, che hanno ricevuto, direttamente o indirettamente, finanziamenti dall'industria.

I Consulenti d'Ufficio hanno al riguardo osservato: "*Inoltre, anche alla luce dell'ampia documentazione sui conflitti di interesse di diversi ricercatori coinvolti nello studio INTERPHONE, pure prodotta dai consulenti dell'appellante, si ritiene che debba essere dato minor peso agli studi pubblicati da*

autori che non hanno dichiarato l'esistenza di conflitti di interesse invece sussistenti e che debba essere dato maggior peso ai risultati di studi condotti da ricercatori esenti da tali conflitti, come ad esempio gli studi effettuati da Hardell e collaboratori.

*Nel caso in esame, possono concretizzare situazioni di conflitto di interesse rispetto alla valutazione dell'effetto sulla salute delle RF, ad esempio, quei casi in cui l'autore dello studio ha effettuato consulenze per l'industria telefonica o ha ricevuto finanziamenti per la realizzazione di studi dall'industria telefonica oppure (come anche stabilito anche dal Karolinska Institutet di Stoccolma, in relazione all'esposto presentato contro il prof. Ahlbom, poi destituito dalla presidenza del gruppo di lavoro IARC sulle RF proprio a causa della sua appartenenza all'ICNIRP) nel caso in cui l'autore stesso sia membro dell'ICNIRP (International Commission on Non-Ionizing Radiation). Infatti l'**ICNIRP** è un'organizzazione privata, le cui linee guida sulle RF hanno una grande importanza economica e strategica per l'industria delle telecomunicazioni, con la quale peraltro diversi membri dell'ICNIRP hanno legami attraverso rapporti di consulenza ... A parte possibili legami con l'industria, appare evidente che i membri dell'ICNIRP dovrebbero astenersi dal valutare l'effetto sulla salute di livelli di RF che l'ICNIRP stesso ha già dichiarato sicuri e quindi non nocivi per la salute (**Hardell, 2017**)*" (v. pag. 107 relazione).

L'impostazione dei Consulenti d'Ufficio è del tutto condivisibile, essendo evidente che l'indagine, e le conclusioni, di autori indipendenti diano maggiori garanzie di attendibilità rispetto a quelle commissionate, gestite o finanziate almeno in parte, da soggetti interessati all'esito degli studi.

L'ampia letteratura scientifica citata ed applicata dai Consulenti d'Ufficio, del tutto indipendente, deve quindi ritenersi affidabile, così come le conclusioni, a livello epidemiologico, a cui essa è pervenuta.

Del resto, proprio in una controversia nei confronti dell'INAIL relativa a malattia professionale (tumore intracranico) per esposizione a radiofrequenze da telefono cellulare, la S.C. ha ritenuto che "*L'ulteriore rilievo circa la maggiore attendibilità proprio di tali studi, stante la loro posizione di indipendenza, ossia per non essere stati cofinanziati, a differenza di altri, anche dalle stesse ditte produttrici di cellulari, costituisce ulteriore e non illogico fondamento delle conclusioni accolte"* (v. Cass. 12.10.2012 n. 17438).

Trattandosi di malattia professionale non tabellata e ad eziologia multifattoriale, la prova della causa di lavoro, indubbiamente gravante sul lavoratore, per costante giurisprudenza di legittimità deve essere valutata in termini di ragionevole certezza, e quindi, esclusa la rilevanza della mera possibilità dell'origine professionale, essa può essere ravvisata in presenza di un rilevante grado di probabilità (cfr., tra le molte, Cass. 10.4.2018

n. 8773), grado che, per le ragioni illustrate, è emerso dalla c.t.u. disposta nel presente grado.

La percentuale di invalidità nella misura del 23%, già riconosciuta nella c.t.u. disposta dal Tribunale e ribadita dalla consulenza espletata nel presente grado, è stata espressamente accettata dall'appellato (v. pag. 3, punto a, memoria appellato).

In conclusione, l'appello dev'essere respinto.

Le spese del grado seguono la soccombenza e si liquidano in dispositivo in conformità ai parametri vigenti, tenuto conto del valore della causa e dell'attività difensiva svolta, con distrazione in favore dei difensori.

Le spese di c.t.u., viste le conclusioni a cui essa è pervenuta, vanno poste a carico definitivo dell'INAIL.

Al rigetto dell'appello consegue *ex lege* (art. 1, commi 17-18, l. 228/2012) la dichiarazione che sussistono i presupposti per l'ulteriore pagamento, a carico dell'appellante, di un importo pari a quello del contributo unificato dovuto per l'impugnazione.

P . Q . M .

Visto l'art. 437 c.p.c.,

respinge l'appello;

condanna l'Inail a rimborsare all'appellato le spese del grado, liquidate in euro 10.000,00, oltre rimborso forfettario, Iva e Cpa, con distrazione in favore dei difensori;

pone a carico dell'appellante gli oneri di CTU, liquidati come da separato decreto;

dichiara la sussistenza delle condizioni per l'ulteriore pagamento,

a carico dell'appellante, di un importo pari a quello del contributo unificato dovuto per l'impugnazione.

Così deciso all'udienza del 3.12.2019

IL CONSIGLIERE Est. **LA PRESIDENTE**

Dott.ssa Silvia CASARINO Dott.ssa Rita MANCUSO

VERIZON COMMUNICATIONS INC

FORM 10-K
(Annual Report)

Filed 02/21/20 for the Period Ending 12/31/19

Address	1095 AVENUE OF THE AMERICAS NEW YORK, NY, 10036
Telephone	212-395-1000
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Symbol	VZ
SIC Code	4813 - Telephone Communications (No Radiotelephone)
Industry	Integrated Telecommunications Services
Sector	Telecommunication Services
Fiscal Year	12/31

UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-K

(Mark one)

☒ ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

For the fiscal year ended December 31, 2019

OR

☐ TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

For the transition period from to

Commission file number: 1-8606

Verizon Communications Inc.

(Exact name of registrant as specified in its charter)

Delaware	**23-2259884**
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification No.)
1095 Avenue of the Americas	
New York, New York	**10036**
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: (212) 395-1000

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class	Trading Symbol(s)	Name of Each Exchange on Which Registered
Common Stock, par value $0.10	VZ	New York Stock Exchange
Common Stock, par value $0.10	VZ	The NASDAQ Global Select Market
2.375% Notes due 2022	VZ22A	New York Stock Exchange
0.500% Notes due 2022	VZ22B	New York Stock Exchange
1.625% Notes due 2024	VZ24B	New York Stock Exchange
4.073% Notes due 2024	VZ24C	New York Stock Exchange
0.875% Notes due 2025	VZ25	New York Stock Exchange
3.250% Notes due 2026	VZ26	New York Stock Exchange
1.375% Notes due 2026	VZ26B	New York Stock Exchange
0.875% Notes due 2027	VZ27E	New York Stock Exchange
1.375% Notes due 2028	VZ28	New York Stock Exchange
1.875% Notes due 2029	VZ29B	New York Stock Exchange
1.250% Notes due 2030	VZ30	New York Stock Exchange
1.875% Notes due 2030	VZ30A	New York Stock Exchange
2.625% Notes due 2031	VZ31	New York Stock Exchange
2.500% Notes due 2031	VZ31A	New York Stock Exchange
0.875% Notes due 2032	VZ32	New York Stock Exchange
4.750% Notes due 2034	VZ34	New York Stock Exchange
3.125% Notes due 2035	VZ35	New York Stock Exchange
3.375% Notes due 2036	VZ36A	New York Stock Exchange
2.875% Notes due 2038	VZ38B	New York Stock Exchange
1.500% Notes due 2039	VZ39C	New York Stock Exchange

Securities registered pursuant to Section 12(g) of the Act: None

Indicate by check mark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. ☒ Yes ☐ No

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Act. ☐ Yes ☒ No

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. ☒ Yes ☐ No

Indicate by check mark whether the registrant has submitted electronically every Interactive Data File required to be submitted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit such files). ☒ Yes ☐ No

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer, a smaller reporting company, or an emerging growth company. See the definitions of "large accelerated filer," "accelerated filer," "smaller reporting company," and "emerging growth company" in Rule 12b-2 of the Exchange Act. (Check one):

Large accelerated filer	☒	Accelerated filer	☐
Non-accelerated filer	☐	Smaller reporting company	☐
		Emerging growth company	☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Act). ☐ Yes ☒ No

At June 30, 2019, the aggregate market value of the registrant's voting stock held by non-affiliates was approximately $236,226,048,492.

At January 31, 2020, 4,135,863,778 shares of the registrant's common stock were outstanding, after deducting 155,569,868 shares held in treasury.

Documents Incorporated By Reference:

Portions of the registrant's Annual Report to Shareholders for the year ended December 31, 2019 (Parts I and II).

Portions of the registrant's definitive Proxy Statement to be delivered to shareholders in connection with the registrant's 2020 Annual Meeting of Shareholders (Part III).

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PART I

Item 1. Business

General

Verizon Communications Inc. (Verizon or the Company) is a holding company that, acting through its subsidiaries, is one of the world's leading providers of communications, information and entertainment products and services to consumers, businesses and government entities. With a presence around the world, we offer voice, data and video services and solutions on our networks that are designed to meet customers' demand for mobility, reliable network connectivity, security and control. Formerly known as Bell Atlantic Corporation (Bell Atlantic), we were incorporated in 1983 under the laws of the State of Delaware. We began doing business as Verizon on June 30, 2000 following our merger with GTE Corporation. We have a highly diverse workforce of approximately 135,000 employees as of December 31, 2019.

Our principal executive offices are located at 1095 Avenue of the Americas, New York, New York 10036 (telephone number 212-395-1000).

In November 2018, we announced a strategic reorganization of our business. Under the new structure, effective April 1, 2019, there are two reportable segments that we operate and manage as strategic business units - Verizon Consumer Group (Consumer) and Verizon Business Group (Business).

Verizon Consumer Group

Our Consumer segment provides consumer-focused wireless and wireline communications services and products. Our wireless services are provided across one of the most extensive wireless networks in the United States (U.S.) under the Verizon brand and through wholesale and other arrangements. Our wireline services are provided in nine states in the Mid-Atlantic and Northeastern U.S., as well as Washington D.C., over our 100% fiber-optic network under the Fios brand and over a traditional copper-based network to customers who are not served by Fios. In 2019, the Consumer segment's revenues were $91.1 billion, representing approximately 69% of Verizon's consolidated revenues. As of December 31, 2019, Consumer had approximately 95 million wireless retail connections, 6 million broadband connections and 4 million Fios video connections.

Verizon Business Group

Our Business segment provides wireless and wireline communications services and products, video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various Internet of Things (IoT) services and products. We provide these products and services to businesses, government customers and wireless and wireline carriers across the U.S. and select products and services to customers around the world. In 2019, the Business segment's revenues were $31.4 billion, representing approximately 24% of Verizon's consolidated revenues. As of December 31, 2019, Business had approximately 25 million wireless retail postpaid connections and approximately 489 thousand broadband connections.

Additional discussion of our reportable segments is included in the 2019 Verizon Annual Report to Shareholders under the headings "Management's Discussion and Analysis of Financial Condition and Results of Operations - Overview" and - "Segment Results of Operations" and in Note 13 to the consolidated financial statements of Verizon Communications Inc. and Subsidiaries, which are incorporated by reference into this report.

Service and Product Offerings

Our Consumer segment's wireless and wireline products and services are available to our retail customers, as well as resellers that purchase wireless network access from us on a wholesale basis. Our Business segment's wireless and wireline products and services are organized by the primary customer groups targeted by these offerings: Global Enterprise, Small and Medium Business, Public Sector and Other, and Wholesale.

Wireless

We offer wireless services and equipment to customers of both Consumer and Business.

Wireless Services

Our Consumer and Business segments provide a wide variety of wireless services accessible on a broad range of devices. Customers can obtain our wireless services on a postpaid or prepaid basis. Retail (non-wholesale) postpaid accounts primarily represent retail customers that are directly served and managed by Verizon and use Verizon branded services. A single account may include monthly wireless services for a variety of connected devices. A retail postpaid connection represents an individual line of service for a wireless device for which a customer is generally billed one month in advance for a monthly access charge in return for access to and usage of network services. Our prepaid service is offered only to Consumer customers and enables individuals to obtain wireless services without credit verification by paying for all services in advance. Approximately 96% of our Consumer retail connections were postpaid connections as of December 31, 2019.

We offer various postpaid and prepaid service plans tailored to the needs of our customers. Depending on those needs at a particular time, our plans may include features related to, among other things: unlimited or metered domestic and/or international voice, data, and texting; the ability to share data allowances and/or use data allowances in different periods; high definition voice and video features; the ability to use a device as a Wi-Fi hotspot; and varying data rates depending on the plan and usage on that plan. Our service offerings vary from time to time based on customer needs, technology changes and market conditions and may be provided as standard plans or as part of limited time promotional offers.

Access to the Internet is available on all smartphones and nearly all basic phones. In addition, our customers can access the Internet at broadband speeds on notebook computers and tablets that are either wireless-enabled or that are used in conjunction with separate dedicated devices that provide a mobile Wi-Fi connection.

As of January 2017, we no longer offer Consumer customers new fixed-term, subsidized service plans for phones; however, we continue to offer subsidized plans to our Business customers. We also continue to service existing plans for customers who have not yet purchased and activated devices under the Verizon device payment program.

Wireless Equipment

Consumer and Business offer several categories of wireless equipment to customers, including a variety of smartphones and other handsets, wireless-enabled Internet devices, such as tablets, laptop computers and netbooks, and other wireless-enabled connected devices, such as smart watches and other wearables. In certain cases, we permit customers to acquire equipment from us using device payment plans, which permit the customer to pay for the device in installments over time.

Verizon Consumer Group

In addition to the wireless services and equipment discussed above, Consumer sells residential fixed connectivity solutions, including Internet, video and voice services, and wireless network access to resellers on a wholesale basis.

Residential Fixed Services. We provide residential fixed connectivity solutions to customers over our 100% fiber-optic network under the Fios brand, and over a traditional copper-based network to customers who are not served by Fios. During 2018, we commercially launched fifth-generation (5G) wireless technology for the home (5G Home) on proprietary standards in four U.S. markets and on global standards in a fifth market in 2019.

We offer residential fixed services tailored to the needs of our customers. Depending on those needs at a particular time, our services may include features related to, among other things: Internet access at different speed tiers using fiber-optic, copper or wireless technology; video services that may feature a variety of channel options, video on demand products, cloud-based services and digital video recording capabilities; over-the-top video services; and voice services.

Network Access Services. We sell network access to mobile virtual network operators (MVNOs) on a wholesale basis, who in turn resell wireless service under their own brand(s) to consumers.

Verizon Business Group

In addition to the wireless services and equipment discussed above, our Business segment provides communications products and enhanced services, including video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various IoT products and services.

Global Enterprise

Global Enterprise offers services to large businesses, which are identified based on their size and volume of business with Verizon, as well as non-U.S. public sector customers. In 2019, Global Enterprise revenues were $10.8 billion, representing approximately 34% of Business's total revenues.

Global Enterprise offers a broad portfolio of connectivity, security and professional services designed to enable our customers to optimize their business operations, mitigate business risks and capitalize on data. These services include the following:

- *Network Services*. We offer a portfolio of network connectivity products to help our customers connect with their employees, partners, vendors, and customers. These products include private networking services, private cloud connectivity services, virtual and software defined networking services, and Internet access services.
- *Advanced Communications Services*. We offer a suite of services to our customers to help them communicate with their employees, partners, vendors, constituents and customers. These products include Internet Protocol (IP)-based voice services, unified communications and collaboration tools and customer contact center solutions.
- *Security services*. We offer a suite of management and data security services that help our customers protect, detect and respond to security threats to their networks, data, applications and infrastructure.
- *Core services*. We provide a portfolio of domestic and global voice and data solutions utilizing traditional telecommunications technology, including voice calling, messaging services, conferencing, contact center solutions and private line and data access networks. Core services also include the provision of customer premises equipment, and installation, maintenance and site services.

- *IoT services*. We provide the network access required to deliver various IoT products and services. We work with companies that purchase network access from us to connect their Open Development-certified devices, bundled together with their own solutions, which they sell to end users. We are building IoT capabilities by leveraging business models that monetize usage on our networks at the connectivity, platform and solution layers.

Small and Medium Business

Small and Medium Business offers wireless services and equipment, tailored voice and networking products, Fios services, IP networking, advanced voice solutions, security and managed information technology (IT) services to our U.S.-based customers that do not meet the requirements to be categorized as Global Enterprise. In 2019, Small and Medium Business revenues were $11.5 billion, representing approximately 36% of Business's total revenues.

In addition to the wireless services and equipment discussed above, Small and Medium Business provides fixed connectivity solutions comparable to the residential fixed services provided by Consumer, as well as business services and connectivity similar to the products and services offered by Global Enterprise, in each case with features and pricing designed to address the needs of small and medium businesses.

Public Sector and Other

Public Sector and Other offers wireless products and services as well as wireline connectivity and managed solutions to U.S. federal, state and local governments and educational institutions. These services include the business services and connectivity similar to the products and services offered by Global Enterprise, in each case, with features and pricing designed to address the needs of governments and educational institutions. In 2019, Public Sector and Other revenues were $5.9 billion, representing approximately 19% of Business's total revenues.

Public Sector and Other also includes solutions that support fleet tracking management, compliance management, field service management, asset tracking and other types of mobile resource management in the United States and around the world.

Wholesale

Wholesale offers wireline communications services including data, voice, local dial tone and broadband services primarily to local, long distance, and wireless carriers that use our facilities to provide services to their customers. In 2019, Wholesale revenues were $3.2 billion, representing approximately 10% of Business's total revenues. A portion of Wholesale revenues are generated by a few large telecommunications companies, most of which compete directly with us. Wholesale's services include:

- *Data services*. We offer a portfolio of data services with varying speeds and options to enhance our Wholesale customers' networks and provide connections to their end-users and subscribers.

- *Voice services*. We provide switched access services that allow carriers to complete their end-user calls that originate or terminate within our territory. In addition, we provide originating and terminating voice services throughout the U.S. and globally utilizing our TDM and VoIP networks.

- *Local services.* We offer an array of local dial tone and broadband services to competitive local exchange carriers, some of which are offered to comply with telecommunications regulations. In addition, we offer services such as colocation, resale and unbundled network elements in compliance with applicable regulations.

Distribution

We use a combination of direct, indirect and alternative distribution channels to market and distribute our products and services to Consumer customers.

Our direct channel, including our company-operated stores, is a core component of our distribution strategy. Our sales and service centers also represent a significant distribution channel for our services.

Our indirect/digital partners channel includes agents that sell our wireless and wireline products and services at retail locations throughout the U.S., as well as through the Internet. The majority of these sales are made under exclusive selling arrangements with us. We also have relationships with high-profile national retailers that sell our wireless and wireline products and services, as well as convenience store chains that sell our wireless prepaid products and services. In 2019, we grew our digital channel and expanded omni channel experiences for our customers to offer choice and convenience.

In addition to our direct channel, our Business segment has additional distribution channels that include business solution fulfillment provided by resellers, non-stocked device fulfillment performed by distributors and integrated mobility services provided by system integrators and resellers.

Competition and Related Trends

The telecommunications industry is highly competitive. We expect competition to remain intense as traditional and non-traditional participants seek increased market share.

With respect to our wireless connectivity products and services, we compete against other national wireless service providers, including AT&T Inc., Sprint Corporation and T-Mobile USA, Inc., as well as various regional wireless service providers. We also compete for retail activations with resellers that buy bulk wholesale service from wireless service providers, including Verizon, and resell it to their customers. Resellers may include cable companies. Competition remains intense as a result of high rates of smartphone penetration in the wireless market, increased network investment by our competitors, the development and deployment of new technologies, such as 5G, the introduction of new products and services, offerings that include additional premium content, new market entrants, the availability of additional licensed and unlicensed spectrum, and regulatory changes. In 2019, we began offering Apple Music and Disney+ to customers as part of their Unlimited plans. Competition may also increase as smaller, stand-alone wireless service providers merge or transfer licenses to larger, better capitalized wireless service providers and as MVNOs resell wireless communication services.

We also face competition from other communications and technology companies seeking to increase their brand recognition and capture customer revenue with respect to the provision of wireless products and services, in addition to non-traditional offerings in mobile data. For example, Microsoft Corporation, Alphabet Inc., Apple Inc. and others are offering alternative means for making wireless voice calls that, in certain cases, can be used in lieu of the wireless provider's voice service, as well as alternative means of accessing video content.

With respect to our wireline connectivity services, we compete against cable companies, wireless service providers, domestic and foreign telecommunications providers, satellite television companies, Internet service providers, over-the-top (OTT) providers and other companies that offer network services and managed enterprise solutions. Cable operators have increased the size and capacity of their networks in order to deliver digital products and services. We introduced offerings that provide customized Internet and video packages. Several major cable operators also offer bundles with wireless services through strategic relationships. Customers have more choices for obtaining video content from various online services. We expect the market will continue to shift from traditional linear video to OTT offerings. We expect customer migration from traditional voice services to wireless services to continue as a growing number of customers place greater value on mobility and wireless companies position their services as a landline alternative. We also face increasing competition from cable operators and other providers of VoIP services, as well as Internet portal providers.

We believe that the following are the most important competitive factors and trends in the telecommunications industry:

- *Network reliability, speed and coverage.* We consider networks that consistently provide high-quality, fast and reliable service to be a key differentiator in the market and driver of customer satisfaction. Lower prices, improved service quality and new service offerings, which in many cases include video content, have led to increased customer usage of connectivity services. We and other network-based providers must ensure that our networks can meet these increasing capacity usage requirements and offer highly reliable national coverage.

- *Pricing.* With respect to wireless services and equipment, pricing plays an important role in the wireless competitive landscape. As the demand for wireless services continues to grow, wireless service providers are offering a range of service plans at competitive prices. Many wireless service providers also bundle wireless service offerings with other products and offer promotional pricing and incentives, some of which may be targeted specifically to customers of Verizon. We and other wireless service providers, as well as equipment manufacturers, offer device payment options, which provide customers with the ability to pay for their device over a period of time, and some providers offer device leasing arrangements. In addition, aggressive device promotions have become more common in an effort to gain a greater share of subscribers interested in changing carriers. With respect to wireline services, pricing is used by competitors to capture market share from incumbents, and it is a significant factor as non-traditional modes of providing communication services emerge and new entrants compete for customers. For example, VoIP and portal-based voice and video calling is often free or nearly free to customers and is often supported by advertising revenues.

- *Customer service.* We believe that high-quality customer service is a key factor in retaining customers and attracting new customers, including those of other providers. Our customer service, retention and satisfaction programs are based on providing customers with convenient and easy-to-use products and services and focusing on their needs in order to promote long-term relationships and minimize churn. The Verizon Up program, for example, was launched to promote long-term relationships with our Consumer customers. The program offers a variety of rewards to customers in exchange for points in connection with their account-related transactions.

  Customer service is highly valued by our Business customers. We provide Global Enterprise and Public Sector and Other customers with ready access to their system and performance information, and we conduct proactive testing of our networks to identify issues before they affect our customers. We service our Small and Medium Business customers through service representatives and online support, as well as through store-based representatives for small business customers. For Wholesale customers, we pursue service improvement through continued system automation initiatives.

- *Product differentiation.* Customer and revenue growth are increasingly dependent on the development of new and enhanced products and services, as the delivery of new and innovative products and services has been accelerating. Customers are shifting their focus from access to applications and are seeking ways to leverage their broadband, video and wireless connections. To compete effectively, providers need to continuously review, improve and refine their product portfolio and develop and rapidly deploy new products and services tailored to the needs of customers. We continue to pursue the development and rapid deployment of new and innovative products and services, both independently and in collaboration with application providers, content providers and device manufacturers. Features such as wireless and wireline inter-operability are becoming increasingly important, driven by both customer demand and technological advancement.

- *Sales and distribution.* A key to achieving sales success in the consumer and small and medium business sectors of the wireless industry is the reach and quality of sales channels and distribution points. We seek to optimally vary distribution channels among our company-operated stores selling wireless products and services, outside sales teams and telemarketing, web-based sales and fulfillment capabilities, our extensive indirect distribution network of retail outlets and our sale of wireless service to resellers, which resell wireless services to their end-users.

In addition to these competitive factors and trends, companies with a global presence are increasingly competing with us in our Business segment. A relatively small number of telecommunications and integrated service providers with global operations serve customers in the global enterprise market and, to a lesser extent, the global wholesale market. We compete with these providers for large contracts to provide integrated solutions to global enterprises. Many of these companies have strong market presence, brand recognition and existing customer relationships, all of which contribute to intensifying competition that may affect our future revenue growth.

In the Global Enterprise and Public Sector and Other markets, competition remains high, primarily as a result of increased industry focus on technology convergence. We compete in this area with system integrators, carriers, and hardware and software providers. In addition, some of the largest information technology services companies are making strategic acquisitions, divesting non-strategic assets and forging new alliances to improve their cost structure. Many new alliances and acquisitions have focused on emerging fields, such as cloud computing, software defined network, communication applications and other computing tasks via networks, rather than by the use of in-house machines.

In the Small and Medium Business market, customer purchasing behaviors and preferences continue to evolve. Solution speed and simplicity with user interfaces that have a consumer-like "look and feel" are becoming key differentiators for customers who are seeking full life-cycle offers that simplify the process of starting, running and growing their businesses. Several major cable operators also offer bundles with wireless services through strategic relationships.

Our Wholesale business competes with traditional carriers for long-haul, voice and IP services. In addition, mobile video and data needs are driving a greater need for wireless backhaul. Network providers, cable companies and niche players are competitors for this business opportunity.

Verizon Media

Our media business, Verizon Media, includes diverse media and technology brands that serve both consumers and businesses. Verizon Media provides consumers with owned and operated and third-party search properties as well as mail, news, finance, sports and entertainment offerings, and provides other businesses and partners access to consumers through digital advertising, content delivery and video streaming platforms. In 2019, Verizon Media's revenues were $7.5 billion.

Verizon Media Products and Solutions

Ad Platform

Our Verizon Media Ad Platform provides advertisers and publishers with a simplified suite of intelligent advertising solutions across desktop, mobile and television devices. Verizon Media's business is comprised primarily of search advertising, display advertising, Ecommerce and subscriptions.

- *Search advertising*. Our search properties serve as a guide for users to discover information on the Internet. Verizon Media serves click-based search advertisements generated by proprietary algorithmic technology, as well as advertisements from partners. Verizon Media provides the underlying search products that facilitate user searches within Verizon Media and third-party partner properties.

- *Display advertising.* Display advertising is made up of both graphical and performance-based advertising and takes the form of impression-based contracts, time-based contracts and performance-based contracts. Verizon Media display ads leverage proprietary data signals to identify and engage users on Verizon Media properties and across the web. Through Verizon Media Ad Platform, we provide customers the ability to buy advertising inventory, measure campaigns across screens and advertising formats using self-serve technology or our managed services. We also provide publishers with the ability to monetize their ad inventory.

- *Ecommerce.* Our Ecommerce offering includes different types of business models, including facilitating transactions between businesses and consumers, enabling businesses that facilitate transactions for other businesses, and facilitating transactions between consumers.

- *Subscription memberships.* Our paid subscription offerings include premium content and services across our mail, news, finance, sports and entertainment properties, privacy and security solutions and computer protection.

Verizon Media Platform

As the digital platform reshapes the delivery of media and entertainment content, there is an increasing need for stable, high-quality video delivery platforms. Our Media Platform offers a scalable platform for delivering content, including live broadcasts, video on demand, games, software and websites to our customers on their devices at any time. This platform is targeted at media and entertainment companies and other businesses that deliver their digital products and services through the Internet.

Global Network and Technology

Our global network architecture is used by both Consumer and Business. Our network technology platforms include both wireless and wireline technologies.

Network Evolution

We are evolving the architecture of our networks to a next-generation multi-use platform, providing improved efficiency and virtualization, increased automation and opportunities for edge computing services that will support both our fiber-based and radio access network technologies. We call this the Intelligent Edge Network. We expect that this new architecture will simplify operations by eliminating legacy network elements, improve our fourth-generation (4G) Long-Term Evolution (LTE) wireless coverage, speed the deployment of 5G wireless technology and create new opportunities in the business market.

5G Deployment

Over the past several years, we have been leading the development of 5G wireless technology industry standards and the ecosystems for fixed and mobile 5G wireless services. We believe 5G technology will be able to provide users with eight capabilities, or currencies. The eight currencies are peak data rates, mobile data volumes, mobility, number of connected devices, energy efficiency of connected devices, service deployment, reduced latency and improved reliability. We expect that 5G technology will provide higher throughput and lower latency than the current 4G LTE technology and enable our networks to handle more traffic as the number of Internet-connected devices grows. During 2018, we commercially launched 5G Home on proprietary standards in four U.S. markets and on global standards in a fifth U.S. market in 2019. We also launched our 5G Ultra Wideband Network in 31 U.S. markets in 2019, as well as several 5G-compatible smartphones.

4G LTE

Our primary wireless network technology platform is 4G LTE, which provides higher data throughput performance for data services at a lower cost compared to that offered by 3G technology. As of December 31, 2019, our 4G LTE network is available in over 700 markets covering approximately 327 million people, including those in areas served by our LTE in Rural America partners. Under this program, we have collaborated with wireless carriers in rural areas to build and operate a 4G LTE network using each carrier's network assets and our core 4G LTE equipment and 700 Megahertz (MHz) C Block and Advanced Wireless Services (AWS) spectrum.

Wireless Network Reliability and Build-Out

We consider the reliability, coverage and speed of our wireless network as key factors for our continued success. We believe that steady and consistent network and platform investments provide the foundation for innovative products and services. As we design and deploy our network, we focus on the number of successful data sessions the network enables, delivering on our advertised throughput speeds, and the number of calls that are connected on the first attempt and completed without being dropped. We utilize three strategies to maintain the quality of our network: increasing the density of our network elements, deploying new technologies as they are developed and putting additional wireless spectrum into service. We choose among these strategies based on the circumstances present at different times in each of our service areas.

We have been densifying our network by utilizing small cell technology, in-building solutions and distributed antenna systems. Network densification enables us to add capacity to address increasing mobile video consumption and the growing demand for IoT products and services on our 4G LTE and 5G networks. We are also utilizing existing network capabilities to handle increased traffic without interrupting the quality of the customer experience. We continue to deploy advanced technologies to increase both network capacity and data rates.

In order to build and upgrade our existing 4G LTE network and deploy our 5G network, we must complete a variety of steps, which can include securing rights to a large number of sites as well as obtaining zoning and other governmental approvals and fiber facilities, for our macro and small cells, in-building systems and antennas and related radio equipment that comprise distributed antenna systems. We have relationships with a wide variety of vendors that supply various products and services that support our wireless network operations. We utilize tower site management firms as lessors or managers of a portion of our existing leased and owned tower sites.

Our networks include various elements of redundancy designed to enhance the reliability of the services provided to our customers. To mitigate the impact of power disruptions on our operations, we have battery backup at every switch and every macro cell. We also utilize backup generators at a majority of our macro cells and at every switch location. In addition, we have a fleet of portable backup generators that can be deployed, if needed. We further enhance reliability by using a fully redundant Multiprotocol Label Switching backbone network in critical locations.

In addition to our own network coverage, we have roaming agreements with a number of wireless service providers to enable our customers to receive wireless service in nearly all other areas in the U.S. where wireless service is available. We also offer a variety of international wireless voice and data services to our customers through roaming arrangements with wireless service providers outside the U.S.

Fios

Residential broadband service has seen significant growth in bandwidth demand over the past several years, and we believe that demand will continue to grow. We expect the continued emergence of new video services, new data applications and the proliferation of IP devices in the home will continue to drive new network requirements for increased data speeds and throughput. We believe that the Passive Optical Network (PON) technology underpinning Fios positions us well to meet these demands in a cost-effective and efficient manner.

While deployed initially as a consumer broadband network, our PON infrastructure is also experiencing more widespread application in the Business segment, especially as businesses increasingly migrate to Ethernet-based access services.

Global IP

Verizon owns and operates one of the largest global fiber-optic networks in the world, providing connectivity to Business customers in more than 150 countries. Our global IP network includes long-haul, metro and submarine assets that span over 1 million route miles and enable and support international operations.

Global business is rapidly evolving to an "everything-as-a-service" model in which Business customers seek cloud-based, converged enterprise solutions delivered securely via managed and professional services. We are continuing to deploy packet optical transport technology in order to create a global network platform to meet this demand.

Spectrum

The spectrum licenses we hold can be used for mobile wireless voice, video and data communications services. We are licensed by the Federal Communications Commission (FCC) to provide these wireless services on portions of the 800 MHz band, also known as cellular spectrum, the 1800-1900 MHz band, also known as Personal Communication Services (PCS) spectrum, portions of the 700 MHz upper C band and AWS 1 and 3 spectrum in the 1700 and 2100 MHz bands, in areas that, collectively, cover nearly all of the population of the U.S. This spectrum is collectively called low and mid-band spectrum. We are using our low and mid-band spectrum to provide both 3G and 4G LTE wireless services. However, we are increasingly reallocating spectrum previously used for 3G service to provide 4G LTE service. We are also repurposing low and mid-band spectrum to complement our spectrum licenses in the 28 and 39 Gigahertz (GHz) band, collectively called millimeter wave spectrum.

Millimeter wave spectrum is being used for our 5G technology deployment. We anticipate that we will need additional spectrum to meet future demand. This increasing demand is driven by growth in customer connections and the increased usage of wireless broadband services that use more bandwidth and require faster rates of speed, as well as the wider deployment of 5G mobile and fixed services. We can meet our future 4G and 5G spectrum needs by acquiring licenses or leasing spectrum from other licensees, or by acquiring new spectrum licenses from the FCC, if and when future FCC spectrum auctions occur.

From time to time we have exchanged spectrum licenses with other wireless service providers through secondary market swap transactions. We expect to continue to pursue similar opportunities to trade spectrum licenses in order to meet capacity and expansion needs in the future. In certain cases, we have entered into intra-market spectrum swaps designed to increase the amount of contiguous spectrum within frequency bands in a specific market. Contiguous spectrum improves network performance and efficiency. These swaps, as well as any spectrum purchases, require us to obtain governmental approvals.

Information regarding spectrum license transactions is included in the 2019 Verizon Annual Report to Shareholders in Note 3 to the consolidated financial statements of Verizon Communications Inc. and Subsidiaries, which is incorporated by reference into this report.

Strategic Transactions

During March 2015, we completed a transaction with American Tower Corporation (American Tower) pursuant to which American Tower acquired the exclusive rights to lease and operate approximately 11,300 of our wireless towers and corresponding ground leases for an upfront payment of $5.0 billion. We have subleased capacity on the towers from American Tower for a minimum of 10 years at current market rates, with options to renew. Under the terms of the lease agreements, American Tower has exclusive rights to lease and operate towers over an average term of approximately 28 years. As the leases expire, American Tower has fixed-price purchase options to acquire these towers based on their anticipated fair market values at the end of the lease terms. As part of this transaction, we also sold 162 towers for $71 million.

In June 2015, we completed our acquisition of AOL Inc. (AOL), a leader in digital content and advertising. The aggregate cash consideration paid by Verizon at the closing was approximately $3.8 billion.

In April 2016, we completed the sale (Access Line Sale) of our local exchange business and related landline activities in California, Florida and Texas, including Fios Internet and video customers, switched and special access lines and high-speed Internet service and long distance voice accounts in these three states to Frontier Communications Corporation (Frontier) for approximately $10.5 billion (approximately $7.3 billion net of income taxes), subject to certain adjustments and including the assumption of $612 million of indebtedness from Verizon by Frontier. The transaction included the acquisition by Frontier of the equity interests of Verizon's incumbent local exchange carriers (ILECs) in California, Florida and Texas.

The transaction resulted in Frontier acquiring approximately 3.3 million voice connections, 1.6 million Fios Internet subscribers, 1.2 million Fios video subscribers and the related ILEC businesses from Verizon. Approximately 9,300 Verizon employees who served customers in California, Florida and Texas continued employment with Frontier.

In July 2016, we acquired Telogis, Inc., a global, cloud-based mobile enterprise management software business, for $877 million of cash consideration.

In November 2016, we completed the acquisition of Fleetmatics Group PLC, a global provider of fleet and mobile workforce management solutions, for $60.00 per ordinary share in cash. The aggregate merger consideration was approximately $2.5 billion, including cash acquired of $112 million.

In December 2016, we entered into a definitive agreement, which was subsequently amended in March 2017, with Equinix, Inc. (Equinix) pursuant to which we agreed to sell 23 customer-facing data center sites in the U.S. and Latin America for approximately $3.6 billion, subject to certain adjustments (Data Center Sale). The transaction closed in May 2017.

In February 2016, we entered into a purchase agreement to acquire XO Holdings' wireline business (XO), which owned and operated one of the largest fiber-based IP and Ethernet networks in the U.S. Concurrently, we entered into a separate agreement to utilize certain wireless spectrum from a wholly-owned subsidiary of XO Holdings, NextLink, that held XO's millimeter-wave wireless spectrum. The agreement included an option, subject to certain conditions, to acquire NextLink. In February 2017, we completed our acquisition of XO for total cash consideration of approximately $1.5 billion, of which $100 million was paid in 2015.

In April 2017, we exercised our option to buy NextLink for approximately $493 million, subject to certain adjustments, of which $320 million was prepaid in the first quarter of 2017. The transaction closed in January 2018. The acquisition of NextLink was accounted for as an asset acquisition, as substantially all of the value related to the acquired spectrum. Upon closing, we recorded approximately $657 million of wireless licenses, $110 million of a deferred tax liability and $58 million of other liabilities.

In June 2017, we completed our acquisition of the operating business of Yahoo! Inc. (Yahoo), a leader in search, communications, digital content and advertising. Pursuant to the Purchase Agreement, upon the terms and subject to the conditions thereof, we agreed to acquire the stock of one or more subsidiaries of Yahoo holding all of Yahoo's operating business for approximately $4.83 billion in cash, subject to certain adjustments. In February 2017, Verizon and Yahoo entered into an amendment to the Purchase Agreement, pursuant to which the Transaction purchase price was reduced by $350 million to approximately $4.48 billion in cash, subject to certain adjustments.

In August 2017, we entered into a definitive agreement to purchase certain fiber-optic network assets in the Chicago market from WideOpenWest, Inc. (WOW!), a leading provider of communications services. The transaction closed in December 2017. In addition, the parties entered into a separate agreement pursuant to which WOW! was to complete the build-out of the network assets in 2019. This build-out was completed in 2019. The total cash consideration for the transactions was approximately $275 million, of which $226 million was paid in December 2017. During 2019 and 2018, the remaining cash consideration was paid.

In 2017, we entered into a transaction to acquire Straight Path Communications Inc., which held certain millimeter-wave spectrum licenses. The transaction closed in February 2018 for total consideration reflecting an enterprise value of approximately $3.1 billion. We are using the spectrum acquired for our 5G technology deployment.

Additional information regarding certain of these strategic transactions is included in the 2019 Verizon Annual Report to Shareholders in Note 3 to the consolidated financial statements of Verizon Communications Inc. and Subsidiaries, which is incorporated by reference into this report.

Patents, Trademarks and Licenses

We own or have licenses to various patents, copyrights, trademarks, domain names and other intellectual property rights necessary to conduct our business. We actively pursue the filing and registration of patents, copyrights, domain names, trademarks and service marks to protect our intellectual property rights within the United States and abroad. We also actively grant licenses, in exchange for appropriate fees or other consideration and subject to appropriate safeguards and restrictions, to other companies that enable them to utilize certain of our intellectual property rights and proprietary technology as part of their products and services. Such licenses enable the licensees to take advantage of the results of Verizon's research and development efforts. While these licenses result in valuable consideration for Verizon, we do not believe that the loss of such consideration, or the expiration of any of our intellectual property rights, would have a material effect on our results of operations.

We periodically receive offers from third parties to purchase or obtain licenses for patents and other intellectual property rights in exchange for royalties or other payments. We also periodically receive notices alleging that our products or services infringe on third-party patents or other intellectual property rights. These claims, whether against us directly or against third-party suppliers of products or services that we sell to our customers, if successful, could require us to pay damages or royalties, or cease offering the relevant products or services.

Acquisitions and Divestitures

Information about our acquisitions and divestitures is included in the 2019 Verizon Annual Report to Shareholders under the heading "Management's Discussion and Analysis of Financial Condition and Results of Operations - Acquisitions and Divestitures" and in Note 3 to the consolidated financial statements of Verizon Communications Inc. and subsidiaries, which is incorporated by reference into this report.

Regulatory and Competitive Trends

Regulatory and Competitive Landscape

Verizon operates in a regulated and highly competitive market. Current and potential competitors include other voice and data service providers, such as other wireless companies, traditional telephone companies, cable companies, Internet service providers, software and application providers and other non-traditional competitors. Many of these companies have strong market presence, brand recognition and existing customer relationships, all of which contribute to a highly competitive market that may affect our future revenue growth. Some of our competitors also are subject to fewer regulatory constraints than Verizon. For many services offered by Verizon, the FCC is our primary regulator. The FCC has jurisdiction over interstate telecommunications services and other matters under the Communications Act of 1934, as amended (Communications Act or Act). Other Verizon services are subject to state and local regulation.

Federal, State and Local Regulation

Wireless Services

The FCC regulates several aspects of our wireless operations. Generally, the FCC has jurisdiction over the construction, operation, acquisition and transfer of wireless communications systems. All wireless services require use of radio frequency spectrum, the assignment and distribution of which is subject to FCC oversight. Verizon anticipates that it will need additional spectrum to meet future demand. We can meet our needs for licensed spectrum by purchasing licenses or leasing spectrum from others, or by participating in a competitive bidding process to acquire new spectrum from the FCC. Those processes are subject to certain reviews, approvals and potential conditions.

Today, Verizon holds FCC spectrum licenses that allow it to provide a wide range of mobile and fixed communications services, including both voice and data services. FCC spectrum licenses typically have a term of 10 years, at which time they are subject to renewal. While the FCC has routinely renewed all of Verizon's wireless licenses, challenges could be raised in the future. If a wireless license was revoked or not renewed, Verizon would not be permitted to provide services on the spectrum covered by that license. Some of our licenses require us to comply with so-called "open access" FCC regulations, which generally require licensees of particular spectrum to allow customers to use devices and applications of their choice, subject to certain technical limitations. The FCC has also imposed certain specific mandates on wireless carriers, including construction and geographic coverage requirements, technical operating standards, provision of enhanced 911 services, roaming obligations and requirements for wireless tower and antenna facilities.

The Act generally preempts regulation by state and local governments of the entry of, or the rates charged by, wireless carriers. The Act does not prohibit states from regulating the other "terms and conditions" of wireless service. For example, some states attempt to regulate wireless customer billing matters and impose reporting requirements. Several states also have laws or regulations that address safety issues (e.g., use of wireless handsets while driving) and taxation matters. In addition, wireless tower and antenna facilities are often subject to state and local zoning and land use regulation, and securing approvals for new or modified facilities is often a lengthy and expensive process.

Broadband

Verizon offers many different broadband services. Traditionally, the FCC recognized broadband Internet access services as "information services" subject to a "light touch" regulatory approach rather than to the traditional, utilities-style regulations. In 2015, the FCC declared that broadband Internet access services are "telecommunications services" subject to common carriage regulation under Title II of the Communications Act. In December 2017, the FCC adopted an order reversing the 2015 Title II Order to return to "light touch" regulation of broadband Internet access services. The "light touch" portions of this order have been upheld by the U.S. Court of Appeals for the D.C. Circuit but further appeals are likely. The part of the FCC order automatically preempting state action on the subject was vacated, and a number of states are likely to join those that have taken steps to regulate broadband. Regardless of regulation, Verizon remains committed to the open Internet, which provides consumers with competitive choices and unblocked access to lawful websites and content, and our commitment to our customers can be found on our website at http://responsibility.verizon.com/broadband-commitment.

Wireline Voice

Verizon offers many different wireline voice services, including traditional telephone service and other services that rely on technologies such as VoIP. For regulatory purposes, legacy telephone services are generally considered to be "common carrier" services. Common carrier services are subject to heightened regulatory oversight with respect to rates, terms and conditions and other aspects of the services. The FCC has not decided the regulatory classification of VoIP but has said VoIP service providers must comply with certain rules, such as 911 capabilities and law enforcement assistance requirements.

State public utility commissions regulate Verizon's telephone operations with respect to certain telecommunications intrastate matters. Verizon operates as an "incumbent local exchange carrier" in nine states and the District of Columbia. These incumbent operations are subject to various levels of pricing flexibility and other state oversight and requirements. Verizon also has other wireline operations that are more lightly regulated.

Video

Verizon offers a multichannel video service that is regulated like traditional cable service. The FCC has a body of rules that apply to cable operators, and these rules also generally apply to Verizon. In areas where Verizon offers its facilities-based multichannel video services, Verizon has been required to obtain a cable franchise from local government entities, or in some cases a state-wide franchise, and comply with certain one-time and ongoing obligations as a result.

Privacy and Data Security

We are subject to federal, state and international laws and regulations relating to privacy and data security that impact all parts of our business, including wireline, wireless, broadband and the development and roll out of new products, such as those in the media and IoT space. At the federal level, our voice business is subject to the FCC's privacy requirements. Oversight of broadband Internet access privacy and data security is governed by the Federal Trade Commission (FTC). Generally, attention to privacy and data security requirements is increasing at both the state and federal level, and several privacy-related bills have been introduced or are under considerations at each level. Europe's General Data Protection Regulation, which went into effect in May 2018, includes significant penalties for non-compliance. In addition, a new privacy law took effect in California at the beginning of 2020, an additional privacy law is scheduled to take effect in Maine in 2020, and other states are considering additional regulations. These regulations could have a significant impact on our businesses.

Public Safety and Cybersecurity

The FCC plays a role in addressing public safety concerns by regulating emergency communications services and mandating widespread availability of both media (broadcast/cable) and wireless emergency alerting services. In response to cyber attacks that have occurred or could occur in the future, however, the FCC or other regulators may attempt to increase regulation of the cybersecurity practices of providers. The FCC is also addressing the use by American companies of equipment produced by companies imposing potential national security risks, but Verizon does not use such equipment in its network. In addition, due to recent natural disasters, federal and state agencies may attempt to impose regulations to ensure continuity of service during disasters.

Intercarrier Compensation and Network Access

The FCC regulates some of the rates that carriers pay each other for the exchange of voice traffic (particularly traditional wireline traffic) over different networks and other aspects of interconnection for some voice services. The FCC also regulates some of the rates and terms and conditions for certain wireline "business data services" and other services and network facilities. Verizon is both a seller and a buyer of these services, and both makes and receives interconnection payments. The FCC has focused in recent years on whether changes in the rates, terms and conditions for both the exchange of traffic and for business data services may be appropriate.

Information About Our Executive Officers

See Part III, Item 10. "Directors, Executive Officers and Corporate Governance" of this Annual Report on Form 10-K for information about our executive officers.

Employees

As of December 31, 2019, Verizon and its subsidiaries had approximately 135,000 employees. Labor unions represent approximately 23% of our employees.

Information on Our Internet Website

We make available, free of charge on our website, our annual reports on Form 10-K, quarterly reports on Form 10-Q, current reports on Form 8-K, and all amendments to those reports at http://www.verizon.com/about/investors as soon as reasonably practicable after such reports are electronically filed with the Securities and Exchange Commission (SEC). These reports and other information are also available on the SEC's website at www.sec.gov. We periodically provide other information for investors on this website, as well, including news and announcements regarding our financial performance, information on corporate governance and details related to our annual meeting of shareholders. We encourage investors, the media, our customers, business partners and other stakeholders to review the information we post on this channel. Website references in this report are provided as a convenience and do not constitute, and should not be viewed as, incorporation by reference of the information contained on, or available through, the websites. Therefore, such information should not be considered part of this report.

Cautionary Statement Concerning Forward-Looking Statements

In this report we have made forward-looking statements. These statements are based on our estimates and assumptions and are subject to risks and uncertainties. Forward-looking statements include the information concerning our possible or assumed future results of operations. Forward-looking statements also include those preceded or followed by the words "anticipates," "believes," "estimates," "expects," "hopes" or similar expressions. For those statements, we claim the protection of the safe harbor for forward-looking statements contained in the Private Securities Litigation Reform Act of 1995. We undertake no obligation to revise or publicly release the results of any revision to these forward-looking statements, except as required by law. Given these risks and uncertainties, readers are cautioned not to place undue reliance on such forward-looking statements.

The following important factors, along with those discussed elsewhere in this report and in other filings with the SEC, could affect future results and could cause those results to differ materially from those expressed in the forward-looking statements:

- cyber attacks impacting our networks or systems and any resulting financial or reputational impact;
- natural disasters, terrorist attacks or acts of war or significant litigation and any resulting financial or reputational impact;
- disruption of our key suppliers’ or vendors' provisioning of products or services;
- material adverse changes in labor matters and any resulting financial or operational impact;

- the effects of competition in the markets in which we operate;
- failure to take advantage of developments in technology and address changes in consumer demand;
- performance issues or delays in the deployment of our 5G network resulting in significant costs or a reduction in the anticipated benefits of the enhancement to our networks;
- the inability to implement our business strategy;
- adverse conditions in the U.S. and international economies;
- changes in the regulatory environment in which we operate, including any increase in restrictions on our ability to operate our networks;
- our high level of indebtedness;
- an adverse change in the ratings afforded our debt securities by nationally accredited ratings organizations or adverse conditions in the credit markets affecting the cost, including interest rates, and/or availability of further financing;
- significant increases in benefit plan costs or lower investment returns on plan assets;
- changes in tax laws or treaties, or in their interpretation; and
- changes in accounting assumptions that regulatory agencies, including the SEC, may require or that result from changes in the accounting rules or their application, which could result in an impact on earnings.

Item 1A. Risk Factors

The following discussion of "Risk Factors" identifies the most significant factors that may adversely affect our business, operations, financial condition or future performance. This information should be read in conjunction with "Management's Discussion and Analysis of Financial Condition and Result of Operations" and the consolidated financial statements and related notes. The following discussion of risks is not all-inclusive but is designed to highlight what we believe are important factors to consider when evaluating our business and expectations. These factors could cause our future results to differ materially from our historical results and from expectations reflected in forward-looking statements.

Operational Risks

Cyber attacks impacting our networks or systems could have an adverse effect on our business.

Cyber attacks, including through the use of malware, computer viruses, dedicated denial of services attacks, credential harvesting, social engineering and other means for obtaining unauthorized access to or disrupting the operation of our networks and systems and those of our suppliers, vendors and other service providers, could have an adverse effect on our business. Cyber attacks may cause equipment failures, loss of information, including sensitive personal information of customers or employees or valuable technical and marketing information, as well as disruptions to our or our customers' operations. Cyber attacks against companies, including Verizon, have increased in frequency, scope and potential harm in recent years. They may occur alone or in conjunction with physical attacks, especially where disruption of service is an objective of the attacker. The development and maintenance of systems to prevent such attacks is costly and requires ongoing monitoring and updating to address their increasing prevalence and sophistication. While, to date, we have not been subject to cyber attacks that, individually or in the aggregate, have been material to Verizon's operations or financial condition, the preventive actions we take to reduce the risks associated with cyber attacks, including protection of our systems and networks, may be insufficient to repel or mitigate the effects of a major cyber attack in the future.

The inability to operate or use our networks and systems or those of our suppliers, vendors and other service providers as a result of cyber attacks, even for a limited period of time, may result in significant expenses to Verizon and/or a loss of market share to other communications providers. The costs associated with a major cyber attack on Verizon could include expensive incentives offered to existing customers and business partners to retain their business, increased expenditures on cybersecurity measures and the use of alternate resources, lost revenues from business interruption and litigation. Further, certain of Verizon's businesses, such as those offering security solutions and infrastructure and cloud services to business customers, could be negatively affected if our ability to protect our own networks and systems is called into question as a result of a cyber attack. Our presence in the IoT industry, which includes offerings of telematics products and services, could also increase our exposure to potential costs and expenses and reputational harm in the event of cyber attacks impacting these products or services. In addition, a compromise of security or a theft or other compromise of valuable information, such as financial data and sensitive or private personal information, could result in lawsuits and government claims, investigations or proceedings. Any of these occurrences could damage our reputation, adversely impact customer and investor confidence and result in a material adverse effect on Verizon's results of operation or financial condition.

Natural disasters, terrorist acts or acts of war could cause damage to our infrastructure and result in significant disruptions to our operations.

Our business operations are subject to interruption by power outages, terrorist attacks, other hostile acts and natural disasters, including an increasing prevalence of wildfires and intensified storm activities. Such events could cause significant damage to our infrastructure upon which our business operations rely, resulting in degradation or disruption of service to our customers, as well as significant recovery time and expenditures to resume operations. Our system redundancy may be ineffective or inadequate to sustain our operations through all such events. We are implementing, and will continue to implement, measures to protect our infrastructure and operations from the impacts of these events in the future, but these measures and our overall disaster recovery planning may not be sufficient for all eventualities. These events could also damage the infrastructure of the suppliers that provide us with the equipment and services that we need to operate our business and provide products to our customers. These occurrences could result in lost revenues from business interruption, damage to our reputation and reduced profits.

We depend on key suppliers and vendors to provide equipment that we need to operate our business.

We depend on various key suppliers and vendors to provide us, directly or through other suppliers, with equipment and services, such as fiber, switch and network equipment, smartphones and other wireless devices that we need in order to operate our business and provide products to our customers. For example, our smartphone and other device suppliers often rely on one vendor for the manufacture and supply of critical components, such as chipsets, used in their devices, and there are a limited number of companies capable of supplying the network infrastructure equipment on which we depend. These suppliers or vendors could fail to provide equipment or service on a timely basis, or fail to meet our performance expectations, for a number of reasons, including, for example, disruption to the global supply chain as a result of the coronavirus. If such failures occur, we may be unable to provide products and services as and when requested by our customers, or we may be unable to continue to maintain or upgrade our networks. Because of the cost and time lag that can be associated with transitioning from one supplier to another, our business could be substantially disrupted if we were required to, or chose to, replace the products or services of one or more major suppliers with products or services from another source, especially if the replacement became necessary on short notice. Any such disruption could increase our costs, decrease our operating efficiencies and have a material adverse effect on our business, results of operations and financial condition.

The suppliers and vendors on which we rely may also be subject to litigation with respect to technology on which we depend, including litigation involving claims of patent infringement. Such claims are frequently made in the communications industry. We are unable to predict whether our

business will be affected by any such litigation. We expect our dependence on key suppliers to continue as we develop and introduce more advanced generations of technology.

A significant portion of our workforce is represented by labor unions, and we could incur additional costs or experience work stoppages as a result of the renegotiation of our labor contracts.

As of December 31, 2019, approximately 23% of our workforce was represented by labor unions. While we have labor contracts in place with these unions, with subsequent negotiations we could incur additional costs and/or experience work stoppages, which could adversely affect our business operations. In addition, while a small percentage of the workforce of our wireless and other businesses outside of wireline is represented by unions, we cannot predict what level of success unions may have in further organizing this workforce or the potentially negative impact it would have on our operations.

Economic and Strategic Risks

We face significant competition that may reduce our profits.

We face significant competition in our industries. The rapid development of new technologies, services and products have eliminated many of the traditional distinctions among wireless, cable, Internet and local and long distance communication services and brought new competitors to our markets, including other telephone companies, cable companies, wireless service providers, satellite providers and application and device providers. While these changes have enabled us to offer new types of products and services, they have also allowed other providers to broaden the scope of their own competitive offerings. If we are unable to compete effectively, we could experience lower than expected revenues and earnings. A projected sustained decline in any of our reporting units' revenues and earnings could have a significant impact on its fair value and has caused us in the past, and may cause us in the future, to record goodwill impairment charges. The amount of any impairment charge could be significant and could have a material adverse impact on our results of operations for the period in which the charge is taken. In addition, wireless service providers are significantly altering the financial relationships with their customers through commercial offers that vary service and device pricing, promotions, incentives and levels of service provided – in some cases specifically targeting our customers. Our ability to compete effectively will depend on, among other things, our network quality, capacity and coverage, the pricing of our products and services, the quality of our customer service, our development of new and enhanced products and services, the reach and quality of our sales and distribution channels and our capital resources. It will also depend on how successfully we anticipate and respond to various factors affecting our industries, including new technologies and business models, changes in consumer preferences and demand for existing services, demographic trends and economic conditions. If we are not able to respond successfully to these competitive challenges, we could experience reduced profits.

If we are not able to take advantage of developments in technology and address changing consumer demand on a timely basis, we may experience a decline in the demand for our services, be unable to implement our business strategy and experience reduced profits.

Our industries are rapidly changing as new technologies are developed that offer consumers an array of choices for their communications needs and allow new entrants into the markets we serve. In order to grow and remain competitive, we will need to adapt to future changes in technology, enhance our existing offerings and introduce new offerings to address our customers' changing demands. If we are unable to meet future challenges from competing technologies on a timely basis or at an acceptable cost, we could lose customers to our competitors. We may not be able to accurately predict technological trends or the success of new services in the market.

The deployment of our 5G network is subject to a variety of risks, including those related to equipment availability, unexpected costs, and regulatory permitting requirements that could cause deployment delays or network performance issues. These issues could result in significant costs or reduce the anticipated benefits of the enhancements to our networks. If our services fail to gain acceptance in the marketplace, or if costs associated with the implementation and introduction of these services materially increase, our ability to retain and attract customers could be adversely affected.

In addition to introducing new offerings and technologies, such as 5G technology, we must phase out outdated and unprofitable technologies and services. If we are unable to do so on a cost-effective basis, we could experience reduced profits. In addition, there could be legal or regulatory restraints on our ability to phase out current services.

Adverse conditions in the U.S. and international economies could impact our results of operations.

Unfavorable economic conditions, such as a recession or economic slowdown in the U.S. or elsewhere, could negatively affect the affordability of and demand for some of our products and services. In difficult economic conditions, consumers may seek to reduce discretionary spending by forgoing purchases of our products, electing to use fewer higher margin services, dropping down in price plans or obtaining lower-cost products and services offered by other companies. Similarly, under these conditions, the business customers that we serve may delay purchasing decisions, delay full implementation of service offerings or reduce their use of services. In addition, adverse economic conditions may lead to an increased number of our consumer and business customers that are unable to pay for services. If these events were to occur, it could have a material adverse effect on our results of operations.

Regulatory and Legal Risks

Changes in the regulatory framework under which we operate could adversely affect our business prospects or results of operations.

Our domestic operations are subject to regulation by the FCC and other federal, state and local agencies, and our international operations are regulated by various foreign governments and international bodies. These regulatory regimes frequently restrict or impose conditions on our ability to operate in designated areas and provide specified products or services. We are frequently required to maintain licenses for our operations and conduct our operations in accordance with prescribed standards. We are often involved in regulatory and other governmental proceedings or inquiries related to the application of these requirements. It is impossible to predict with any certainty the outcome of pending federal and state regulatory proceedings relating to our operations, or the reviews by federal or state courts of regulatory rulings. Without relief, existing laws and regulations may inhibit our ability to expand our business and introduce new products and services. Similarly, we cannot guarantee that we will be successful in obtaining the licenses needed to carry out our business plan or in maintaining our existing licenses. For example, the FCC grants wireless licenses for terms generally lasting 10 years, subject to renewal. The loss of, or a material limitation on, certain of our licenses could have a material adverse effect on our business, results of operations and financial condition.

New laws or regulations or changes to the existing regulatory framework at the federal, state and local, or international level, such as those described below, or new laws or regulations enacted to address the potential impacts of climate change, could restrict the ways in which we manage our wireline and wireless networks and operate our Media business, impose additional costs, impair revenue opportunities and potentially impede our ability to provide services in a manner that would be attractive to us and our customers.

- *Privacy and data protection* - we are subject to federal, state and international laws related to privacy and data protection. Europe's General Data Protection Regulation, which went into effect in May 2018, includes significant penalties for non-compliance. In addition, a new privacy law took effect in California at the beginning of 2020, an additional privacy law is scheduled to take effect in Maine in 2020, and other states are considering additional regulations. These regulations could have a significant impact on our businesses.

- *Regulation of broadband Internet access services* - In its 2015 Title II Order, the FCC nullified its longstanding "light touch" approach to regulating broadband Internet access services and "reclassified" these services as telecommunications services subject to utilities-style common carriage regulation. The FCC repealed the 2015 Title II Order in December 2017, and returned to its traditional light-touch approach for these services. The 2017 order has been affirmed in part by the D.C. Circuit, but further appeals and challenges are possible; the outcome and timing of these or any other challenge remains uncertain. Several states have also adopted or are considering adopting laws or executive orders that would impose net neutrality and other requirements on some of our services (in some cases different from the FCC's 2015 rules). The enforceability and effect of these state rules is uncertain.

- *"Open Access"* - we hold certain wireless licenses that require us to comply with so-called "open access" FCC regulations, which generally require licensees of particular spectrum to allow customers to use devices and applications of their choice. Moreover, certain services could be subject to conflicting regulation by the FCC and/or various state and local authorities, which could significantly increase the cost of implementing and introducing new services.

The further regulation of broadband, wireless and our other activities and any related court decisions could restrict our ability to compete in the marketplace and limit the return we can expect to achieve on past and future investments in our networks.

We are subject to a significant amount of litigation, which could require us to pay significant damages or settlements.

We are subject to a substantial amount of litigation, including, from time to time, shareholder derivative suits, patent infringement lawsuits, antitrust class actions, wage and hour class actions, personal injury claims, property claims, and lawsuits relating to our advertising, sales, billing and collection practices. In addition, our wireless business also faces personal injury and wrongful death lawsuits relating to alleged health effects of wireless phones or radio frequency transmitters. We may incur significant expenses in defending these lawsuits. In addition, we may be required to pay significant awards or settlements.

Financial Risks

Verizon has significant debt, which could increase further if Verizon incurs additional debt in the future and does not retire existing debt.

As of December 31, 2019, Verizon had approximately $99.1 billion of outstanding unsecured indebtedness, $9.4 billion of unused borrowing capacity under its existing revolving credit facility and $12.4 billion of outstanding secured indebtedness. Verizon's debt level and related debt service obligations could have negative consequences, including:

- requiring Verizon to dedicate significant cash flow from operations to the payment of principal, interest and other amounts payable on its debt, which would reduce the funds Verizon has available for other purposes, such as working capital, capital expenditures, dividend payments and acquisitions;

- making it more difficult or expensive for Verizon to obtain any necessary future financing for working capital, capital expenditures, debt service requirements, debt refinancing, acquisitions or other purposes;

- reducing Verizon's flexibility in planning for or reacting to changes in its industries and market conditions;

- making Verizon more vulnerable in the event of a downturn in its business; and

- exposing Verizon to increased interest rate risk to the extent that its debt obligations are at variable interest rates.

Adverse changes in the credit markets and other factors could increase our borrowing costs and the availability of financing.

We require a significant amount of capital to operate and grow our business. We fund our capital needs in part through borrowings in the public and private credit markets. Adverse changes in the credit markets, including increases in interest rates, could increase our cost of borrowing and/or make it more difficult for us to obtain financing for our operations or refinance existing indebtedness. In addition, our ability to obtain funding under asset-backed debt transactions is subject to our ability to continue to originate a sufficient amount of assets eligible to be securitized. Our borrowing costs also can be affected by short- and long-term debt ratings assigned by independent rating agencies, which are based, in significant part, on our performance as measured by customary credit metrics. A decrease in these ratings would likely increase our cost of borrowing and/or make it more difficult for us to obtain financing. A severe disruption in the global financial markets could impact some of the financial institutions with which we do business, and such instability could also affect our access to financing.

Increases in costs for pension benefits and active and retiree healthcare benefits may reduce our profitability and increase our funding commitments.

With approximately 135,000 employees and approximately 191,000 retirees as of December 31, 2019 eligible to participate in Verizon's benefit plans, the costs of pension benefits and active and retiree healthcare benefits have a significant impact on our profitability. Our costs of maintaining these plans, and the future funding requirements for these plans, are affected by several factors, including the legislative and regulatory uncertainty regarding the potential modification of the Patient Protection and Affordable Care Act, increases in healthcare costs, decreases in investment returns on funds held by our pension and other benefit plan trusts and changes in the discount rate and mortality assumptions used to calculate pension and other postretirement expenses. If we are unable to limit future increases in the costs of our benefit plans, those costs could reduce our profitability and increase our funding commitments.

Item 1B. Unresolved Staff Comments

None.

Item 2. Properties

Our principal properties do not lend themselves to simple description by character and location. Our total gross investment in property, plant and equipment was approximately $266 billion at December 31, 2019 and $253 billion at December 31, 2018, including the effect of retirements, but before deducting accumulated depreciation. Our gross investment in property, plant and equipment consisted of the following:

At December 31,	**2019**	2018
Network equipment	**77.3%**	78.0%
Land, buildings and building equipment	**12.0%**	12.4%
Furniture and other	**10.7%**	9.6%
	100.0%	100.0%

Network equipment consists primarily of cable (aerial, buried, underground or undersea) and the related support structures of poles and conduit, wireless plant, switching equipment, network software, transmission equipment and related facilities. Land, buildings and building equipment consists of land and land improvements, central office buildings or any other buildings that house network equipment, and buildings that are used for administrative and other purposes. Substantially all the switching centers are located on land and in buildings we own due to their critical role in the networks and high set-up and relocation costs. We also maintain facilities throughout the U.S. comprised of administrative and sales offices, customer care centers, retail sales locations, garage work centers, switching centers, cell sites and data centers. Furniture and other consists of telephone equipment, furniture, data processing equipment, office equipment, motor vehicles, plant under construction and leasehold improvements.

Item 3. Legal Proceedings

In October 2013, the California Attorney General's Office notified certain Verizon companies of potential violations of California state hazardous waste statutes primarily arising from the disposal of electronic components, batteries and aerosol cans at certain California facilities. We are cooperating with this investigation and continue to review our operations relating to the management of hazardous waste. While penalties relating to the alleged violations could exceed $100,000, we do not expect that any penalties ultimately incurred will be material.

Item 4. Mine Safety Disclosures

None.

PART II

Item 5. Market for Registrant's Common Equity, Related Stockholder Matters and Issuer Purchases of Equity Securities

The principal market for trading in the common stock of Verizon is the New York Stock Exchange under the symbol "VZ". As of December 31, 2019, there were 605,414 shareholders of record.

Stock Repurchases

In February 2020, the Verizon Board of Directors authorized a share buyback program to repurchase up to 100 million shares of the Company's common stock. The program will terminate when the aggregate number of shares purchased reaches 100 million, or a new share repurchase plan superseding the current plan is authorized, whichever is sooner. Under the program, shares may be repurchased in privately negotiated transactions, on the open market, or otherwise, including through plans complying with Rule 10b5-1 under the Exchange Act. The timing and number of shares purchased under the program, if any, will depend on market conditions and the Company's capital allocation priorities.

During the years ended December 31, 2019 and 2018, Verizon did not repurchase any shares of Verizon's common stock under our previously authorized share buyback program. At December 31, 2019, the maximum number of shares that could be purchased by or on behalf of Verizon under our share buyback program was 100 million.

For other information required by this item, see the section entitled "Stock Performance Graph" in the 2019 Verizon Annual Report to Shareholders, which is incorporated herein by reference.

Item 6. Selected Financial Data

Information required by this item is included in the 2019 Verizon Annual Report to Shareholders under the heading "Selected Financial Data," which is incorporated herein by reference.

Item 7. Management's Discussion and Analysis of Financial Condition and Results of Operations

Information required by this item is included in the 2019 Verizon Annual Report to Shareholders under the heading "Management's Discussion and Analysis of Financial Condition and Results of Operations," which is incorporated herein by reference.

Item 7A. Quantitative and Qualitative Disclosures About Market Risk

Information required by this item is included in the 2019 Verizon Annual Report to Shareholders under the heading "Management's Discussion and Analysis of Financial Condition and Results of Operations - Market Risk," which is incorporated herein by reference.

Item 8. Financial Statements and Supplementary Data

Information required by this item is included in the consolidated financial statements and related notes of Verizon Communications Inc. and Subsidiaries in the 2019 Verizon Annual Report to Shareholders, which is incorporated herein by reference.

Item 9. Changes in and Disagreements with Accountants on Accounting and Financial Disclosure

None.

Item 9A. Controls and Procedures

Our Chief Executive Officer and Chief Financial Officer have evaluated the effectiveness of the registrant's disclosure controls and procedures (as defined in Rules 13a-15(e) and 15d-15(e) of the Securities Exchange Act of 1934), as of the end of the period covered by this Annual Report, that ensure that information relating to the registrant which is required to be disclosed in this report is recorded, processed, summarized and reported within required time periods using the criteria for effective internal control established in Internal Control–Integrated Framework issued by the Committee of Sponsoring Organizations of the Treadway Commission in 2013. Based on this evaluation, our Chief Executive Officer and Chief Financial Officer have concluded that the registrant's disclosure controls and procedures were effective as of December 31, 2019.

In the ordinary course of business, we routinely review our system of internal control over financial reporting and make changes to our systems and processes that are intended to ensure an effective internal control environment. There were no changes in the Company's internal control over financial reporting during the fourth quarter of 2019 that have materially affected, or are reasonably likely to materially affect, our internal control over financial reporting.

Management's report on internal control over financial reporting and the attestation report of Verizon's independent registered public accounting firm are included in the 2019 Verizon Annual Report to Shareholders and are incorporated herein by reference.

Item 9B. Other Information

None.

PART III

Item 10. Directors, Executive Officers and Corporate Governance

Set forth below is information with respect to our executive officers.

Name	Age	Office	Held Since
Hans Vestberg	54	Chairman and Chief Executive Officer	2019
Ronan Dunne	56	Executive Vice President and Group CEO - Verizon Consumer	2019
Matthew D. Ellis	48	Executive Vice President and Chief Financial Officer	2016
Tami A. Erwin	55	Executive Vice President and Group CEO - Verizon Business	2019
K. Guru Gowrappan	39	Executive Vice President and Group CEO - Verizon Media	2019
Kyle Malady	52	Executive Vice President and Chief Technology Officer	2019
Christine Pambianchi	51	Executive Vice President and Chief Human Resources Officer	2019
Rima Qureshi	55	Executive Vice President and Chief Strategy Officer	2017
Craig L. Silliman	52	Executive Vice President and Chief Administrative, Legal and Public Policy Officer	2019
Anthony T. Skiadas	51	Senior Vice President and Controller	2013

Prior to serving as an executive officer, each of the above officers has held high-level managerial positions with the Company or one of its subsidiaries for at least five years, with the exception of Hans Vestberg, who has been with the Company since 2017, Ronan Dunne, who has been with the Company since 2016, K. Guru Gowrappan, who has been with the Company since 2018, Christine Pambianchi, who has been with the Company since 2019 and Rima Qureshi, who has been with the Company since 2017. Officers are not elected for a fixed term of office and may be removed from office at any time at the discretion of the Board of Directors.

Hans Vestberg is the Chairman and Chief Executive Officer of Verizon. Mr. Vestberg joined the Company in April 2017 as Executive Vice President and President - Global Networks and Technology. He began serving in his current role of Chief Executive Officer in August 2018 and was elected Chairman in March 2019. Prior to joining Verizon, Mr. Vestberg served for six years as President and Chief Executive Officer of Ericsson, a multinational networking and telecommunications equipment and services company headquartered in Sweden.

Ronan Dunne is the Executive Vice President and Group CEO - Verizon Consumer. Mr. Dunne joined the Company in September 2016 as Executive Vice President and President of Verizon Wireless. Prior to joining Verizon, Mr. Dunne served for eight years as Chief Executive Officer of Telefónica UK Limited (O2), the second largest wireless operator in the United Kingdom.

K. Guru Gowrappan is the Executive Vice President and Group CEO - Verizon Media. Mr. Gowrappan joined the Company in April 2018 as the President and Chief Operating Officer of Oath. He began serving in his current role in October 2018. Prior to joining Verizon, Mr. Gowrappan served as the Global Managing Director of Alibaba Inc. from 2015 to 2018 and as the Chief Operating Officer for Quixey, a mobile search engine, from 2012 to 2015.

Christine Pambianchi is the Executive Vice President and Chief Human Resources Officer. Ms. Pambianchi joined the Company in July 2019. Prior to joining Verizon, Ms. Pambianchi led the Human Resources function at Corning Incorporated, a leading innovator in materials science, where she served as Executive Vice President, People and Digital, from 2018 to 2019 and as Senior Vice President, Human Resources, from 2010 to 2018.

Rima Qureshi is Executive Vice President and Chief Strategy Officer of Verizon. Ms. Qureshi joined the Company in November 2017. Prior to joining Verizon, Ms. Qureshi served as President and Chief Executive Officer of Ericsson North America from 2016 to 2017 and as Senior Vice President and Chief Strategy Officer and head of mergers and acquisitions of Ericsson from 2014 to 2016. Ms. Qureshi also served as Vice President of Ericsson's CDMA Mobile Systems Group, Senior Vice President of Strategic Projects, Chairman of Ericsson's Northern Europe, Russia and Central Asia Group and Chairman of Ericsson's Modem division before becoming Chief Strategy Officer.

For other information required by this item, see the sections entitled "Governance — Item 1: Election of Directors — Nominees for Election and — Election Process, — Our Approach to Governance — Where to Find More Information on Governance at Verizon, — Our Board Composition and Structure — Board Committees — Audit Committee and — Our Approach to Strategy and Risk Oversight — Other Risk-Related Matters — Business Conduct and Ethics" in our definitive Proxy Statement to be filed with the Securities and Exchange Commission and delivered to shareholders in connection with our 2020 Annual Meeting of Shareholders, which are incorporated herein by reference.

Item 11. Executive Compensation

For information with respect to executive compensation, see the sections entitled "Governance — Non-Employee Director Compensation" and "Executive Compensation — Compensation Discussion and Analysis, — Compensation Committee Report and — Compensation Tables" in our definitive Proxy Statement to be filed with the Securities and Exchange Commission and delivered to shareholders in connection with our 2020 Annual Meeting of Shareholders, which are incorporated by reference herein. There were no relationships to be disclosed under paragraph (e)(4) of Item 407 of Regulation S-K.

Item 12. Security Ownership of Certain Beneficial Owners and Management and Related Stockholder Matters

For information with respect to the security ownership of the Directors and Executive Officers, see the section entitled "Stock Ownership —Security Ownership of Certain Beneficial Owners and Management" in our definitive Proxy Statement to be filed with the SEC and delivered to shareholders in connection with our 2020 Annual Meeting of Shareholders, which is incorporated herein by reference. In addition, the following table provides other equity compensation plan information:

The following table provides information as of December 31, 2019 for (i) all equity compensation plans previously approved by the Company's shareholders, and (ii) all equity compensation plans not previously approved by the Company's shareholders. From May 9, 2009 until May 4, 2017, the Company only issued awards under the 2009 Verizon Communications Inc. Long-Term Incentive Plan and, after May 4, 2017, the Company only issued awards under the 2017 Verizon Communications. Inc. Long-Term Incentive Plan (2017 LTIP). Each of these plans provides for awards of stock options, restricted stock, restricted stock units, performance stock units and other equity-based hypothetical stock units to employees of Verizon and its subsidiaries. No new awards are permitted to be issued under any equity compensation plan other than the 2017 LTIP. In accordance with SEC rules, the table does not include outstanding awards that are payable solely in cash by the terms of the award, and such awards do not reduce the number of shares remaining for issuance under the 2017 LTIP.

Plan Category	Number of securities to be issued upon exercise of outstanding options, warrants and rights (a)	Weighted-average exercise price of outstanding options, warrants and rights (b)	Number of securities remaining available for future issuance under equity compensation plans (excluding securities reflected in column (a)) (c)
Equity compensation plans approved by security holders	7,259,237 [1]	$ — [2]	88,717,670 [3]
Equity compensation plans not approved by security holders	120,272 [4]	—	—
Total	**7,379,509**	**$ —**	**88,717,670**

[1] This amount includes: 7,259,237 of common stock subject to outstanding restricted stock units and performance stock units, including dividend equivalents accrued on such awards through December 31, 2019. This does not include performance stock units, deferred stock units and deferred share equivalents payable solely in cash.

[2] Verizon's outstanding restricted stock units, performance stock units and deferred stock units do not have exercise prices associated with the settlement of these awards.

[3] This number reflects the number of shares of common stock that remained available for future issuance under the 2017 LTIP.

[4] This number reflects shares subject to deferred stock units credited to the Verizon Income Deferral Plan, which were awarded in 2002 under the Verizon Communications Broad-Based Incentive Plan. No new awards are permitted to be issued under this plan.

Item 13. Certain Relationships and Related Transactions, and Director Independence

For information with respect to certain relationships and related transactions and Director independence, see the sections entitled "Governance — Our Approach to Governance — Our Approach to Strategy and Risk Oversight — Other Risk-Related Matters — Related Person Transactions and — Our Board Composition and Structure — Our Board's Independence" in our definitive Proxy Statement to be filed with the Securities and Exchange Commission and delivered to shareholders in connection with our 2020 Annual Meeting of Shareholders, which are incorporated by reference.

Item 14. Principal Accounting Fees and Services

For information with respect to principal accounting fees and services, see the section entitled "Audit Matters — Item 3: Ratification of Appointment of Independent Registered Public Accounting Firm" in our definitive Proxy Statement to be filed with the Securities and Exchange Commission and delivered to shareholders in connection with our 2020 Annual Meeting of Shareholders, which are incorporated by reference.

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PART IV

Item 15. Exhibits, Financial Statement Schedules

(a) Documents filed as part of this report:

* Incorporated herein by reference to the appropriate portions of the registrant's Annual Report to Shareholders for the fiscal year ended December 31, 2019. (See Part II.)

(5) Exhibits

Exhibits identified in parentheses below, on file with the SEC, are incorporated herein by reference as exhibits hereto. Unless otherwise indicated, all exhibits so incorporated are from File No. 1-8606.

Exhibit Number	Description
3a	Restated Certificate of Incorporation of Verizon Communications Inc. (Verizon) (filed as Exhibit 3a to Form 10-Q for the period ended June 30, 2014 and incorporated herein by reference).
3b	Bylaws of Verizon, as amended and restated, effective as of December 5, 2019 (filed as Exhibit 3b to Form 8-K filed on December 5, 2019 and incorporated herein by reference).
4a	Indenture between Verizon, both individually and as successor in interest to Verizon Global Funding Corp., and U.S. Bank National Association, as successor trustee to Wachovia Bank, National Association, formerly known as First Union National Bank, as Trustee, dated as of December 1, 2000 (incorporated by reference to Verizon Global Funding Corp.'s Registration Statement on Form S-4, Registration No. 333-64792, Exhibit 4.1).
4b	First Supplemental Indenture between Verizon, both individually and as successor in interest to Verizon Global Funding Corp., and U.S. Bank National Association, as successor trustee to Wachovia Bank, National Association, formerly known as First Union National Bank, as Trustee, dated as of May 15, 2001 (incorporated by reference to Verizon Global Funding Corp.'s Registration Statement on Form S-3, Registration No. 333-67412, Exhibit 4.2).
4c	Second Supplemental Indenture between Verizon, both individually and as successor in interest to Verizon Global Funding Corp., and U.S. Bank National Association, as successor trustee to Wachovia Bank, National Association, formerly known as First Union National Bank, as Trustee, dated as of September 29, 2004 (incorporated by reference to Form 8-K filed on February 9, 2006, Exhibit 4.1).
4d	Third Supplemental Indenture between Verizon, both individually and as successor in interest to Verizon Global Funding Corp., and U.S. Bank National Association, as successor trustee to Wachovia Bank, National Association, formerly known as First Union National Bank, as Trustee, dated as of February 1, 2006 (incorporated by reference to Form 8-K filed on February 9, 2006, Exhibit 4.2).
4e	Fourth Supplemental Indenture between Verizon, both individually and as successor in interest to Verizon Global Funding Corp., and U.S. Bank National Association, as successor trustee to Wachovia Bank, National Association, formerly known as First Union National Bank, as Trustee, dated as of April 4, 2016 (incorporated by reference to Verizon Communications Inc.'s Registration Statement on Form S-4, Registration No. 333-212307, Exhibit 4.5).
	Except for Exhibits 4a – 4e above, no other instrument which defines the rights of holders of long-term debt of Verizon and its consolidated subsidiaries is filed herewith pursuant to Regulation S-K, Item 601(b)(4)(iii)(A). Pursuant to this regulation, Verizon hereby agrees to furnish a copy of any such instrument to the SEC upon request.
4f	Description of Verizon's Securities Registered Pursuant to Section 12 of the Securities and Exchange Act of 1934, filed herewith.
10a	NYNEX Directors' Charitable Award Program (filed as Exhibit 10i to Form 10-K for the year ended December 31, 2000 and incorporated herein by reference).**
10b	2009 Verizon Long-Term Incentive Plan, As Amended and Restated (incorporated by reference to Appendix D of the Registrant's Proxy Statement included in Schedule 14A filed on March 18, 2013).**
10b(i)	Form of Performance Stock Unit Agreement 2016-2018 Award Cycle (filed as Exhibit 10a to Form 10-Q for the period ended March 31, 2016 and incorporated herein by reference).**
10b(ii)	Form of Restricted Stock Unit Agreement 2016-2018 Award Cycle (filed as Exhibit 10b to Form 10-Q for the period ended March 31, 2016 and incorporated herein by reference).**
10b(iii)	Form of 2017 Performance Stock Unit Agreement pursuant to the 2009 Verizon Communications Inc. Long-Term Incentive Plan. (filed as Exhibit 10a to Form 10-Q for the period ended March 31, 2017 and incorporated herein by reference).**
10b(iv)	Form of 2017 Restricted Stock Unit Agreement pursuant to the 2009 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10b to Form 10-Q for the period ended March 31, 2017 and incorporated herein by reference).**
10b(v)	2017 Special Performance Stock Unit Agreement pursuant to the 2009 Verizon Communications Inc. Long-Term Incentive Plan for J. Stratton (filed as Exhibit 10c to Form 10-Q for the period ended March 31, 2017 and incorporated herein by reference).**
10c	2017 Verizon Communications Inc. Long-Term Incentive Plan (incorporated by reference to Appendix B of the Registrant's Proxy Statement included in Schedule 14A filed on March 20, 2017).**
10c(i)	Form of 2017 Performance Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan. (filed as Exhibit 10a to Form 10-Q for the period ended June 30, 2017 and incorporated herein by reference).**
10c(ii)	Form of 2017 Restricted Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10b to Form 10-Q for the period ended June 30, 2017 and incorporated herein by reference).**
10c(iii)	2017 Special Restricted Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c to Form 10-Q for the period ended June 30, 2017 and incorporated herein by reference).**

10c(iv)	Form of 2017 Restricted Stock Unit Agreement (cash-settled) pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c(iv) to Form 10-K for period ended December 31, 2017 and incorporated herein by reference).**
10c(v)	Form of 2018 Performance Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10a to Form 10-Q for the period ended March 31, 2018 and incorporated herein by reference).**
10c(vi)	Form of 2018 Restricted Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan. (filed as Exhibit 10b to Form 10-Q for the period ended March 31, 2018 and incorporated herein by reference).**
10c(vii)	2018 Special Performance Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan for H. Vestberg (filed as Exhibit 10 to Form 10-Q for the period ended September 30, 2018 and incorporated herein by reference).**
10c(viii)	2018 Restricted Stock Unit Agreement for G. Gowrappan pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c(viii) to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
10c(ix)	Special Performance Restricted Stock Unit Agreement for R. Dunne pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c(ix) to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
10c(x)	Special Performance Restricted Stock Unit Agreement for G. Gowrappan pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c(x) to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
10c(x)(i)	Amendment to Special Performance Restricted Stock Unit Agreement for G. Gowrappan pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c(x)(i) to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
10c(xii)	Form of 2019 Performance Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10b to Form 10-Q for the period ended March 31, 2019 and incorporated herein by reference).**
10c(xiii)	Form of 2019 Restricted Stock Unit Agreement pursuant to the 2017 Verizon Communications Inc. Long-Term Incentive Plan (filed as Exhibit 10c to Form 10-Q for the period ended March 31, 2019 and incorporated herein by reference).**
10d	Verizon Communications Inc. Short-Term Incentive Plan (filed as Exhibit 10a to Form 10-Q for the period ended March 31, 2019 and incorporated herein by reference).**
10e	Verizon Executive Deferral Plan (filed as Exhibit 10e to Form 10-K for the period ended December 31, 2017 and incorporated herein by reference).**
10f	Verizon Income Deferral Plan (filed as Exhibit 10f to Form 10-Q for the period ended June 30, 2002 and incorporated herein by reference).**
10f(i)	Description of Amendment to Plan (filed as Exhibit 10o(i) to Form 10-K for the year ended December 31, 2004 and incorporated herein by reference).**
10g	Verizon Excess Pension Plan (filed as Exhibit 10p to Form 10-K for the year ended December 31, 2004 and incorporated herein by reference).**
10g(i)	Description of Amendment to Plan (filed as Exhibit 10p(i) to Form 10-K for the year ended December 31, 2004 and incorporated herein by reference).**
10h	GTE's Executive Salary Deferral Plan, as amended (filed as Exhibit 10.10 to GTE's Form 10-K for the year ended December 31, 1998, File No. 1-2755 and incorporated herein by reference).**
10i	Bell Atlantic Senior Management Long-Term Disability and Survivor Protection Plan, as amended (filed as Exhibit 10h to Form SE filed on March 27, 1986 and Exhibit 10b(ii) to Form 10-K for the year ended December 31, 1997 and incorporated herein by reference).**
10j	GTE Executive Retiree Life Insurance Plan (filed as Exhibit 10q to Form 10-K for the year ended December 31, 2010 and incorporated herein by reference).**
10k	Verizon Executive Life Insurance Plan, As Amended and Restated September 2009 (filed as Exhibit 10s to Form 10-K for the year ended December 31, 2010 and incorporated herein by reference).**
10l	Form of Aircraft Time Sharing Agreement (filed as Exhibit 10l to Form 10-K for year ended December 31, 2017 and incorporated herein by reference).**
10m	NYNEX Deferred Compensation Plan for Non-Employee Directors (filed as Exhibit 10iii 5a to NYNEX's Quarterly Report on Form 10-Q for the period ended June 30, 1996, File No. 1-8608 and incorporated herein by reference).**

10n	Verizon Senior Manager Severance Plan (filed as Exhibit 10d to Form 10-Q for the period ended March 31, 2010 and incorporated herein by reference).**
10o	AOL Inc. Long-Term Incentive Plan (filed as Exhibit 10o to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
10o(i)	Founders' Grant Unit Agreement for T. Armstrong pursuant to the AOL Inc. Long-Term Incentive Plan (filed as Exhibit 10o(i) to Form 10-K for the period ended December 31, 2018 and incorporated herein by reference).**
13	Portions of Verizon's Annual Report to Shareholders for the fiscal year ended December 31, 2019 filed herewith. Only the information incorporated by reference into this Form 10-K is included in the exhibit.
21	List of principal subsidiaries of Verizon, filed herewith.
23	Consent of Ernst & Young LLP, filed herewith.
24	Powers of Attorney, filed herewith.
31.1	Certification of Chief Executive Officer pursuant to Section 302 of the Sarbanes-Oxley Act of 2002, filed herewith.
31.2	Certification of Chief Financial Officer pursuant to Section 302 of the Sarbanes-Oxley Act of 2002, filed herewith.
32.1	Certification of Chief Executive Officer pursuant to Section 906 of the Sarbanes-Oxley Act of 2002, filed herewith.
32.2	Certification of Chief Financial Officer pursuant to Section 906 of the Sarbanes-Oxley Act of 2002, filed herewith.
101.INS	XBRL Instance Document - the instance document does not appear in the interactive data file because its XBRL tags are embedded within the inline XBRL document.
101.SCH	XBRL Taxonomy Extension Schema Document.
101.PRE	XBRL Taxonomy Presentation Linkbase Document.
101.CAL	XBRL Taxonomy Calculation Linkbase Document.
101.LAB	XBRL Taxonomy Label Linkbase Document.
101.DEF	XBRL Taxonomy Extension Definition Linkbase Document.
104	Cover Page Interactive Data File (formatted as inline XBRL with applicable taxonomy extension information contained in Exhibits 101).
**	Indicates management contract or compensatory plan or arrangement.

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Schedule II - Valuation and Qualifying Accounts

Verizon Communications Inc. and Subsidiaries

For the Years Ended December 31, 2019, 2018 and 2017

(dollars in millions)

Description	Balance at Beginning of Period	Additions: Charged to Expenses	Additions: Charged to Other Accounts[a]	Deductions[b]	Balance at End of Period[c]
Allowance for Uncollectible Accounts Receivable:					
Year 2019	**$ 930**	**$ 1,441**	**$ 133**	**$ 1,644**	**$ 860**
Year 2018	1,199	776	216	1,261	930
Year 2017	1,146	1,167	205	1,319	1,199

Description	Balance at Beginning of Period	Additions: Charged to Expenses	Additions: Charged to Other Accounts[d]	Deductions[e]	Balance at End of Period
Valuation Allowance for Deferred Tax Assets:					
Year 2019	**$ 2,741**	**$ 402**	**$ 8**	**$ 891**	**$ 2,260**
Year 2018	3,293	251	112	915	2,741
Year 2017	2,473	765	273	218	3,293

[a] Charged to Other Accounts primarily includes amounts previously written off which were credited directly to this account when recovered.
[b] Deductions primarily include amounts written off as uncollectible or transferred to other accounts or utilized.
[c] Allowance for Uncollectible Accounts Receivable includes approximately $127 million, $165 million and $260 million at December 31, 2019, 2018, and 2017, respectively, related to long-term device payment plan receivables.
[d] Charged to Other Accounts includes current year increase to valuation allowance charged to equity and reclassifications from other balance sheet accounts.
[e] Reductions to valuation allowances related to deferred tax assets.

Item 16. Form 10-K Summary

None.

Signatures

Pursuant to the requirements of Section 13 or 15(d) of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

VERIZON COMMUNICATIONS INC.

By: /s/ Anthony T. Skiadas — Date: February 21, 2020

Anthony T. Skiadas
Senior Vice President and Controller

Pursuant to the requirements of the Securities Exchange Act of 1934, this report has been signed below by the following persons on behalf of the registrant and in the capacities and on the dates indicated.

Principal Executive Officer:

/s/ Hans E. Vestberg Hans E. Vestberg	Chairman and Chief Executive Officer	February 21, 2020

Principal Financial Officer:

/s/ Matthew D. Ellis Matthew D. Ellis	Executive Vice President and Chief Financial Officer	February 21, 2020

Principal Accounting Officer:

/s/ Anthony T. Skiadas Anthony T. Skiadas	Senior Vice President and Controller	February 21, 2020

*	Director	February 21, 2020
Hans E. Vestberg		
*	Director	February 21, 2020
Shellye L. Archambeau		
*	Director	February 21, 2020
Mark T. Bertolini		
*	Director	February 21, 2020
Vittorio Colao		
*	Director	February 21, 2020
Melanie L. Healey		
*	Director	February 21, 2020
Clarence Otis, Jr.		
*	Director	February 21, 2020
Daniel H. Schulman		
*	Director	February 21, 2020
Rodney E. Slater		
*	Director	February 21, 2020
Kathryn A. Tesija		
*	Director	February 21, 2020
Carol B. Tomé		
*	Director	February 21, 2020
Gregory G. Weaver		

* By: /s/ Anthony T. Skiadas

Anthony T. Skiadas

(as attorney-in-fact)

EXHIBIT 4f

DESCRIPTION OF VERIZON'S SECURITIES
REGISTERED PURSUANT TO SECTION 12 OF
THE SECURITIES EXCHANGE ACT OF 1934

As of December 31, 2019, Verizon Communications Inc. had the following twenty-one classes of securities registered under Section 12 of the Securities Exchange Act of 1934, as amended: (i) common stock, $.0.10 par value per share ("Common Stock"), (ii) 2.375% Notes due 2022 (the "2.375% 2022 Notes"), (iii) 0.500% Notes due 2022 (the "0.500% 2022 Notes"), (iv) 1.625% Notes due 2024 (the "1.625% 2024 Notes"), (v) 4.073% Notes due 2024 (the "4.073% 2024 Notes"), (vi) 0.875% Notes due 2025 (the "2025 Notes"), (vii) 3.250% Notes due 2026 (the "3.250% 2026 Notes"), (viii) 1.375% Notes due 2026 (the "1.375% 2026 Notes"), (ix) 0.875% Notes due 2027 (the "2027 Notes"), (x) 1.375% Notes due 2028 (the "2028 Notes"), (xi) 1.875% Notes due 2029 (the "2029 Notes"), (xii) 1.250% Notes due 2030 (the "1.250% 2030 Notes"), (xiii)1.875% Notes due 2030 (the "1.875% 2030 Notes"), (xiv) 2.625% Notes due 2031 (the "2.625% 2031 Notes"), (xv) 2.500% Notes due 2031 (the "2.500% 2031 Notes"), (xvi) 0.875% Notes due 2032 (the "2032 Notes"), (xvii) 4.750% Notes due 2034 (the "2034 Notes"), (xviii) 3.125% Notes due 2035 (the "2035 Notes"), (xix) 3.375% Notes due 2036 (the "2036 Notes"), (xx) 2.875% Notes due 2038 (the "2038 Notes") and (xxi) 1.500% Notes due 2039 (the "2039 Notes," and together with the 2.375% 2022 Notes, the 0.500% 2022 Notes, the 1.625% 2024 Notes, the 4.073% 2024 Notes, the 2025 Notes, the 3.250% 2026 Notes, the 1.375% 2026 Notes, the 2027 Notes, the 2028 Notes, the 2029 Notes, the 1.250% 2030 Notes, the 1.875% 2030 Notes, the 2.625% 2031 Notes, the 2.500% 2031 Notes, the 2032 Notes, the 2034 Notes, the 2035 Notes, the 2036 Notes and the 2038 Notes, the "Notes"). In this exhibit, "we," "our," "us" and "Verizon Communications" refer to Verizon Communications Inc.

COMMON STOCK

Our restated certificate of incorporation provides authority to issue up to 6,500,000,000 shares of stock of all classes, of which 6,250,000,000 are shares of Common Stock, and 250,000,000 are shares of preferred stock, $0.10 par value per share.

Subject to any preferential rights of the preferred stock, holders of shares of our Common Stock are entitled to receive dividends on that stock out of assets legally available for distribution when, as and if authorized and declared by the board of directors and to share ratably in assets legally available for distribution to our shareholders in the event of our liquidation, dissolution or winding-up. We may not pay any dividend or make any distribution of assets on shares of Common Stock until cumulative dividends on shares of preferred stock then outstanding, if any, having dividend or distribution rights senior to the Common Stock have been paid.

Holders of Common Stock are entitled to one vote per share on all matters voted on generally by the shareholders, including the election of directors. In addition, the holders of Common Stock possess all voting power except as otherwise required by law or except as provided for by any series of preferred stock. Our restated certificate of incorporation does not provide for cumulative voting for the election of directors.

No holder of any shares of Common Stock has any preemptive or preferential right to acquire or subscribe for any unissued shares of any class of stock or any authorized securities convertible into or carrying any right, option or warrant to subscribe for or acquire shares of any class of stock.

The Common Stock is listed on the New York Stock Exchange and the NASDAQ Global Select Market under the symbol "VZ."

Our board of directors is authorized at any time to provide for the issuance of all or any shares of our preferred stock in one or more classes or series, and to fix for each class or series voting powers, full or limited, or no voting powers, and distinctive designations, preferences and relative, participating, optional or other special rights and any qualifications, limitations or restrictions, as shall be stated and expressed in the resolution or resolutions adopted by the board of directors providing for the issuance of the preferred stock and to the fullest extent as may be permitted by Delaware law. This authority includes, but is not limited to, the authority to provide that any class or series be:

- subject to redemption at a specified time or times and at a specified price or prices;
- entitled to receive dividends (which may be cumulative or non-cumulative) at specified rates, on specified conditions and at specified times, and payable in preference to, or in relation to, the dividends payable on any other class or classes or any other series;
- entitled to rights upon the dissolution of, or upon any distribution of the assets of, Verizon Communications; or
- convertible into, or exchangeable for, shares of any class or classes of our stock, or our other securities or property, at a specified price or prices or at specified rates of exchange and with any specified adjustments.

Although no shares of preferred stock are outstanding as of December 31, 2019, in the event of the issuance of any shares of preferred stock, the rights evidenced by, or amounts payable with respect to, the Common Stock may be materially limited or qualified by the terms of such preferred stock.

NOTES

The following description of the Notes is a summary and does not purport to be complete. This description is qualified in its entirety by reference to the indenture between the Company and U.S. Bank National Association (as successor to Wachovia Bank, National Association,

formerly known as First Union National Bank), as trustee, dated as of December 1, 2000, as amended (the "Indenture") and the terms of the global securities representing the Notes.

Principal Amount, Maturity, Interest and Listing

The following table sets forth for each series of Notes the applicable date of initial issuance, principal amount initially issued, principal amount outstanding as of December 31, 2019, maturity date, interest rate per annum, interest payment and record dates, and New York Stock Exchange listing symbol:

Notes	Date of Initial Issuance	Principal Amount Initially Issued	Principal Amount Outstanding as of 12/31/2019	Maturity Date	Interest Rate Per Annum	Interest Payment Date	Record Date	NYSE Listing Symbol
2.375% 2022 Notes	February 12, 2014	€1,750,000,000	€935,347,000	February 17, 2022	2.375%	February 17	February 3	VZ22A
0.500% 2022 Notes	November 2, 2016	€1,000,000,000	€453,963,000	June 2, 2022	0.500%	June 2	May 19	VZ22B
1.625% 2024 Notes	December 1, 2014	€1,400,000,000	€684,827,000	March 1, 2024	1.625%	March 1	February 15	VZ24B
4.073% 2024 Notes	June 18, 2014	£694,804,000	£412,534,000	June 18, 2024	4.073%	June 18	June 4	VZ24C
2025 Notes	November 2, 2016	€1,000,000,000	€1,000,000,000	April 2, 2025	0.875%	April 2	March 19	VZ25
3.250% 2026 Notes	February 12, 2014	€1,250,000,000	€1,250,000,000	February 17, 2026	3.250%	February 17	February 3	VZ26
1.375% 2026 Notes	October 27, 2017	€1,250,000,000	€1,250,000,000	October 27, 2026	1.375%	October 27	October 12	VZ26B
2027 Notes	April 8, 2019	€1,250,000,000	€1,250,000,000	April 8, 2027	0.875%	April 8	March 24	VZ27E
2028 Notes	November 2, 2016	€1,250,000,000	€1,250,000,000	November 2, 2028	1.375%	November 2	October 19	VZ28
2029 Notes	October 27, 2017	€750,000,000	€750,000,000	October 26, 2029	1.875%	October 26	October 11	VZ29B
1.250% 2030 Notes	April 8, 2019	€1,250,000,000	€1,250,000,000	April 8, 2030	1.250%	April 8	March 24	VZ30
1.875% 2030 Notes	September 19, 2019	£550,000,000	£550,000,000	September 19, 2030	1.875%	September 19	September 4	VZ30A
2.625% 2031 Notes	December 1, 2014	€1,000,000,000	€1,000,000,000	December 1, 2031	2.625%	December 1	November 15	VZ31
2.500% 2031 Notes	April 8, 2019	£500,000,000	£500,000,000	April 8, 2031	2.500%	April 8	March 24	VZ31A
2032 Notes	September 19, 2019	€800,000,000	€800,000,000	March 19, 2032	0.875%	March 19	March 4	VZ32
2034 Notes	February 12, 2014	£850,000,000	£456,624,000	February 17, 2034	4.750%	February 17	February 3	VZ34
2035 Notes	November 2, 2016	£450,000,000	£450,000,000	November 2, 2035	3.125%	November 2	October 19	VZ35
2036 Notes	October 27, 2017	£1,000,000,000	£1,000,000,000	October 27, 2036	3.375%	October 27	October 12	VZ36A
2038 Notes	October 27, 2017	€1,500,000,000	€1,500,000,000	January 15, 2038	2.875%	January 15	January 1	VZ38B
2039 Notes	September 19, 2019	€500,000,000	€500,000,000	September 19, 2039	1.500%	September 19	September 4	VZ39C

Interest on each series of Notes is payable annually in arrears and will be computed on the basis of the actual number of days in the period for which interest is being calculated and the actual number of days from and including the last date on which interest was paid on such series (or the date of initial issuance of such series, if no interest has been paid on such series), to but excluding the next scheduled interest payment date. This payment convention is referred to as ACTUAL/ACTUAL (ICMA) as defined by the rulebook of the International Capital Market Association.

If interest or principal on any of the 2.375% 2022 Notes, 0.500% 2022 Notes, 1.625% 2024 Notes, 2025 Notes, 3.250% 2026 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 2.625% 2031 Notes, 2032 Notes, 2038 Notes and 2039 Notes (collectively, the "Euro Notes") is payable on a Saturday, Sunday or any other day when commercial banks are not open for business in The City of New York or London or any day on which the Trans- European Automated Real-time Gross settlement Express Transfer payment system or any successor thereto is not open for transfer of payments, we will make the payment on the next succeeding business day in such locations, and no additional interest will accrue as a result of the delay in payment. If interest or principal on any of the 4.073% 2024 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2034 Notes, 2035 Notes and 2036 Notes (collectively, the "Sterling Notes") is payable on a Saturday, Sunday or any other day when commercial banks are not open for business in The City of New York or London, we will make the payment on the next business day in such locations, and no additional interest will accrue as a result of the delay in payment.

We may issue additional Notes of any series in the future.

Ranking

Each series of Notes is unsecured and ranks equally with all of our unsecured and unsubordinated indebtedness.

Currency Conversion

All payments of principal, interest and additional amounts, if any, including any payments made upon any redemption, on the Euro Notes will be payable in euro.

All payments of principal, interest and additional amounts, if any, including any payments made upon any redemption, on the Sterling Notes will be payable in GBP.

If either euro or GBP, as applicable, is unavailable to us due to the imposition of exchange controls or other circumstances beyond our control (including the dissolution of the euro), then all payments in respect of the relevant Notes will be made in U.S. dollars until euro or GBP, as the case may be, is again available to us. The amount payable on any date in euro or GBP, as applicable, will be converted into U.S. dollars at a rate mandated by the U.S. Federal Reserve Board as of the close of business on the second business day prior to the relevant payment date or, in the event the U.S. Federal Reserve Board has not mandated a rate of conversion, on the basis of the latest U.S. dollar/euro exchange rate or U.S. dollar/GBP exchange rate, as applicable, available on or prior to the second business day prior to the relevant payment date as determined by us in our sole discretion. Any payment in respect of the Notes alternatively made in U.S. dollars will not constitute an event of default under the Notes or the Indenture.

Optional Redemption

2.375% 2022 Notes, 0.500% 2022 Notes, 1.625% 2024 Notes, 4.073% 2024 Notes, 2025 Notes, 3.250% 2026 Notes, 1.375% 2026 Notes, 2028 Notes, 2029 Notes, 2.625% 2031 Notes, 2034 Notes, 2035 Notes, 2036 Notes and 2038 Notes

We have the option to redeem each of the 2.375% 2022 Notes, 0.500% 2022 Notes, 1.625% 2024 Notes, 4.073% 2024 Notes, 2025 Notes, 3.250% 2026 Notes, 1.375% 2026 Notes, 2028 Notes, 2029 Notes, 2.625% 2031 Notes, 2034 Notes, 2035 Notes, 2036 Notes and 2038 Notes on not less than 30 nor more than 60 days' notice, in whole or in part, at any time prior to maturity, at a redemption price equal to the greater of:

(i) 100% of the principal amount of the Notes of such series being redeemed, or

(ii) the sum of the present values of the remaining scheduled payments of principal and interest on the Notes of such series being redeemed (exclusive of interest accrued to the date of redemption), as the case may be, discounted to the date of redemption on an annual basis (ACTUAL/ACTUAL (ICMA)) at (A) the Comparable Government Bond Rate plus 20 basis points for the 2.375% due 2022 Notes, (B) the Comparable Government Bond Rate plus 15 basis points for the 0.500% 2022 Notes, (C) the Comparable Government Bond Rate plus 15 basis points for the 1.625% 2024 Notes, (D) the Comparable Government Bond Rate plus 25 basis points for the 4.073% 2024 Notes, (E) the Comparable Government Bond Rate plus 20 basis points for the 2025 Notes, (F) the Comparable Government Bond Rate plus 25 basis points for the 3.250% 2026 Notes, (G) the Comparable Government Bond Rate plus 20 basis points for the 1.375% 2026 Notes, (H) the Comparable Government Bond Rate plus 20 basis points for the 2028 Notes, (I) the Comparable Government Bond Rate plus 25 basis points for the 2029 Notes, (J) the Comparable Government Bond Rate plus 25 basis points for the 2.625% 2031 Notes, (K) the Comparable Government Bond Rate plus 25 basis points for the 2034 Notes, (L) the Comparable Government Bond Rate plus 25 basis points for the 2035 Notes, (M) the Comparable Government Bond Rate plus 25 basis points for the 2036 Notes and (N) the Comparable Government Bond Rate plus 30 basis points for the 2038 Notes,

plus, in each case, accrued and unpaid interest on the principal amount being redeemed to, but excluding, the date of redemption.

2027 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes and 2039 Notes

We have the option to redeem the 2027 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes and 2039 Notes on not less than 10 nor more than 60 days' notice, in whole or in part,

(i) at any time prior to (A) January 8, 2027 (three months prior to the maturity date of the 2027 Notes) (the "2027 Notes par call date") with respect to the 2027 Notes, (B) January 8, 2030 (three months prior to the maturity date of the 1.250% 2030 Notes) (the "1.250% 2030 Notes par call date") with respect to the 1.250% 2030 Notes (C) June 19, 2030 (three months prior to the maturity date of the 1.875% 2030 Notes) (the "1.875% 2030 Notes par call date") with respect to the 1.875% 2030 Notes (D) January 8, 2031 (three months prior to the maturity date of the 2.500% 2031 Notes) (the "2.500% 2031

Notes par call date") with respect to the 2.500% 2031 Notes, (E) December 19, 2031 (three months prior to the maturity date of the 2032 Notes) (the "2032 Notes par call date") with respect to the 2032 Notes and (F) March 19, 2039 (six months prior to the maturity date of the 2039 Notes) (the "2039 Notes par call date") with respect to the 2039 Notes, at a redemption price equal to the greater of:

(a) 100% of the principal amount of the Notes of such series being redeemed, or

(b) the sum of the present values of the remaining scheduled payments of principal and interest on the Notes of such series being redeemed (exclusive of interest accrued to the date of redemption), assuming for such purpose that the (A) 2027 Notes matured on the 2027 Notes par call date, (B) 1.250% 2030 Notes matured on the 1.250% 2030 Notes par call date, (C) 1.875% 2030 Notes matured on the 1.875% 2030 Notes par call date, (D) 2.500% 2031 Notes matured on the 2.500% 2031 par call date, (E) 2032 Notes matured on the 2032 Notes par call date and (F) 2039 Notes matured on the 2039 Notes par call date, discounted to the date of redemption on an annual basis (ACTUAL/ACTUAL (ICMA)) at (AA) the Comparable Government Bond Rate plus 20 basis points for the 2027 Notes, (BB) the Comparable Government Bond Rate plus 25 basis points for the 1.250% 2030 Notes, (CC) the Comparable Government Bond Rate plus 25 basis points for the 1.875% 2030 Notes, (DD) the Comparable Government Bond Rate plus 25 basis points for the 2.500% 2031 Notes, (EE) at the Comparable Government Bond Rate plus 25 basis points for the 2032 Notes and (FF) the Comparable Government Bond Rate plus 30 basis points for the 2039 Notes; and

(ii) at any time on or after (A) the 2027 Notes par call date with respect to the 2027 Notes, (B) the 1.250% 2030 Notes par call date with respect to the 1.250% 2030 Notes, (C) the 1.875% 2030 Notes par call date with respect to the 1.875% 2030 Notes, (D) the 2.500% 2031 Notes par call date with respect to the 2.500% 2031 Notes (E) the 2032 Notes par call date with respect to the 2032 Notes and (F) the 2039 Notes par call date with respect to the 2039 Notes, at a redemption price equal to 100% of the principal amount of the Notes of such series being redeemed,

plus, in each case, accrued and unpaid interest on the principal amount of such series being redeemed to, but excluding, the date of redemption.

Defined Terms

The "Comparable Government Bond Rate" will be determined on the third business day preceding the redemption date and means, with respect to any date of redemption, the rate per annum equal to the yield to maturity calculated in accordance with customary financial practice in pricing new issues of comparable corporate debt securities paying interest on an annual basis (ACTUAL/ACTUAL (ICMA)) of the applicable Comparable Government Bond, assuming a price for the applicable Comparable Government Bond (expressed as a percentage of its principal amount) equal to the applicable Comparable Government Bond Price for such date of redemption.

"Calculation Agent" means an independent investment banking or commercial banking institution of international standing appointed by us.

"Comparable Government Bond" means (i) with respect to any series of Euro Notes, the Federal Republic of Germany government security or securities selected by one of the Reference Government Bond Dealers appointed by us as having an actual or interpolated maturity comparable with the remaining term of such series of Euro Notes that would be utilized, at the time of selection and in accordance with customary financial practice, in pricing new issues of euro-denominated corporate debt securities of a maturity comparable to the remaining term of such series of Euro Notes and (ii) with respect to any series of Sterling Notes, the United Kingdom government security or securities selected by one of the Reference Government Bond Dealers appointed by us as having an actual or interpolated maturity comparable with the remaining term of such series of Sterling Notes that would be utilized, at the time of selection and in accordance with customary financial practice, in pricing new issues of sterling-denominated corporate debt securities of a maturity comparable to the remaining term of such series of Sterling Notes.

"Comparable Government Bond Price" means, with respect to any redemption date, (A) the arithmetic average of the Reference Government Bond Dealer Quotations for such redemption date, after excluding the highest and lowest such Reference Government Bond Dealer Quotations, or (B) if the Calculation Agent obtains fewer than four such Reference Government Bond Dealer Quotations, the arithmetic average of all such quotations.

"Reference Government Bond Dealer" means each of five banks selected by us, which are (A) primary European government securities dealers, and their respective successors, or (B) market makers in pricing corporate bond issues.

"Reference Government Bond Dealer Quotations" means, with respect to each Reference Government Bond Dealer and any redemption date, the arithmetic average, as determined by the Calculation Agent, of the bid and offered prices for the applicable Comparable Government Bond (expressed in each case as a percentage of its principal amount) at 11:00 a.m., Central European Time (or in the case of the 4.073% 2024 Notes, London Time), on the third business day preceding such date for redemption quoted in writing to the Calculation Agent by such Reference Government Bond Dealer.

Tax Redemption

2.375% 2022 Notes, 1.625% 2024 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes, 2.625% 2031 Notes and 2034 Notes

Each of the 2.375% 2022 Notes, 1.625% 2024 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes, 2.625% 2031 Notes and 2034 Notes may be redeemed at our option, in whole but not in part, at any time on giving not less than 30 nor more than 60 days' notice to the noteholders (which notice shall be irrevocable), at their principal amount, together with interest accrued to, but excluding, the date fixed for redemption, if:

(i) we have or will become obliged to pay additional amounts with respect to such series of Notes as provided or referred to under "-Withholding Taxes-*2.375% 2022 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes and 2034 Notes*" below in the case of the 2.375% 2022 Notes, 1.625% 2024 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes, 2.625% 2031 Notes and 2034 Notes, or under "-Withholding Taxes-*1.625% 2024 Notes and 2.625% 2031 Notes*" below in the case of the 1.625% 2024 Notes and 2.625% 2031 Notes, as a result of any change in, or amendment to, the laws, treaties, or rulings of the United States or any political subdivision or any authority thereof or therein having the power to tax, or any change in the application or official interpretation of such laws or regulations or rulings (including a holding by a court of competent jurisdiction in the United States), which change or amendment is enacted or adopted on or after the issue date of such series of Notes; provided that, prior to the publication of any notice of redemption pursuant to this paragraph, we have delivered to the trustee a certificate signed by one of our officers stating that we are entitled to effect such redemption and setting forth a statement of facts showing that the conditions precedent to our right so to redeem have occurred; or

(ii) on or after the issue date of such series of Notes, any action is taken by a taxing authority of, or any decision has been rendered by a court of competent jurisdiction in, the United States or any political subdivision of or in the United States or any authority thereof or therein having the power to tax, including any of those actions specified in clause (i) above, whether or not such action was taken or decision was rendered with respect to us, or any change, amendment, application or interpretation is officially proposed, which, in any such case, in the written opinion of independent tax counsel of nationally recognized standing, will result in a material probability that we will become obliged to pay additional amounts with respect to such series of Notes; provided that, prior to the publication of any notice of redemption pursuant to this paragraph, we have delivered to the trustee a certificate signed by one of our officers stating that we are entitled to effect such redemption and setting forth a statement of facts showing that the conditions precedent to our right so to redeem have occurred. However, no such notice of redemption shall be given less than 30 or more than 90 days prior to the earliest date on which we would be obliged to pay such additional amounts if a payment in respect of such series of Notes were then due.

0.500% 2022 Notes, 2025 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes, 2035 Notes, 2036 Notes, 2038 Notes and 2039 Notes

Each of the 0.500% 2022 Notes, 2025 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes, 2035 Notes, 2036 Notes, 2038 Notes and 2039 Notes may be redeemed at our option, in whole but not in part, at any time on giving not less than 30 nor more than 90 days' notice to the noteholders (which notice shall be irrevocable), at their principal amount, together with interest accrued to the date fixed for redemption, if:

(i) we have or will become obliged to pay additional amounts with respect to such series of Notes as provided or referred to under "-Withholding Taxes-*0.500% 2022 Notes, 2025 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes, 2035 Notes, 2036 Notes, 2038 Notes and 2039 Notes*" below as a result of any change in, or amendment to, the laws, treaties, or rulings of the United States or any political subdivision or any authority thereof or therein having the power to tax, or any change in the application or official interpretation of such laws or regulations or rulings (including a holding by a court of competent jurisdiction in the United States), which change or amendment is enacted or adopted on or after the issue date of such series of Notes; or

(ii) on or after the issue date of such series of Notes, any action is taken by a taxing authority of, or any decision has been rendered by a court of competent jurisdiction in, the United States or any political subdivision of or in the United States or any authority thereof or therein having the power to tax, including any of those actions specified in clause (i) above, whether or not such action was taken or decision was rendered with respect to us, or any change, amendment, application or interpretation is officially proposed, which, in any such case, will result in a material probability that we will become obliged to pay additional amounts with respect to such series of Notes; provided that, prior to the publication of any notice of redemption pursuant to this paragraph, we have delivered to the trustee a certificate signed by one of our officers stating that we are entitled to effect such redemption and setting forth a statement of facts showing that the conditions precedent to our right so to redeem have occurred and a copy of an opinion of a reputable independent counsel of our choosing to that effect based on that statement of facts. However no such notice of redemption shall be given less than 30 nor more than 90 days prior to the earliest date on which we would be obliged to pay such additional amounts if a payment in respect of such series of Notes were then due.

Withholding Taxes

For purposes of all clauses described under "-Withholding Taxes", references to the holder or beneficial owner of a Note include a fiduciary, settlor, beneficiary or person holding power over such holder or beneficial owner, if such holder or beneficial owner is an estate or trust, or a partner, member or shareholder of such holder or beneficial owner, if such holder or beneficial owner is a partnership, limited liability company or corporation. In addition, we will not pay additional amounts to the holder of a Note if such holder or the beneficial owner of such Note is a fiduciary, partnership, limited liability company or other fiscally transparent entity, or if the holder of such Note is not the sole beneficial owner of such Note, as the case may be, to the extent that a beneficiary or settlor with respect to the fiduciary, or a beneficiary, partner or member of the partnership, limited liability company or other fiscally transparent entity, or a beneficial owner would not have been entitled to the payment of an additional amount had the beneficiary, settlor, beneficial owner, partner or member received directly its beneficial or distributive share of the payment. For purposes of "-Withholding Taxes," the term "Non-U.S. Person" means any person that is, for U.S. federal income tax purposes, a foreign corporation, nonresident alien individual, a nonresident fiduciary of a foreign estate or foreign trust or a foreign partnership one or more of the partners of which is such a foreign corporation, nonresident alien individual or nonresident fiduciary.

Any additional amounts paid pursuant to any clause described under "-Withholding Taxes" on the Euro Notes or the Sterling Notes will be paid in euro or GBP, respectively.

2.375% 2022 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes and 2034 Notes

All payments of principal, interest and premium (if any) in respect of the 2.375% 2022 Notes, 4.073% 2024 Notes, 3.250% 2026 Notes and 2034 Notes by us or a paying agent on our behalf shall be made without withholding or deduction for or on account of any present or future taxes, duties, assessments or other governmental charges ("Taxes") imposed by or on behalf of the United States or any political subdivision thereof or any authority therein or thereof having the power to tax, unless the withholding or deduction of such Taxes is required by law. In that event, we shall pay to a holder that is a Non-U.S. Person such additional amounts as may be necessary to ensure that the net amount received by such holder, after withholding or deduction for or on account of such Taxes, will be equal to the amount such holder would have received in the absence of such withholding or deduction. However, no additional amounts shall be payable with respect to any Note if the beneficial owner is subject to taxation solely for reasons other than its ownership of Notes, nor shall additional amounts be payable for or on account of:

(i) any Tax that would not have been imposed, withheld or deducted but for any present or former connection (other than the mere fact of being a holder or beneficial owner of such Note) between the holder or the beneficial owner of such Note and the United States or the applicable political subdivision or authority, including, without limitation, such holder or beneficial owner being or having been a citizen or resident of the United States or the applicable political subdivision or authority or treated as being or having been a resident thereof;

(ii) any Tax that would not have been imposed, withheld or deducted but for the holder or beneficial owner of such Note being or having been with respect to the United States a personal holding company, a controlled foreign corporation, a passive foreign investment company, a foreign private foundation or other foreign tax-exempt organization, or a corporation that accumulates earnings to avoid U.S. federal income tax;

(iii) any Tax that is payable other than by withholding or deduction by us or a paying agent from payments in respect of such Note;

(iv) any gift, estate, inheritance, sales, transfer, personal property, excise or similar Tax;

(v) any Tax that would not have been imposed, withheld or deducted but for a change in any law, treaty, regulation, or administrative or judicial interpretation that becomes effective after the applicable payment becomes due or is duly provided for, whichever occurs later, to the extent such change in law, treaty, regulation or administrative interpretation would apply retroactively to such payment;

(vi) any Tax that would not have been imposed, withheld or deducted but for the presentation of such Note more than 30 days after the applicable payment becomes due or is duly provided for, whichever occurs later, except to the extent that such holder would have been entitled to such additional amounts on presenting such Note for payment on the last date of such period of 30 days;

(vii) any Tax that would not have been imposed, withheld or deducted but for the failure of a direct or indirect holder or beneficial owner of such Note to comply with applicable certification, information, documentation or other reporting requirements concerning the nationality, residence, identity or connection with the United States of such holder or beneficial owner;

(viii) any Tax that would not have been imposed, withheld or deducted but for the failure of the holder or beneficial owner of such Note to meet the requirements (including the statement requirements) of Section 871(h) or Section 881(c) of the Internal Revenue Code of 1986, as amended (the "Code"); or

(ix) any combination of items (i)-(viii).

1.625% 2024 Notes and 2.625% 2031 Notes

All payments of principal, interest and premium (if any) in respect of the 1.625% 2024 Notes and 2.625% 2031 Notes by us or a paying agent on our behalf shall be made without withholding or deduction for or on account of any Taxes imposed by or on behalf of the United States or any political subdivision thereof or any authority therein or thereof having the power to tax, unless the withholding or deduction of such Taxes is required by law. In that event, we shall pay to a holder that is a Non-U.S. Person such additional amounts as may be necessary to ensure that the net amount received by such holder, after withholding or deduction for or on account of such Taxes, will be equal to the amount such holder would have received in the absence of such withholding or deduction. However, no additional amounts shall be payable with respect to any Note if the beneficial owner is subject to taxation solely for reasons other than its ownership of Notes, nor shall additional amounts be payable for or on account of:

(i) any Tax that would not have been imposed, withheld or deducted but for any present or former connection (other than the mere fact of being a holder or beneficial owner of such Note) between the holder or the beneficial owner of such Note and the United States or the applicable political subdivision or authority, including, without limitation, such holder or beneficial owner being or having been a citizen or resident of the United States or the applicable political subdivision or authority or treated as being or having been a resident thereof;

(ii) any Tax that would not have been imposed, withheld or deducted but for the holder or beneficial owner of such Note being or having been with respect to the United States a personal holding company, a controlled foreign corporation, a passive

foreign investment company, a foreign private foundation or other foreign tax-exempt organization, or a corporation that accumulates earnings to avoid U.S. federal income tax;

(iii) any Tax that is payable other than by withholding or deduction by us or a paying agent from payments in respect of such Note;

(iv) any gift, estate, inheritance, sales, transfer, personal property, excise or similar Tax;

(v) any Tax that would not have been imposed, withheld or deducted but for a change in any law, treaty, regulation, or administrative or judicial interpretation that becomes effective after the applicable payment becomes due or is duly provided for, whichever occurs later, to the extent such change in law, treaty, regulation or administrative interpretation would apply retroactively to such payment;

(vi) any Tax that would not have been imposed, withheld or deducted but for the presentation of such Note more than 30 days after the applicable payment becomes due or is duly provided for, whichever occurs later, except to the extent that such holder would have been entitled to such additional amounts on presenting such Note for payment on the last date of such period of 30 days;

(vii) any Tax that would not have been imposed, withheld or deducted but for the failure of a direct or indirect holder or beneficial owner of such Note to comply with applicable certification, information, documentation or other reporting requirements concerning the nationality, residence, identity or connection with the United States of such holder or beneficial owner;

(viii) any Tax that would not have been imposed, withheld or deducted but for the failure of the holder or beneficial owner of such Note to meet the requirements (including the statement requirements) of Section 871(h) or Section 881(c) of the Code; or

(ix) any Tax imposed pursuant to Sections 1471 through 1474 of the Code, any current or future regulations or official interpretations thereof, any agreements entered pursuant to Section 1471(b) of the Code and any intergovernmental agreements (and related legislation or official administrative guidance) implementing the foregoing; or

(x) any combination of items (i)-(ix).

0.500% 2022 Notes, 2025 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes, 2035 Notes, 2036 Notes, 2038 Notes and 2039 Notes

All payments of principal, interest and premium (if any) in respect of the 0.500% 2022 Notes, 2025 Notes, 1.375% 2026 Notes, 2027 Notes, 2028 Notes, 2029 Notes, 1.250% 2030 Notes, 1.875% 2030 Notes, 2.500% 2031 Notes, 2032 Notes, 2035 Notes, 2036 Notes, 2038 Notes and 2039 Notes by us or a paying agent on our behalf shall be made without withholding or deduction for or on account of any Taxes imposed by or on behalf of the United States or any political subdivision thereof or any authority therein or thereof having the power to tax, unless the withholding or deduction of such Taxes is required by law. In that event, we shall pay to a holder that is a Non-U.S. Person such additional amounts as may be necessary to ensure that the net amount received by such holder, after withholding or deduction for or on account of such Taxes, will be equal to the amount such holder would have received in the absence of such withholding or deduction. However, no additional amounts shall be payable with respect to any Note if the beneficial owner is subject to taxation solely for reasons other than its ownership of Notes, nor shall additional amounts be payable for or on account of:

(i) any Tax that would not have been imposed, withheld or deducted but for any present or former connection (other than the mere fact of being a holder or beneficial owner of such Note) between the holder or the beneficial owner of such Note and the United States or the applicable political subdivision or authority, including, without limitation, such holder or beneficial owner being or having been a citizen or resident of the United States or the applicable political subdivision or authority or treated as being or having been a resident thereof;

(ii) any Tax that would not have been imposed, withheld or deducted but for the holder or beneficial owner of such Note being or having been with respect to the United States a personal holding company, a controlled foreign corporation, a passive foreign investment company, a foreign private foundation or other foreign tax-exempt organization, or a corporation that accumulates earnings to avoid U.S. federal income tax;

(iii) any Tax that is payable other than by withholding or deduction by us or a paying agent from payments in respect of such Note;

(iv) any gift, estate, inheritance, sales, transfer, value added, personal property, excise or similar Tax;

(v) any Tax that would not have been imposed, withheld or deducted but for a change in any law, treaty, regulation, or administrative or judicial interpretation that becomes effective after the applicable payment becomes due or is duly provided for, whichever occurs later;

(vi) any Tax that would not have been imposed, withheld or deducted but for the presentation of such Note for payment more than 30 days after the applicable payment becomes due or is duly provided for, whichever occurs later, except to the extent

that such holder would have been entitled to such additional amounts on presenting such Note for payment on the last date of such period of 30 days;

(vii) any Tax that would not have been imposed, withheld or deducted but for the failure of the holder or beneficial owner of such Note to comply with applicable certification, information, documentation or other reporting requirements concerning the nationality, residence, identity or connection with the United States of such holder or beneficial owner;

(viii) any Tax that would not have been imposed, withheld or deducted but for the failure of the holder or beneficial owner (or any financial institution or other person through which the holder or beneficial owner holds any Notes) to comply with any certification, information, identification, documentation or other reporting requirements with respect to itself or any beneficial owner or account holders thereof;

(ix) any Tax that would not have been imposed, withheld or deducted but for the failure of the holder or beneficial owner of such Note to meet the requirements (including the statement requirements) of Section 871(h) or Section 881(c) of the Code;

(x) any Tax imposed by the Foreign Account Tax Compliance Act pursuant to Sections 1471 through 1474 of the Code, any current or future regulations or official interpretations thereof, any agreements entered into pursuant to Section 1471(b) of the Code and any intergovernmental agreements (and related legislation or official administrative guidance) implementing the foregoing; or

(xi) any combination of items (i)-(x).

Book-Entry Only Form

Each series of Notes was issued in book-entry only form, which means that it is represented by one or more permanent global securities registered in the name of The Depository Trust Company, New York, New York ("DTC"), or its nominee. We refer to this form as "book-entry only."

For debt securities issued in book-entry only form, DTC keeps a computerized record of its participants (for example, a broker) whose clients have purchased the securities. Each participant then keeps a record of its clients who purchased the securities. A global security may not be transferred, except that DTC, its nominees and their successors may transfer an entire global security to one another.

For book-entry only debt securities, we wire principal and interest payments to DTC's nominee. We and the trustee treat DTC's nominee as the owner of the global securities for all purposes. Accordingly, neither we nor the trustee have any direct responsibility or liability to pay amounts due on the debt securities issued under the Indenture to owners of beneficial interests in the global securities.

Under book-entry only form, we have not issued physical certificates representing beneficial interests in the global securities to individual holders of the debt securities. Beneficial interests in global securities will be shown on, and transfers of global securities will be made only through, records maintained by DTC and its participants and will be exchangeable for debt securities in certificated form with the same terms in authorized denominations only if:

- DTC notifies us that it is unwilling or unable to continue as depository;
- DTC ceases to be a clearing agency registered under applicable law and a successor depository is not appointed by us within 90 days; or
- We instruct the trustee that the global security is exchangeable for debt securities in certificated form.

Defeasance

The Indenture permits us to discharge or defease certain of our obligations on any series of debt securities issued under the Indenture at any time. We may defease such obligations relating to a series of debt securities by depositing with the trustee sufficient cash or government securities to pay all sums due on that series of debt securities.

Liens on Assets

The Notes and other debt securities will not be secured. However, if at any time we mortgage, pledge or subject to any lien any of our property or assets, the Indenture requires us to secure the Notes and other debt securities issued under the Indenture equally and ratably with the debt or obligations secured by such mortgage, pledge or lien for as long as such debt or obligations remain secured. Exceptions to this requirement include the following:

- purchase-money mortgages or liens;
- liens on any property or asset that existed at the time when we acquired that property or asset;
- any deposit or pledge to secure public or statutory obligations;
- any deposit or pledge with any governmental agency required to qualify us to conduct any part of our business, to entitle us to maintain self-insurance or to obtain the benefits of any law relating to workmen's compensation, unemployment insurance, old age pensions or other social security; or

- any deposit or pledge with any court, board, commission or governmental agency as security for the proper conduct of any proceeding before it.

The Indenture does not prevent any of our affiliates from mortgaging, pledging or subjecting to any lien, any property or asset, even if the affiliate acquired that property or asset from us.

We may issue or assume an unlimited amount of debt under the Indenture.

Changes to the Indenture

The Indenture may be changed with the consent of holders owning more than 50% of the principal amount of the outstanding debt securities of each series affected by the change. However, we may not change principal or interest payment terms of the Notes or the percentage required to change other terms of the Indenture without consent of the holders of the Notes and the consent of others similarly affected.

We may enter into supplemental indentures for other specified purposes, including the creation of any new series of debt securities, without the consent of any holder of debt securities issued under the Indenture.

Consolidation, Merger or Sale

The Indenture provides that we may not merge with another company or sell, transfer or lease all or substantially all of our property to another company unless:

- the successor corporation expressly assumes:
- payment of principal, interest and any premium on the debt securities issued under the Indenture; and
- performance and observance of all covenants and conditions in the Indenture;
- after giving effect to the transaction, there is no default under the Indenture;
- we have delivered to the trustee an officers' certificate and opinion of counsel stating that such transaction complies with the conditions set forth in the Indenture; and
- if as a result of the transaction, our property would become subject to a lien that would not be permitted by the asset lien restriction, we secure the debt securities Issued under the Indenture equally and ratably with, or prior to, all indebtedness secured by that lien.

Events of Default

An event of default means, for any series of debt securities issued under the Indenture, any of the following:

- failure to pay interest on that series of debt securities for 90 days after payment is due;
- failure to pay principal or any premium on that series of debt securities when due;
- failure to perform any other covenant relating to that series of debt securities for 90 days after notice to us;
- certain events of bankruptcy, insolvency and reorganization; and
- any other event of default provided for in the supplement to the Indenture, board resolution or officers' certificate designating the specific terms of such series of debt securities.

An event of default for a particular series of debt securities does not necessarily impact any other series of debt securities issued under the Indenture.

If an event of default for any series of debt securities occurs and continues, the trustee or the holders of at least 25% of the outstanding principal amount of the debt securities of such series may declare the entire principal of all the debt securities of that series to be due and payable immediately. If this happens, subject to certain conditions, the holders of a majority of the outstanding principal amount of the debt securities of that series can rescind the declaration if there has been deposited with the trustee a sum sufficient to pay all matured installments of interest, principal and any premium.

The holders of more than 50% of the outstanding principal amount of any series of the debt securities, may, on behalf of the holders of all of the debt securities of that series, control any proceedings resulting from an event of default or waive any past default except a default in the payment of principal, interest or any premium. We are required to file an annual certificate with the trustee stating whether we are in compliance with all of the conditions and covenants under the Indenture.

Concerning the Trustee

Within 90 days after a default occurs with respect to a particular series of Notes, the trustee must notify the holders of such series of Notes of all defaults known to the trustee if we have not remedied them (default is defined to mean any event which is, or after notice or lapse of time or both would become, an event of default with respect to such series of Notes as specified above under "-Events of Default"). If a default described in the third bullet point under "-Events of Default" occurs, the trustee will not give notice to the holders of the series until at

least 60 days after the occurrence of that default. The trustee may withhold notice to the holders of the Notes of any default (except in the payment of principal, interest or any premium) if it in good faith believes that withholding this notice is in the interest of the holders.

Prior to an event of default, the trustee is required to perform only the specific duties stated in the Indenture, and after an event of default, must exercise the same degree of care as a prudent individual would exercise in the conduct of his or her own affairs. The trustee is not required to take any action permitted by the Indenture at the request of holders of the debt securities, unless those holders protect the trustee against costs, expenses and liabilities. The trustee is not required to spend its own funds or become financially liable when performing its duties if it reasonably believes that it will not be adequately protected financially.

U.S. Bank National Association, the trustee for the Notes, and its affiliates have commercial banking relationships with us and some of our affiliates and serves as trustee or paying agent under indentures relating to debt securities issued by us and some of our affiliates.

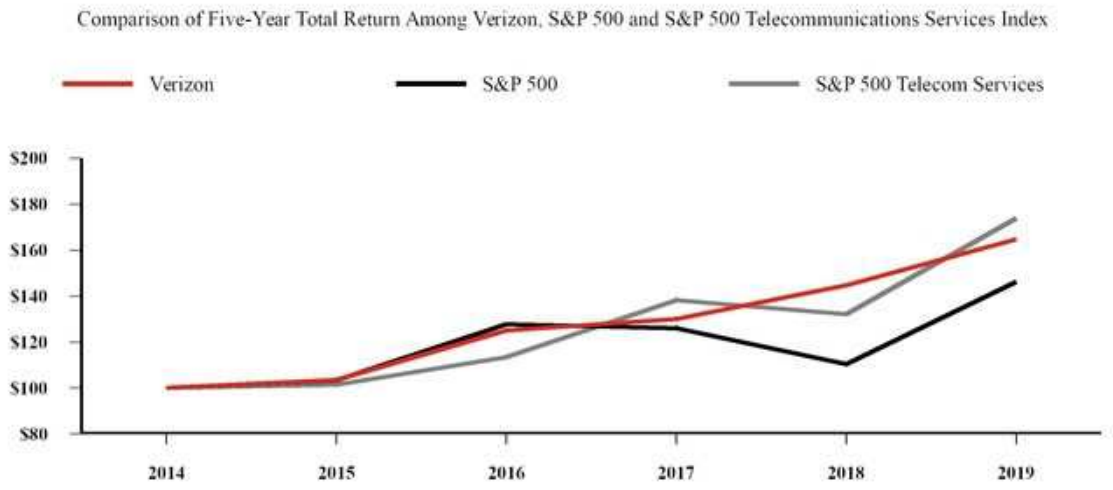
Selected Financial Data Verizon Communications Inc. and Subsidiaries

(dollars in millions, except per share amounts)

	2019	2018	2017	2016	2015
Results of Operations					
Operating revenues	**$ 131,868**	$ 130,863	$ 126,034	$ 125,980	$ 131,620
Operating income	**30,378**	22,278	27,425	29,249	30,615
Net income attributable to Verizon	**19,265**	15,528	30,101	13,127	17,879
Per common share – basic	**4.66**	3.76	7.37	3.22	4.38
Per common share – diluted	**4.65**	3.76	7.36	3.21	4.37
Cash dividends declared per common share	**2.435**	2.385	2.335	2.285	2.230
Net income attributable to noncontrolling interests	**523**	511	449	481	496
Financial Position					
Total assets	**$ 291,727**	$ 264,829	$ 257,143	$ 244,180	$ 244,175
Debt maturing within one year	**10,777**	7,190	3,453	2,645	6,489
Long-term debt	**100,712**	105,873	113,642	105,433	103,240
Employee benefit obligations	**17,952**	18,599	22,112	26,166	29,957
Noncontrolling interests	**1,440**	1,565	1,591	1,508	1,414
Equity attributable to Verizon	**61,395**	53,145	43,096	22,524	16,428

- Significant events affecting our historical earnings trends in 2018 through 2019 are described in "Special Items" in the "Management's Discussion and Analysis of Financial Condition and Results of Operations" section.
- 2017 data includes severance, pension and benefit charges, gain on spectrum license transactions, acquisition and integration related charges, product realignment charges, net gain on sale of divested businesses and early debt redemption costs. 2016 data includes severance, pension and benefit charges, gain on spectrum license transactions, net gain on sale of divested businesses and early debt redemption costs. 2015 data includes severance, pension and benefit credits and gain on spectrum license transactions.
- On January 1, 2019, we adopted several Accounting Standards Updates (ASUs) that were issued by the Financial Accounting Standards Board (FASB) using the modified retrospective basis. On January 1, 2018, we adopted several ASUs that were issued by the FASB. These standards were adopted on different bases, including: (1) prospective; (2) full retrospective; and (3) modified retrospective. Based on the method of adoption, certain figures are not comparable, with full retrospective reflected in all periods. See Note 1 to the consolidated financial statements for additional information.

Stock Performance Graph



	2014	2015	2016	2017	2018	2019
Verizon	$ 100.0	$ 103.6	$ 125.1	$ 130.1	$ 144.7	**$ 164.8**
S&P 500	100.0	103.4	127.7	126.1	110.3	**146.3**
S&P 500 Telecom Services	100.0	101.4	113.5	138.3	132.2	**173.8**

The graph compares the cumulative total returns of Verizon, the S&P 500 Stock Index and the S&P 500 Telecommunications Services Index over a five-year period. It assumes $100 was invested on December 31, 2014 with dividends being reinvested.

Management's Discussion and Analysis of Financial Condition and Results of Operations

Overview

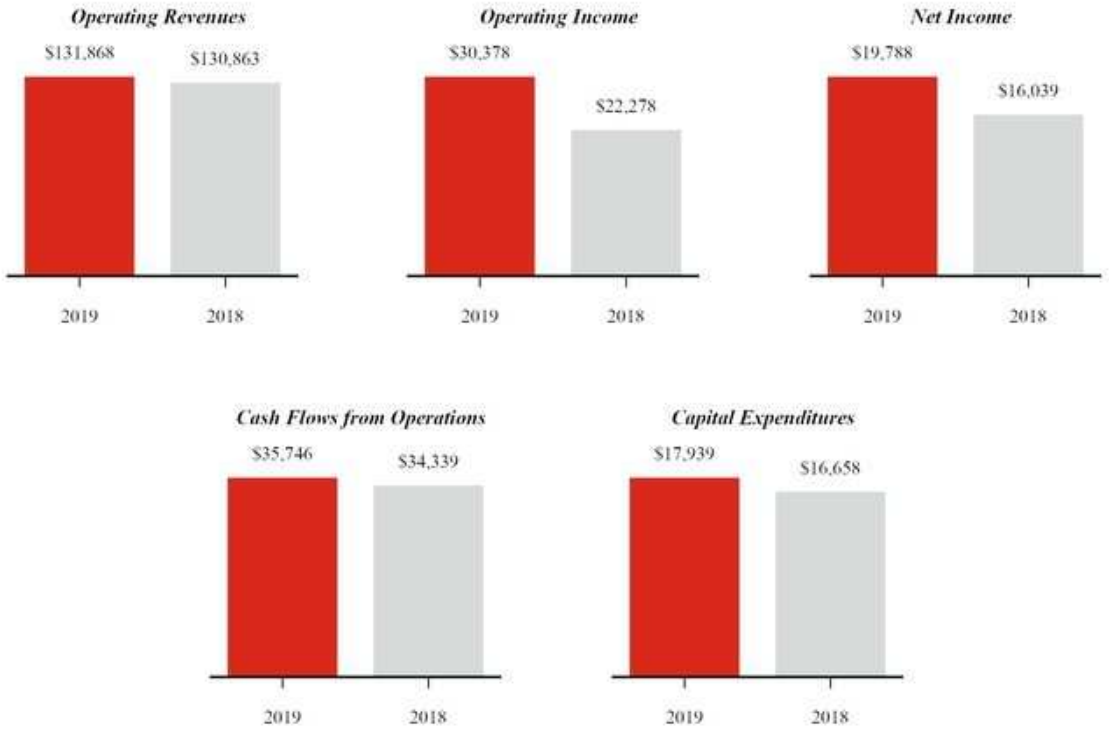
Verizon Communications Inc. (Verizon or the Company) is a holding company that, acting through its subsidiaries, is one of the world's leading providers of communications, information and entertainment products and services to consumers, businesses and government entities. With a presence around the world, we offer voice, data and video services and solutions on our networks that are designed to meet customers' demand for mobility, reliable network connectivity, security and control. We have a highly diverse workforce of approximately 135,000 employees as of December 31, 2019.

To compete effectively in today's dynamic marketplace, we are focused on the capabilities of our high-performing networks to drive growth based on delivering what customers want and need in the new digital world. During 2019, we focused on leveraging our network leadership; retaining and growing our high-quality customer base while balancing profitability; enhancing ecosystems in growth businesses; and driving monetization of our networks and solutions. We are creating business value by earning customers', employees' and shareholders' trust, limiting our environmental impact and continuing our customer growth while creating social benefit through our products and services. Our strategy requires significant capital investments primarily to acquire wireless spectrum, put the spectrum into service, provide additional capacity for growth in our networks, invest in the fiber that supports our businesses, evolve and maintain our networks and develop and maintain significant advanced information technology systems and data system capabilities. We believe that steady and consistent investments in our networks and platforms will drive innovative products and services and fuel our growth.

We are consistently deploying new network architecture and technologies to extend our leadership in both fourth-generation (4G) and fifth-generation (5G) wireless networks. We expect that our next-generation multi-use platform, which we call the Intelligent Edge Network, will simplify operations by eliminating legacy network elements, improve 4G Long-Term Evolution (LTE) wireless coverage, speed the deployment of 5G wireless technology and create new opportunities in the business market. Our network leadership is the hallmark of our brand and the foundation for the connectivity, platform and solutions upon which we build our competitive advantage.

Highlights of Our 2019 Financial Results

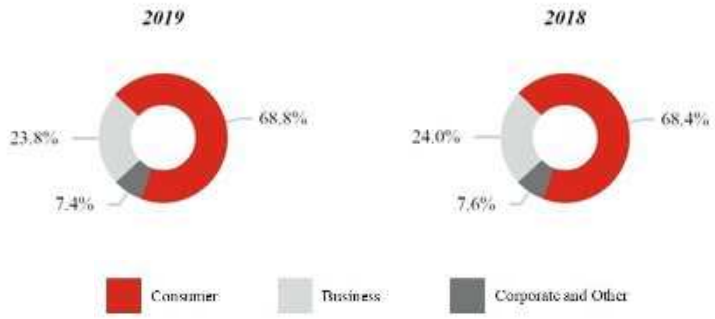
(dollars in millions)



Business Overview

In November 2018, we announced a strategic reorganization of our business. Under the new structure, effective April 1, 2019, there are two reportable segments that we operate and manage as strategic business units - Verizon Consumer Group (Consumer) and Verizon Business Group (Business).

Revenue by Segment



Note: Excludes eliminations.

Verizon Consumer Group

Our Consumer segment provides consumer-focused wireless and wireline communications services and products. Our wireless services are provided across one of the most extensive wireless networks in the United States (U.S.) under the Verizon brand and through wholesale and other arrangements. Our wireline services are provided in nine states in the Mid-Atlantic and Northeastern U.S., as well as Washington D.C., over our 100% fiber-optic network under the Fios brand and over a traditional copper-based network to customers who are not served by Fios. Our Consumer segment's wireless and wireline products and services are available to our retail customers, as well as resellers that purchase wireless network access from us on a wholesale basis.

Customers can obtain our wireless services on a postpaid or prepaid basis. A retail postpaid connection represents an individual line of service for a wireless device for which a customer is generally billed one month in advance for a monthly access charge in return for access to and usage of network services. Our prepaid service is offered only to Consumer customers and enables individuals to obtain wireless services without credit verification by paying for all services in advance. The Consumer segment also offers several categories of wireless equipment to customers, including a variety of smartphones and other handsets, wireless-enabled Internet devices, such as tablets, laptop computers and netbooks, and other wireless-enabled connected devices, such as smart watches and other wearables.

In addition to the wireless services and equipment discussed above, Consumer sells residential fixed connectivity solutions, including Internet, video and voice services, and wireless network access to resellers on a wholesale basis. The Consumer segment's operating revenues for the year ended December 31, 2019 totaled $91.1 billion, an increase of $1.3 billion, or 1.4%, compared to the year ended December 31, 2018. As of December 31, 2019, Consumer had approximately 95 million wireless retail connections, 6 million broadband connections and 4 million Fios video connections.

Verizon Business Group

Our Business segment provides wireless and wireline communications services and products, video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various Internet of Things (IoT) services and products, including solutions that support fleet tracking management, compliance management, field service management, asset tracking and other types of mobile resource management. We provide these products and services to businesses, government customers and wireless and wireline carriers across the U.S. and select products and services to customers around the world. The Business segment's operating revenues for the year ended December 31, 2019 totaled $31.4 billion, a decrease of $91 million, or 0.3%, compared to the year ended December 31, 2018. As of December 31, 2019, Business had approximately 25 million wireless retail postpaid connections and 489 thousand broadband connections.

Corporate and Other

Corporate and other includes the results of our media business, Verizon Media, and other businesses, investments in unconsolidated businesses, insurance captives, unallocated corporate expenses, certain pension and other employee benefit related costs and interest and financing expenses. Corporate and other also includes the historical results of divested businesses and other adjustments and gains and losses that are not allocated in assessing segment performance due to their nature. Although such transactions are excluded from the business segment results, they are included in reported consolidated earnings. Gains and losses from transactions that are not individually significant are included in segment results as these items are included in the chief operating decision maker’s assessment of segment performance.

Verizon Media includes diverse media and technology brands that serve both consumers and businesses. Verizon Media provides consumers with owned and operated and third-party search properties as well as mail, news, finance, sports and entertainment offerings, and provides other businesses and partners access to consumers through digital advertising, content delivery and video streaming platforms. Verizon Media's total operating revenues were $7.5 billion for the year ended December 31, 2019. This was a decrease of 3.0% from the year ended December 31, 2018.

Capital Expenditures and Investments

We continue to invest in our wireless networks, high-speed fiber and other advanced technologies to position ourselves at the center of growth trends for the future. During the year ended December 31, 2019, these investments included $17.9 billion for capital expenditures. See "Cash Flows Used in Investing Activities" and "Operating Environment and Trends" for additional information. We believe that our investments aimed at expanding our portfolio of products and services will provide our customers with an efficient, reliable infrastructure for competing in the information economy.

Global Network and Technology

We are focusing our capital spending on adding capacity and density to our 4G LTE network, while also building our next generation 5G network. We are densifying our network by utilizing small cell technology, in-building solutions and distributed antenna systems. Network densification enables us to add capacity to address increasing mobile video consumption and the growing demand for IoT products and services on our 4G LTE and 5G networks. Over the past several years, we have been leading the development of 5G wireless technology industry standards and the ecosystems for fixed and mobile 5G wireless services. We believe 5G technology will be able to provide users with eight capabilities, or currencies. The eight currencies are peak data rates, mobile data volumes, mobility, number of connected devices, energy efficiency of connected devices, service deployment, reduced latency and improved reliability. We expect that 5G technology will provide higher throughput and lower latency than the current 4G LTE technology and enable our networks to handle more traffic as the number of Internet-connected devices grows. During 2018, we commercially launched 5G Home on proprietary standards in four U.S. markets and on global standards in a fifth U.S. market in 2019. We also launched our 5G Ultra Wideband Network in 31 U.S. markets in 2019, as well as several 5G-compatible smartphones.

To compensate for the shrinking market for traditional copper-based products, we continue to build our wireline business around fiber-based networks supporting data, video and advanced business services - areas where demand for reliable high-speed connections is growing. We are evolving the architecture of our networks to a next-generation multi-use platform, providing improved efficiency and virtualization, increased automation and opportunities for edge computing services that will support both our fiber-based and radio access network technologies. We call this the Intelligent Edge Network. We expect that this new architecture will simplify operations by eliminating legacy network elements, improve our 4G LTE wireless coverage, speed the deployment of 5G wireless technology and create new opportunities in the business market.

Recent Developments

In 2019, the Federal Communications Commission (FCC) completed two millimeter wave spectrum license auctions. Verizon participated in these auctions and was the high bidder on 9 and 1,066 licenses, respectively, in the 24 Gigahertz (GHz) and 28 GHz bands. We submitted an application to the FCC and paid cash of approximately $521 million for the licenses. We received the licenses during the fourth quarter of 2019.

In December 2019, the FCC incentive auction for spectrum licenses in the upper 37 GHz, 39 GHz, and 47 GHz bands commenced. As an incumbent licensee, Verizon received vouchers related to our existing 39 GHz licenses. These vouchers can be converted into cash, the amount of which will not be known until the conclusion of the auction, or applied toward the purchase price of spectrum in the auction. At the conclusion of the auction, all existing licenses will be cancelled and new reconfigured licenses or cash will be distributed depending on the results of the auction. Due to the FCC's rules restricting communications regarding the auction, we will not disclose our financial plans for the auction during the quiet period for this auction unless legally required. In addition, as of this time, until the completion of the auction process, we cannot determine the resulting financial outcome, including a potential gain or loss. Such gain or loss, if any, may be material.

Consolidated Results of Operations

In this section, we discuss our overall results of operations and highlight special items that are not included in our segment results. In "Segment Results of Operations," we review the performance of our two reportable segments in more detail. A detailed discussion of 2017 items and year-over-year comparisons between 2018 and 2017 that are not included in this Form 10-K can be found in the "Management's Discussion and Analysis of Financial Condition and Results of Operations" for the year ended December 31, 2018 filed with our most recent financial statements and included in the Company's Current Report on Form 8-K dated August 8, 2019.

Consolidated Revenues

(dollars in millions)

			Increase/(Decrease)	
Years Ended December 31,	**2019**	2018	**2019 vs. 2018**	
Consumer	**$ 91,056**	$ 89,762	$ 1,294	1.4 %
Business	**31,443**	31,534	(91)	(0.3)
Corporate and other	**9,812**	9,936	(124)	(1.2)
Eliminations	**(443)**	(369)	(74)	20.1
Consolidated Revenues	**$ 131,868**	$ 130,863	$ 1,005	0.8

Consolidated revenues increased $1.0 billion, or 0.8%, during 2019 compared to 2018, primarily due to an increase in revenues at our Consumer segment, partially offset by decreases in revenues at our Business segment and Corporate and other.

Revenues for our segments are discussed separately below under the heading "Segment Results of Operations."

Corporate and other revenues decreased $124 million, or 1.2%, during 2019 compared to 2018, primarily due to a decrease of $232 million in revenues within Verizon Media.

Consolidated Operating Expenses

(dollars in millions)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Cost of services	**$ 31,772**	$ 32,185	$ (413)	(1.3)%
Cost of wireless equipment	**22,954**	23,323	(369)	(1.6)
Selling, general and administrative expense	**29,896**	31,083	(1,187)	(3.8)
Depreciation and amortization expense	**16,682**	17,403	(721)	(4.1)
Media goodwill impairment	**186**	4,591	(4,405)	(95.9)
Consolidated Operating Expenses	**$ 101,490**	$ 108,585	$ (7,095)	(6.5)

Operating expenses for our segments are discussed separately below under the heading "Segment Results of Operations."

Cost of Services

Cost of services includes the following costs directly attributable to a service: salaries and wages, benefits, materials and supplies, content costs, contracted services, network access and transport costs, customer provisioning costs, computer systems support, and costs to support our outsourcing contracts and technical facilities. Aggregate customer care costs, which include billing and service provisioning, are allocated between Cost of services and Selling, general and administrative expense.

Cost of services decreased $413 million, or 1.3%, during 2019 compared to 2018, primarily due to decreases in network access costs, a product realignment charge in 2018 (see "Special Items"), decreases in employee-related costs resulting from the Voluntary Separation Program and decreases in digital content costs. These decreases were partially offset by increases in rent expense as a result of adding capacity to the networks to support demand and the adoption of the new lease accounting standard in 2019, regulatory fees, and costs related to the device protection package offered to our wireless retail postpaid customers.

Cost of Wireless Equipment

Cost of wireless equipment decreased $369 million, or 1.6%, during 2019 compared to 2018, primarily as a result of declines in the number of wireless devices sold as a result of an elongation of the handset upgrade cycle, partially offset by a shift to higher priced devices in the mix of wireless devices sold.

Selling, General and Administrative Expense

Selling, general and administrative expense includes salaries and wages and benefits not directly attributable to a service or product, bad debt charges, taxes other than income taxes, advertising and sales commission costs, call center and information technology costs, regulatory fees, professional service fees, and rent and utilities for administrative space. Also included is a portion of the aggregate customer care costs as discussed above in "Cost of Services."

Selling, general and administrative expense decreased $1.2 billion, or 3.8%, during 2019 compared to 2018, primarily due to decreases in employee-related costs primarily due to the Voluntary Separation Program, a decrease in severance, pension and benefits charges (see "Special Items"), the acquisition and integration related charges in 2018 primarily related to the acquisition of Yahoo's operating business (see "Special Items") and a net gain from dispositions of assets and businesses in 2019 (see "Special Items"), partially offset by increases in advertising expenses, sales commission and bad debt expense. The increase in sales commission expense during 2019 compared to 2018, was primarily due to a lower net deferral of commission costs as a result of the adoption of Topic 606 on January 1, 2018, using a modified retrospective approach.

Depreciation and Amortization Expense

Depreciation and amortization expense decreased $721 million, or 4.1%, during 2019 compared to 2018, primarily due to the change in the mix of net depreciable assets.

Media Goodwill Impairment

The goodwill impairment charges recorded in 2019 and 2018 for Verizon Media were a result of the Company's annual impairment test performed in the fourth quarter (see "Critical Accounting Estimates").

Other Consolidated Results

Other Income (Expense), Net

Additional information relating to Other income (expense), net is as follows:

(dollars in millions)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Interest income	**$ 121**	$ 94	$ 27	28.7 %
Other components of net periodic benefit cost	**627**	3,068	(2,441)	(79.6)
Early debt extinguishment costs	**(3,604)**	(725)	(2,879)	nm
Other, net	**(44)**	(73)	29	39.7
Total	**$ (2,900)**	$ 2,364	$ (5,264)	nm

nm - not meaningful

The change in Other income (expense), net during the year ended December 31, 2019, compared to the similar period in 2018, was primarily driven by early debt redemption costs of $3.6 billion recorded during 2019, compared to $725 million recorded during 2018 (see "Special Items") as well as pension and benefit charges of $126 million recorded in 2019, compared with pension and benefit credits of $2.1 billion recorded in 2018 (see "Special Items").

Interest Expense

(dollars in millions)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Total interest costs on debt balances	**$ 5,386**	$ 5,573	$ (187)	(3.4)%
Less capitalized interest costs	**656**	740	(84)	(11.4)
Total	**$ 4,730**	$ 4,833	$ (103)	(2.1)
Average debt outstanding	**$ 112,901**	$ 115,858		
Effective interest rate	**4.8%**	4.8%		

Total interest costs on debt balances decreased during 2019 primarily due to lower average debt balances.

Provision for Income Taxes

(dollars in millions)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Provision for income taxes	**$ 2,945**	$ 3,584	$ (639)	(17.8)%
Effective income tax rate	**13.0%**	18.3%		

The effective income tax rate is calculated by dividing the provision for income taxes by income before income taxes. The effective income tax rate for 2019 was 13.0% compared to 18.3% for 2018. The decrease in the effective income tax rate and the provision for income taxes was primarily due to the recognition of approximately $2.2 billion of a non-recurring tax benefit in connection with the disposition of preferred stock, representing a minority interest in a foreign affiliate in 2019 compared to the non-recurring deferred tax benefit of approximately $2.1 billion as a result of an internal reorganization of legal entities within the historical Wireless business, which was offset by a goodwill charge that is not deductible for tax purposes in 2018.

A reconciliation of the statutory federal income tax rate to the effective income tax rate for each period is included in Note 12 to the consolidated financial statements.

Consolidated Net Income, Consolidated EBITDA and Consolidated Adjusted EBITDA

Consolidated earnings before interest, taxes, depreciation and amortization expenses (Consolidated EBITDA) and Consolidated Adjusted EBITDA, which are presented below, are non-generally accepted accounting principles (GAAP) measures that we believe are useful to management, investors and other users of our financial information in evaluating operating profitability on a more variable cost basis as they exclude the depreciation and amortization expense related primarily to capital expenditures and acquisitions that occurred in prior years, as well as in evaluating operating performance in relation to Verizon's competitors. Consolidated EBITDA is calculated by adding back interest, taxes, and depreciation and amortization expenses to net income.

Consolidated Adjusted EBITDA is calculated by excluding from Consolidated EBITDA the effect of the following non-operational items: equity in losses of unconsolidated businesses and other income and expense, net, as well as the effect of special items. We believe that this measure is useful to management, investors and other users of our financial information in evaluating the effectiveness of our operations and underlying business trends in a manner that is consistent with management's evaluation of business performance. We believe that Consolidated Adjusted EBITDA is widely used by investors to compare a company's operating performance to its competitors by minimizing impacts caused by differences in capital structure, taxes and depreciation policies. Further, the exclusion of non-operational items and special items enables comparability to prior period performance and trend analysis. See "Special Items" for additional information.

It is management's intent to provide non-GAAP financial information to enhance the understanding of Verizon's GAAP financial information, and it should be considered by the reader in addition to, but not instead of, the financial statements prepared in accordance with GAAP. Each non-GAAP financial measure is presented along with the corresponding GAAP measure so as not to imply that more emphasis should be placed on the non-GAAP measure. We believe that non-GAAP measures provide relevant and useful information, which is used by management, investors and other users of our financial information, as well as by our management in assessing both consolidated and segment performance. The non-GAAP financial information presented may be determined or calculated differently by other companies and may not be directly comparable to that of other companies.

(dollars in millions)

Years Ended December 31,		**2019**		2018
Consolidated Net Income	**$**	**19,788**	$	16,039
Add:				
Provision for income taxes		**2,945**		3,584
Interest expense		**4,730**		4,833
Depreciation and amortization expense		**16,682**		17,403
Consolidated EBITDA		**44,145**		41,859
Add (Less):				
Other (income) expense, net†		**2,900**		(2,364)
Equity in losses of unconsolidated businesses‡		**15**		186
Severance charges		**204**		2,157
Acquisition and integration related charges§		**—**		531
Product realignment charges§		**—**		450
Impairment charges		**186**		4,591
Net gain from dispositions of assets and businesses		**(261)**		—
Consolidated Adjusted EBITDA	**$**	**47,189**	$	47,410

† Includes Pension and benefits mark-to-market adjustments and early debt redemption costs, where applicable.
‡ Includes Product realignment charges and impairment charges, where applicable.
§ Excludes depreciation and amortization expense.

The changes in Consolidated Net Income, Consolidated EBITDA and Consolidated Adjusted EBITDA in the table above were primarily a result of the factors described in connection with operating revenues and operating expenses.

Segment Results of Operations

We have two reportable segments that we operate and manage as strategic business units, Consumer and Business. We measure and evaluate our reportable segments based on segment operating income. The use of segment operating income is consistent with the chief operating decision maker's assessment of segment performance.

To aid in the understanding of segment performance as it relates to segment operating income, we use the following operating statistics to evaluate the overall effectiveness of our segments:

Wireless retail connections are retail customer device postpaid and prepaid connections. Retail connections under an account may include those from smartphones and basic phones (collectively, phones) as well as tablets and other Internet devices, including wearables and retail IoT devices.

Wireless retail postpaid connections are retail postpaid customer device connections. Retail connections under an account may include those from phones, as well as tablets and other Internet devices, including wearables and retail IoT devices.

Fios Internet connections are the total number of connections to the Internet using Fios Internet services.

Fios video connections are the total number of connections to traditional linear video programming using Fios video services.

Broadband connections are the total number of connections to the Internet using Digital Subscriber Line (DSL) and Fios Internet services.

Voice connections are the total number of traditional switched access lines in service and Fios digital voice connections.

Wireless retail connections, net additions are the total number of additional retail customer device postpaid and prepaid connections, less the number of device disconnects within the current period.

Wireless retail postpaid connections, net additions are the total number of additional retail customer device postpaid connections, less the number of device disconnects within the current period.

Churn is the rate at which service to either retail or postpaid retail connections is terminated on a monthly basis.

Wireless retail postpaid ARPA is the calculated average service revenue per account (ARPA) from retail postpaid accounts, which does not include recurring device payment plan billings related to the Verizon device payment program, plan billings related to total mobile protection packages or regulatory fees.

Wireless retail postpaid accounts are retail customers that are directly served and managed under the Verizon brand and use its services. Accounts include unlimited plans, shared data plans and corporate accounts, as well as legacy single connection plans and family plans. A single account may include monthly wireless services for a variety of connected devices.

Wireless retail postpaid connections per account is calculated by dividing the total number of retail postpaid connections by the number of retail postpaid accounts as of the end of the period.

Segment earnings before interest, taxes, depreciation and amortization (Segment EBITDA), which is presented below, is a non-GAAP measure and does not purport to be an alternative to operating income as a measure of operating performance. We believe this measure is useful to management, investors and other users of our financial information in evaluating operating profitability on a more variable cost basis as it excludes the depreciation and amortization expenses related primarily to capital expenditures and acquisitions that occurred in prior years, as well as in evaluating operating performance in relation to our competitors. Segment EBITDA is calculated by adding back depreciation and amortization expense to segment operating income. Segment EBITDA margin is calculated by dividing Segment EBITDA by total segment operating revenues. You can find additional information about our segments in Note 13 to the consolidated financial statements.

Verizon Consumer Group

Our Consumer segment provides consumer-focused wireless and wireline communications services and products. Our wireless services are provided across one of the most extensive wireless networks in the United States under the Verizon brand and through wholesale and other arrangements. Our wireline services are provided in nine states in the Mid-Atlantic and Northeastern U.S., as well as Washington D.C., over our 100% fiber-optic network under the Fios brand and over a traditional copper-based network to customers who are not served by Fios.

Operating Revenues and Selected Operating Statistics

(dollars in millions, except ARPA)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Service	**$ 65,383**	$ 64,223	$ 1,160	1.8 %
Wireless equipment	**18,048**	18,875	(827)	(4.4)
Other	**7,625**	6,664	961	14.4
Total Operating Revenues	**$ 91,056**	$ 89,762	$ 1,294	1.4
Connections ('000):[1]				
Wireless retail connections	**94,544**	94,507	37	—
Wireless retail postpaid connections	**90,481**	89,861	620	0.7
Fios Internet connections	**5,902**	5,760	142	2.5
Fios video connections	**4,152**	4,377	(225)	(5.1)
Broadband connections	**6,467**	6,460	7	0.1
Voice connections	**5,754**	6,332	(578)	(9.1)
Net Additions in Period ('000):[2]				
Wireless retail	**379**	372	7	1.9
Wireless retail postpaid	**970**	1,129	(159)	(14.1)
Wireless retail postpaid phones	**737**	498	239	48.0
Churn Rate:				
Wireless retail	**1.28%**	1.25%		
Wireless retail postpaid	**1.05%**	1.00%		
Wireless retail postpaid phones	**0.79%**	0.76%		
Account Statistics:				
Wireless retail postpaid ARPA	**$ 118.13**	$ 115.48	$ 2.65	2.3
Wireless retail postpaid accounts ('000)[1]	**33,875**	34,086	(211)	(0.6)
Wireless retail postpaid connections per account[1]	**2.67**	2.64	0.03	1.1

[1] As of end of period
[2] Excluding acquisitions and adjustments

Consumer's total operating revenues increased $1.3 billion, or 1.4%, during 2019 compared to 2018, primarily as a result of increases in Service and Other revenues, partially offset by a decrease in Wireless equipment revenue.

Service Revenue

Service revenue increased $1.2 billion, or 1.8%, during 2019 compared to 2018, primarily due to increases in wireless service and Fios revenues, partially offset by decreases in wireline voice and DSL services.

Wireless service revenue increased $1.3 billion, or 2.5%, during 2019 compared to 2018, due to increases in wireless access revenue, driven by customers shifting to higher access plans including unlimited plans and increases in the number of devices per account, the declining fixed-term subsidized plan base and growth from reseller accounts. Wireless retail postpaid ARPA increased 2.3%.

For the year ended December 31, 2019, Fios revenues totaled $10.4 billion and increased $92 million, or 0.9%, compared to 2018. This increase was due to a 2.5% increase in Fios Internet connections, reflecting increased demand in higher broadband speeds, partially offset by a 5.1% decrease in Fios video connections, reflecting the ongoing shift from traditional linear video to over-the-top (OTT) offerings.

Service revenue attributable to wireline voice and DSL broadband services declined during 2019, compared to 2018. The declines are primarily due to a decrease of 9.1% in voice connections resulting primarily from competition and technology substitution with wireless and competing Voice over Internet Protocol (VoIP) and cable telephony services.

Wireless Equipment Revenue

Wireless equipment revenue decreased $827 million, or 4.4%, during 2019 compared to 2018, as a result of declines in wireless device sales primarily due to an elongation of the handset upgrade cycle and increased promotions. These decreases were partially offset by a shift to higher priced units in the mix of wireless devices sold.

Other Revenue

Other revenue includes non-service revenues such as regulatory fees, cost recovery surcharges, revenues associated with our device protection package, leasing and interest on equipment financed under a device payment plan agreement when sold to the customer by an authorized agent.

Other revenue increased $1.0 billion, or 14.4%, during 2019 compared to 2018, primarily due to pricing increases related to our wireless device protection plans, as well as regulatory fees.

Operating Expenses

(dollars in millions)

				Increase/(Decrease)	
Years Ended December 31,	**2019**	2018		**2019 vs. 2018**	
Cost of services	**$ 15,884**	$ 15,335	$	549	3.6 %
Cost of wireless equipment	**18,219**	18,763		(544)	(2.9)
Selling, general and administrative expense	**16,639**	15,701		938	6.0
Depreciation and amortization expense	**11,353**	11,952		(599)	(5.0)
Total Operating Expenses	**$ 62,095**	$ 61,751	$	344	0.6

Cost of Services

Cost of services increased $549 million, or 3.6%, during 2019 compared to 2018, primarily due to increases in rent expense as a result of adding capacity to the networks to support demand as well as an increase due to the adoption of the new lease accounting standard in 2019, increases in costs related to the device protection package offered to our wireless retail postpaid customers, as well as regulatory fees. These increases were partially offset by decreases in employee-related costs primarily due to the Voluntary Separation Program, as well as decreases in access costs and roaming.

Cost of Wireless Equipment

Cost of wireless equipment decreased $544 million, or 2.9%, during 2019 compared to 2018, primarily as a result of declines in the number of wireless devices sold as a result of an elongation of the handset upgrade cycle. These decrease were partially offset by a shift to higher priced devices in the mix of wireless devices sold.

Selling, General and Administrative Expense

Selling, general and administrative expense increased $938 million, or 6.0%, during 2019 compared to 2018, primarily due to increases in sales commission and bad debt expense, and an increase in advertising costs. The increase in sales commission expense during 2019 compared to 2018 was primarily due to a lower net deferral of commission costs as a result of the adoption of Topic 606 on January 1, 2018 using a modified retrospective approach. These increases were partially offset by decreases in employee-related costs primarily due to the Voluntary Separation Program.

Depreciation and Amortization Expense

Depreciation and amortization expense decreased $599 million, or 5.0%, during 2019 compared to 2018, driven by the change in the mix of total Verizon depreciable assets and Consumer's usage of those assets.

Segment Operating Income and EBITDA

(dollars in millions)

				Increase/(Decrease)	
Years Ended December 31,	**2019**	2018		**2019 vs. 2018**	
Segment Operating Income	**$ 28,961**	$ 28,011	$	950	3.4 %
Add Depreciation and amortization expense	**11,353**	11,952		(599)	(5.0)
Segment EBITDA	**$ 40,314**	$ 39,963	$	351	0.9
Segment operating income margin	**31.8%**	31.2%			
Segment EBITDA margin	**44.3%**	44.5%			

The changes in the table above during the periods presented were primarily a result of the factors described in connection with operating revenues and operating expenses.

Verizon Business Group

Our Business segment provides wireless and wireline communications services and products, video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various IoT services and products. We provide these products and services to businesses, government customers and wireless and wireline carriers across the U.S. and select products and services to customers around the world. The Business segment is organized in four customer groups: Global Enterprise, Small and Medium Business, Public Sector and Other, and Wholesale.

Operating Revenues and Selected Operating Statistics

(dollars in millions)

			Increase/(Decrease)	
Years Ended December 31,	**2019**	2018	**2019 vs. 2018**	
Global Enterprise	**$ 10,818**	$ 11,201	$ (383)	(3.4)%
Small and Medium Business	**11,464**	10,752	712	6.6
Public Sector and Other	**5,922**	5,833	89	1.5
Wholesale	**3,239**	3,748	(509)	(13.6)
Total Operating Revenues[1]	**$ 31,443**	$ 31,534	$ (91)	(0.3)
Connections ('000):[2]				
Wireless retail postpaid connections	**25,217**	23,492	1,725	7.3
Fios Internet connections	**326**	307	19	6.2
Fios video connections	**77**	74	3	4.1
Broadband connections	**489**	501	(12)	(2.4)
Voice connections	**4,959**	5,400	(441)	(8.2)
Net Additions in Period ('000): [3]				
Wireless retail postpaid	**1,391**	1,397	(6)	(0.4)
Wireless retail postpaid phones	**698**	625	73	11.7
Churn Rate:				
Wireless retail postpaid	**1.24%**	1.19%		
Wireless retail postpaid phones	**0.99%**	0.98%		

[1] Service and other revenues included in our Business segment amounted to approximately $27.9 billion and $28.1 billion for the years ended December 31, 2019 and 2018, respectively. Wireless equipment revenues included in our Business segment amounted to approximately $3.5 billion and $3.4 billion for the years ended December 31, 2019 and 2018, respectively.
[2] As of end of period
[3] Includes certain adjustments

Business revenues decreased $91 million, or 0.3%, during 2019 compared to 2018, primarily due to decreases in Global Enterprise and Wholesale revenues, partially offset by increases in Small and Medium Business and Public Sector and Other revenues.

Global Enterprise

Global Enterprise offers services to large businesses, which are identified based on their size and volume of business with Verizon, as well as non-U.S. public sector customers.

Global Enterprise revenues decreased $383 million, or 3.4%, during 2019 compared to 2018, primarily due to declines in traditional data and voice communication services as a result of competitive price pressures. These revenue decreases were partially offset by increases in wireless service revenue.

Small and Medium Business

Small and Medium Business offers wireless services and equipment, tailored voice and networking products, Fios services, IP networking, advanced voice solutions, security and managed information technology services to our U.S.-based customers that do not meet the requirements to be categorized as Global Enterprise.

Small and Medium Business revenues increased $712 million, or 6.6%, during 2019 compared to 2018, primarily due to an increase in wireless postpaid service revenue of 11.7% as a result of increases in the amount of wireless retail postpaid connections. These increases were further driven by increased wireless equipment revenue resulting from a shift to higher priced units in the mix of wireless devices sold and increases in

the number of wireless devices sold, increased revenue related to our wireless device protection package, as well as increased revenue related to Fios services. These revenue increases were partially offset by revenue declines related to the loss of voice and DSL service connections.

Small and Medium Business Fios revenues totaled $915 million and increased $110 million, or 13.7%, during 2019 compared to 2018, reflecting the increase in total connections, as well as increased demand for higher broadband speeds.

Public Sector and Other

Public Sector and Other offers wireless products and services as well as wireline connectivity and managed solutions to U.S. federal, state and local governments and educational institutions. These services include the business services and connectivity similar to the products and services offered by Global Enterprise, in each case, with features and pricing designed to address the needs of governments and educational institutions.

Public Sector and Other revenues increased $89 million, or 1.5%, during 2019 compared to 2018, driven by increases in networking and wireless postpaid service revenue as a result of an increase in wireless retail postpaid connections.

Wholesale

Wholesale offers wireline communications services including data, voice, local dial tone and broadband services primarily to local, long distance, and wireless carriers that use our facilities to provide services to their customers.

Wholesale revenues decreased $509 million, or 13.6%, during 2019 compared to 2018, primarily due to declines in core data and traditional voice services resulting from the effect of technology substitution and continuing contraction of market rates due to competition.

Operating Expenses

(dollars in millions)

			Increase/(Decrease)	
Years Ended December 31,	**2019**	2018	**2019 vs. 2018**	
Cost of services	**$ 10,655**	$ 10,859	$ (204)	(1.9)%
Cost of wireless equipment	**4,733**	4,560	173	3.8
Selling, general and administrative expense	**8,188**	7,689	499	6.5
Depreciation and amortization expense	**4,105**	4,258	(153)	(3.6)
Total Operating Expenses	**$ 27,681**	$ 27,366	$ 315	1.2

Cost of Services

Cost of services decreased $204 million, or 1.9%, during 2019 compared to 2018, primarily due to lower access costs resulting from a decline in voice connections, as well as lower employee-related costs associated with the lower headcount resulting from the Voluntary Separation Program, offset by an increase in regulatory fees.

Cost of Wireless Equipment

Cost of wireless equipment increased $173 million, or 3.8%, during 2019 compared to 2018, primarily driven by a shift to higher priced units in the mix of wireless devices sold and an increase in the number of wireless devices sold.

Selling, General and Administrative Expense

Selling, general and administrative expense increased $499 million, or 6.5%, during 2019 compared to 2018, due to increases in advertising expenses and sales commission expense, which were partially offset by decreases in employee-related costs resulting from the Voluntary Separation Program. The increase in sales commission expense was primarily due to a lower net deferral of commission costs in 2019 as compared to 2018 as a result of the adoption of Topic 606 on January 1, 2018 using a modified retrospective approach.

Depreciation and Amortization Expense

Depreciation and amortization expense decreased $153 million, or 3.6%, during 2019 compared to 2018, driven by the change in the mix of total Verizon depreciable assets and Business's usage of those assets.

Segment Operating Income and EBITDA

(dollars in millions)

Years Ended December 31,	2019	2018	Increase/(Decrease) 2019 vs. 2018	
Segment Operating Income	**$ 3,762**	$ 4,168	$ (406)	(9.7)%
Add Depreciation and amortization expense	**4,105**	4,258	(153)	(3.6)
Segment EBITDA	**$ 7,867**	$ 8,426	$ (559)	(6.6)
Segment operating income margin	**12.0%**	13.2%		
Segment EBITDA margin	**25.0%**	26.7%		

The changes in the table above during the periods presented were primarily a result of the factors described in connection with operating revenues and operating expenses.

Special Items

Special items included in Income Before (Provision) Benefit For Income Taxes were as follows:

(dollars in millions)

Years Ended December 31,	2019	2018
Severance, pension and benefits charges (credits)		
Selling, general and administrative expense	**$ 204**	$ 2,157
Other income (expense), net	**126**	(2,107)
Acquisition and integration related charges		
Selling, general and administrative expense	**—**	531
Depreciation and amortization expense	**—**	22
Product realignment charges		
Cost of services	**—**	303
Selling, general and administrative expense	**—**	147
Equity in losses of unconsolidated businesses	**—**	207
Depreciation and amortization expense	**—**	1
Impairment charges		
Media goodwill impairment	**186**	4,591
Equity in losses of unconsolidated businesses	**50**	—
Early debt redemption costs		
Other income (expense), net	**3,604**	725
Net gain from dispositions of assets and businesses		
Selling, general and administrative expense	**(261)**	—
Total	**$ 3,909**	$ 6,577

The Consolidated Adjusted EBITDA non-GAAP measure presented in the Consolidated Net Income, Consolidated EBITDA and Consolidated Adjusted EBITDA discussion (see "Consolidated Results of Operations") excludes all of the amounts included above, as described below.

The income and expenses related to special items included in our consolidated results of operations were as follows:

(dollars in millions)

Years Ended December 31,	2019	2018
Within Total Operating Expenses	**$ 129**	$ 7,752
Within Equity in losses of unconsolidated businesses	**50**	207
Within Other income (expense), net	**3,730**	(1,382)
Total	**$ 3,909**	$ 6,577

Severance, Pension and Benefits Charges (Credits)

During 2019, in accordance with our accounting policy to recognize actuarial gains and losses in the period in which they occur, we recorded net pre-tax pension and benefits charges of $126 million in our pension and postretirement benefit plans. The charges were recorded in Other income (expense), net in our consolidated statements of income and were primarily driven by a decrease in our discount rate assumption used to determine the current year liabilities of our pension plans and postretirement benefit plans from a weighted-average of 4.4% at December 31, 2018 to a weighted-average of 3.3% at December 31, 2019 ($4.3 billion), partially offset by the difference between our estimated return on assets and our actual return on assets ($2.3 billion) and other assumption adjustments of $1.9 billion, of which $1.6 billion related to healthcare claims experience. During 2019, we also recorded net pre-tax severance charges of $204 million in Selling, general and administrative expense in our consolidated statements of income.

During 2018, we recorded net pre-tax pension and benefits credits of $2.1 billion in accordance with our accounting policy to recognize actuarial gains and losses in the period in which they occur. The pension and benefits remeasurement credits of $2.3 billion, which were recorded in Other income (expense), net in our consolidated statements of income, were primarily driven by an increase in our discount rate assumption used to determine the current year liabilities of our pension plans and postretirement benefit plans from a weighted-average of 3.7% at December 31, 2017 to a weighted-average of 4.4% at December 31, 2018 ($2.6 billion), and mortality and other assumption adjustments of $1.7 billion, $1.6 billion of which related to healthcare claims and trend adjustments, offset by the difference between our estimated return on assets of 7.0% and our actual return on assets of (2.7)% ($1.9 billion). The credits were partially offset by $177 million due to the effects of participants retiring under the Voluntary Separation Program. During 2018, we also recorded net pre-tax severance charges of $2.2 billion in Selling, general and administrative expense, primarily driven by the Voluntary Separation Program for select U.S.-based management employees and other headcount reduction initiatives, which resulted in a severance charge of $1.8 billion ($1.4 billion after-tax), and $339 million in severance costs recorded under other existing separation plans.

Due to the presentation of the other components of net periodic benefit cost, we recognize a portion of the pension and benefits charges (credits) in Other income (expense), net in our consolidated statements of income.

See Note 11 to the consolidated financial statements for additional information related to severance, pension and benefits charges (credits).

Acquisition and Integration Related Charges

Acquisition and integration related charges of $553 million recorded during the year ended December 31, 2018 primarily related to the acquisition of Yahoo's operating business in June 2017.

Product Realignment Charges

Product realignment charges of $658 million recorded during the year ended December 31, 2018 primarily related to the discontinuation of the go90 platform and associated content during the second quarter of 2018.

Impairment Charges

The impairment charges consist of write-downs of goodwill and other investments or assets. The goodwill impairment charges of $186 million and $4.6 billion recorded during the years ended December 31, 2019 and 2018, respectively, for Verizon Media were a result of the Company's annual impairment test performed in the fourth quarter (see "Critical Accounting Estimates"). In addition, we recorded an impairment charge of $50 million in Equity in losses of unconsolidated businesses related to a media joint venture investment.

Early Debt Redemption Costs

During 2019 and 2018, we recorded early debt redemptions costs of $3.6 billion and $725 million, respectively.

We recognize early debt redemptions costs in Other income (expense), net in our consolidated statements of income. See Note 7 to the consolidated financial statements for additional information related to our early debt redemptions.

Net Gain from Dispositions of Assets and Businesses

During 2019, we recorded a pre-tax net gain from dispositions of assets and businesses of $261 million in connection with the sale of various real estate properties and businesses.

Operating Environment and Trends

The telecommunications industry is highly competitive. We expect competition to remain intense as traditional and non-traditional participants seek increased market share. Our high-quality customer base and networks differentiate us from our competitors and give us the ability to plan and manage through changing economic and competitive conditions. We remain focused on executing on the fundamentals of the business: maintaining a high-quality customer base, delivering strong financial and operating results and strengthening our balance sheet. We will continue to invest for growth, which we believe is the key to creating value for our shareholders. We continue to lead in 4G LTE performance while building momentum for our 5G network. Our strategy lays the foundation for the future through investments in our Intelligent Edge Network that enable efficiencies throughout our core infrastructure and deliver flexibility to meet customer requirements.

The U.S. wireless market has achieved a high penetration of smartphones, which reduces the opportunity for new phone connection growth for the industry. We expect future revenue growth in the industry to be driven by expanding existing customer relationships, increasing the number of ways customers can connect with wireless networks and services and increasing the penetration of other connected devices including wearables, tablets and IoT devices. We expect 5G technology will provide a significant opportunity for growth in the industry in 2021 and beyond. With respect to our wireless connectivity products and services, we compete against other national wireless service providers, including AT&T Inc., Sprint Corporation and T-Mobile USA, Inc., as well as various regional wireless service providers. We also compete for retail activations with resellers that buy bulk wholesale service from wireless service providers, including Verizon, and resell it to their customers. Resellers may include cable companies. We face competition from other communications and technology companies seeking to increase their brand recognition and capture customer revenue with respect to the provision of wireless products and services, in addition to non-traditional offerings in mobile data. For example, Microsoft Corporation, Alphabet Inc., Apple Inc. and others are offering alternative means for making wireless voice calls that, in certain cases, can be used in lieu of the wireless provider's voice service, as well as alternative means of accessing video content.

With respect to wireless services and equipment, pricing plays an important role in the wireless competitive landscape. We compete in this area by offering our customers services and devices that we believe they will regard as the best available value for the price. As the demand for wireless services continues to grow, wireless service providers are offering a range of service plans at competitive prices. These service offerings will vary from time to time based on customer needs, technology changes and market conditions and may be provided as standard plans or as part of limited time promotional offers.

We expect future service revenue growth opportunities to arise from increased access revenue as customers shift to higher access plans, as well as from increased connections per account. Future service revenue growth opportunities will be dependent on expanding the penetration of our services, increasing the number of ways that our customers can connect with our networks and services and the development of new ecosystems.We and other wireless service providers, as well as equipment manufacturers, offer device payment options, which provide customers with the ability to pay for their device over a period of time, and some providers offer device leasing arrangements.

Current and potential competitors in the wireline service market include cable companies, wireless service providers, domestic and foreign telecommunications providers, satellite television companies, Internet service providers, over-the-top providers and other companies that offer network services and managed enterprise solutions.

In addition, companies with a global presence are increasingly competing with us in our wireline services. A relatively small number of telecommunications and integrated service providers with global operations serve customers in the global enterprise market and, to a lesser extent, the global wholesale market. We compete with these providers for large contracts to provide integrated solutions to global enterprises. Many of these companies have strong market presence, brand recognition and existing customer relationships, all of which contribute to intensifying competition that may affect our future revenue growth.

Despite this challenging environment, we expect that we will be able to grow key aspects of our wireline services. We continue to provide network reliability and offer products, which include fiber-optic Internet access, several video services, and voice services. Further, we will continue to offer our business and government customers more robust IP products and services, and advance our IoT strategies by leveraging business models that monetize usage on our networks at the connectivity, platform and solution layers.

The online advertising market continues to evolve as online users are migrating from traditional desktop to mobile and multiple-device usage. Also, there is a continued shift towards programmatic advertising which presents opportunities to connect online advertisers with the appropriate online users in a rapid environment. Our Media business competes with other online search engines, advertising platforms, digital video services and social networks. We are experiencing pressure from search and desktop usage and believe the pressure in these sectors will continue. We are implementing initiatives to realize synergies across all of our media assets and build services around our core content pillars to diversify revenue and return to growth.

We will also continue to focus on cost efficiencies to ensure we have the maximum flexibility to adjust to changes in the competitive and economic environments and maximize returns to shareholders.

2020 Connection Trends

In our Consumer segment, we expect to continue to attract new customers and maintain high-quality retail postpaid customers, capitalizing on demand for data services and providing our customers new ways of using wireless services in their daily lives. We expect that future connection growth will be driven by smartphones, tablets and other connected devices such as wearables. We believe the combination of our wireless network performance and Mix & Match unlimited plans provides a superior customer experience, supporting increased penetration of data services and the continued attraction and retention of higher valued retail postpaid connections. We expect to manage churn by providing a consistent, reliable experience on our wireless service and focusing on improving the customer experience through simplified pricing and continued focus in our distribution channels. We expect to continue to grow our Fios Internet connections as we seek to increase our penetration rates within our Fios service areas, further supported by the demand for higher speed internet connections. In Fios video, the business continues to face ongoing pressure as observed throughout the linear television market. We expect to manage market pressure by offering customers a choice of video service, including options such as Mix & Match on Fios and other offerings. We have experienced continuing access line and DSL losses as customers have disconnected both primary and secondary lines and switched to alternative technologies such as wireless, VoIP and cable for voice and data services.

In our Business segment, we offer wireless products and services to business and government customers across the U.S. We continue to grow our retail connections while facing a competitive environment. We expect to maintain connection growth in part by adding capacity and density

to our 4G LTE network, in addition to leading the build-out of 5G technology. We expect this connection growth, combined with our industry-leading network assets, will provide additional opportunities to sell solutions, such as those around security, advanced communications and professional services. We expect to expand our existing services offered to business customers through our Intelligent Edge Network, our multi-use platform.

2020 Operating Revenue Trends

In our Consumer segment, we expect to see a continuation of the service revenue trends from 2019 as customers shift to higher access plans with additional services and increase the number of devices they connect with our networks and services. Equipment revenues are largely dependent on wireless device sales volumes, the mix of devices, promotions and upgrades, which are subject to device lifecycles, iconic device launches and competition within the wireless industry. We anticipate an increase in wireless device upgrades in the second half of the year as we expand the availability and reach of our 5G network.

In our Business segment, we expect overall revenue growth in 2020 as wireless services and our high-quality fiber-based products will offset secular declines from legacy technologies and pressure from competition. We expect wireless revenue to expand, driven by connection growth led by Small and Medium Business. We expect our Fios products, through increased penetration, will also contribute to revenue growth. Legacy traditional wireline services continue to face secular pressures.

Our Media Business, Verizon Media, is primarily made up of digital advertising products. We are experiencing revenue pressure from search and desktop usage, which started to improve throughout 2019 and believe the pressure in those sectors will continue. We are focused on returning to revenue growth by implementing initiatives to realize synergies across all of our media assets and building services around our core content pillars. We expect positive growth in mobile services and products.

2020 Operating Expense and Cash Flow from Operations Trends

We expect our consolidated operating income margin and adjusted consolidated EBITDA margin to remain strong as we continue to undertake initiatives to reduce our overall cost structure by improving productivity and gaining efficiencies in our operations throughout the business in 2020 and beyond. Business Excellence initiatives include the adoption of the zero-based budgeting methodology, driving capital efficiencies from the architecture of the networks, evolving our Information Technology strategy and the continuing benefit from the Voluntary Separation Program. We believe our additional investments in our Business segment in both product simplification and continued focus on process improvements and new work tools will drive cost savings and create incremental growth opportunities in areas such as 5G and One Fiber. The goal of the Business Excellence initiative is to take $10 billion of cumulative cash outflows out of the business over four years, beginning with 2018. As part of this initiative, we are focusing on both operating expenses and capital expenditures. Our Business Excellence initiatives produced cumulative cash savings of $5.7 billion through the end of 2019 from a mix of capital and operational expenditure activities. The program remains on track to achieve our goal. Expenses related to programs funded through the reinvestment of program savings are expected to apply offsetting pressures to our margins.

The implementation of Topic 606 resulted in the deferral of commission expense in both our Consumer and Business segments. In 2020, we expect a smaller year-over-year benefit from the adoption of the standard due to the deferral of commission costs as compared to 2018 and 2019. The reduction in benefit creates a year-over-year headwind to operating income.

We create value for our shareholders by investing the cash flows generated by our business in opportunities and transactions that support continued profitable growth, thereby increasing customer satisfaction and usage of our products and services. In addition, we have used our cash flows to maintain and grow our dividend payout to shareholders. Verizon's Board of Directors increased the Company's quarterly dividend by 2.1% during 2019, making this the thirteenth consecutive year in which we have raised our dividend.

Our goal is to use our cash to create long-term value for our shareholders. We will continue to look for investment opportunities that will help us to grow the business, strengthen our balance sheet, acquire spectrum licenses (see "Cash Flows from Investing Activities"), pay dividends to our shareholders and, when appropriate, buy back shares of our outstanding common stock (see "Cash Flows from Financing Activities").

Capital Expenditures

Our 2020 capital program includes capital to fund advanced networks and services, including expanding our core networks, adding capacity and density to our 4G LTE network in order to stay ahead of our customers' increasing data demands and deploying our 5G network, transforming our structure to deploy the Intelligent Edge Network while reducing the cost to deliver services to our customers, and pursuing other opportunities to drive operating efficiencies. We expect that the new network architecture will simplify operations by eliminating legacy network elements, improve our 4G LTE coverage, speed the deployment of 5G technology, and create new enterprise opportunities in the business market. The level and the timing of the Company's capital expenditures within these broad categories can vary significantly as a result of a variety of factors outside of our control, such as material weather events, equipment availability from vendors and permits from local governments. Capital expenditures for 2020 are expected to be in the range of $17.0 billion to $18.0 billion, including the continued investment in our 5G network. Capital expenditures were $17.9 billion in 2019 and $16.7 billion in 2018. We believe that we have significant discretion over the amount and timing of our capital expenditures on a Company-wide basis as we are not subject to any agreement that would require significant capital expenditures on a designated schedule or upon the occurrence of designated events.

Consolidated Financial Condition

(dollars in millions)

Years Ended December 31,		2019		2018
Cash flows provided by (used in)				
Operating activities	**$**	**35,746**	$	34,339
Investing activities		**(17,581)**		(17,934)
Financing activities		**(18,164)**		(15,377)
Increase in cash, cash equivalents and restricted cash	**$**	**1**	$	1,028

We use the net cash generated from our operations to fund expansion and modernization of our networks, service and repay external financing, pay dividends, invest in new businesses and spectrum and, when appropriate, buy back shares of our outstanding common stock. Our sources of funds, primarily from operations and, to the extent necessary, from external financing arrangements, are sufficient to meet ongoing operating and investing requirements. We expect that our capital spending requirements will continue to be financed primarily through internally generated funds. Debt or equity financing may be needed to fund additional investments or development activities or to maintain an appropriate capital structure to ensure our financial flexibility. Our cash and cash equivalents are held both domestically and internationally, and are invested to maintain principal and provide liquidity. See "Market Risk" for additional information regarding our foreign currency risk management strategies.

Our available external financing arrangements include an active commercial paper program, credit available under credit facilities and other bank lines of credit, vendor financing arrangements, issuances of registered debt or equity securities, U.S. retail medium-term notes and other capital market securities that are privately-placed or offered overseas. In addition, we monetize our device payment plan agreement receivables through asset-backed debt transactions.

Cash Flows Provided By Operating Activities

Our primary source of funds continues to be cash generated from operations. Net cash provided by operating activities increased by $1.4 billion during 2019, compared to the similar period in 2018, primarily due to an increase in earnings and a decrease in discretionary contributions to qualified employee benefit plans, offset by changes in working capital, which includes an increase in cash income taxes as well as severance payments as a result of the Voluntary Separation Program. We made $300 million and $1.7 billion in discretionary employee benefits contributions to our defined benefit pension plan during 2019 and 2018, respectively. As a result of the discretionary pension contributions, we expect that there will be no required pension funding until 2026, which will continue to benefit future cash flows. These contributions also improved the funded status of our qualified pension plan.

Cash Flows Used In Investing Activities

Capital Expenditures

Capital expenditures continue to relate primarily to the use of capital resources to facilitate the introduction of new products and services, enhance responsiveness to competitive challenges, maintain our existing infrastructure and increase the operating efficiency and productivity of our networks.

Capital expenditures, including capitalized software, were as follows:

(dollars in millions)

Years Ended December 31,		2019		2018
Capital expenditures (including capitalized software)	**$**	**17,939**	$	16,658
Total as a percentage of revenue		**13.6%**		12.7%

Capital expenditures increased in 2019 primarily due to an increase in investments to support multi-use fiber assets, which support the densification of our 4G LTE network and our 5G technology deployment. Our investments are primarily related to network infrastructure to support the business.

Acquisitions

During 2019 and 2018, we invested $898 million and $1.4 billion, respectively, in acquisitions of wireless licenses. During 2019 and 2018, we also invested an insignificant amount and $230 million, respectively, in acquisitions of businesses, net of cash acquired.

In 2019, the FCC completed two millimeter wave spectrum license auctions. We paid approximately $521 million for spectrum licenses in connection with these auctions. See Note 3 to the consolidated financial statements for additional information.

In January 2018, Verizon acquired NextLink Wireless LLC (NextLink) from a wholly-owned subsidiary of XO Holdings for approximately $493 million, subject to certain adjustments, of which $320 million (an option exercise price to acquire NextLink) was prepaid in the first quarter of 2017. The option exercise price represented the fair value of the option. The remaining cash consideration was paid at the closing of the transaction. The spectrum acquired as part of the transaction is being used for our 5G technology deployment.

In February 2018, Verizon acquired Straight Path Communications Inc. (Straight Path), a holder of millimeter wave spectrum configured for 5G wireless services for total consideration reflecting an enterprise value of approximately $3.1 billion, which was primarily settled with Verizon shares but also included transaction costs payable in cash of approximately $736 million, consisting primarily of a fee paid to the FCC. The spectrum acquired as part of the transaction is being used for our 5G technology deployment.

During 2019 and 2018, we completed various other acquisitions for an insignificant amount of cash consideration.

See "Acquisitions and Divestitures" for information on our acquisitions.

Dispositions

During 2019, we received gross proceeds of approximately $1.0 billion for a sale-leaseback transaction for buildings and real estate. See Note 6 to the consolidated financial statements for additional information.

Cash Flows Used In Financing Activities

We seek to maintain a mix of fixed and variable rate debt to lower borrowing costs within reasonable risk parameters and to protect against earnings and cash flow volatility resulting from changes in market conditions. During 2019 and 2018, net cash used in financing activities was $18.2 billion and $15.4 billion, respectively.

2019

During 2019, our net cash used in financing activities of $18.2 billion was primarily driven by:

- $23.9 billion used for repayments, redemptions and repurchases of long-term borrowings and finance lease obligations, which included $6.3 billion used for prepayments and repayments of asset-backed long-term borrowings;
- $10.0 billion used for dividend payments; and
- $1.8 billion used for net debt related costs.

These uses of cash were partially offset by proceeds from long-term borrowings of $18.7 billion, which included $8.6 billion of proceeds from our asset-backed debt transactions.

Proceeds from and Repayments, Redemptions, and Repurchases of Long-Term Borrowings

At December 31, 2019, our total debt decreased to $111.5 billion as compared to $113.1 billion at December 31, 2018. During both the years ended December 31, 2019 and 2018, our effective interest rate was 4.8%. The substantial majority of our total debt portfolio consists of fixed rate indebtedness, therefore, changes in interest rates do not have a material effect on our interest payments. See also "Market Risk" and Note 7 to the consolidated financial statements for additional information.

At December 31, 2019, approximately $23.5 billion, or 21.1%, of the aggregate principal amount of our total debt portfolio consisted of foreign denominated debt, primarily the Euro and British Pound Sterling. We have entered into cross currency swaps on substantially all of our foreign denominated debt in order to fix our future interest and principal payments in U.S. dollars and mitigate the impact of foreign currency transaction gains or losses. See "Market Risk" for additional information.

Verizon may continue to repurchase debt securities issued by Verizon and its affiliates in the future through open market purchases, privately negotiated transactions, tender offers, exchange offers, or otherwise, upon such terms and at such prices as Verizon may from time to time determine for cash or other consideration.

Other, net

Other, net financing activities during 2019 includes early redemption costs, see "Special Items" for additional information, as well as cash paid on debt exchanges and derivative-related transactions. See Note 15 to the consolidated financial statements for additional information.

Dividends

The Verizon Board of Directors assesses the level of our dividend payments on a periodic basis taking into account such factors as long-term growth opportunities, internal cash requirements and the expectations of our shareholders. During the third quarter of 2019, the Board increased our quarterly dividend payment by 2.1% to $0.6150 from $0.6025 per share from the previous quarter. This is the thirteenth consecutive year that Verizon's Board of Directors has approved a quarterly dividend increase.

As in prior periods, dividend payments were a significant use of capital resources. During 2019, we paid $10.0 billion in dividends.

2018

During 2018, our net cash used in financing activities of $15.4 billion was primarily driven by:

- $14.6 billion used for repayments, redemptions and repurchases of long-term borrowings and finance lease obligations, which included $3.6 billion used for prepayments of asset-backed long-term borrowings; and
- $9.8 billion used for dividend payments.

These uses of cash were partially offset by proceeds from long-term borrowings of $10.8 billion, which included $4.8 billion of proceeds from our asset-backed debt transactions.

Proceeds from and Repayments, Redemptions, and Repurchases of Long-Term Borrowings

At December 31, 2018, our total debt was $113.1 billion, and during the year ended December 31, 2018, our effective interest rate was 4.8%. The substantial majority of our total debt portfolio consisted of fixed rate indebtedness, therefore, changes in interest rates did not have a material effect on our interest payments. See "Market Risk" and Note 7 to the consolidated financial statements for additional information.

At December 31, 2018, approximately $17.1 billion, or 15.1%, of the aggregate principal amount of our total debt portfolio consisted of foreign denominated debt, primarily the Euro and British Pound Sterling. We have entered into cross currency swaps on a majority of our foreign denominated debt in order to fix our future interest and principal payments in U.S. dollars and mitigate the impact of foreign currency transaction gains or losses. See "Market Risk" for additional information.

Other, net

Other, net financing activities during 2018, included early debt redemption costs. See "Special Items" for additional information, as well as cash paid on debt exchanges and derivative-related transactions.

Dividends

During the third quarter of 2018, the Board increased our quarterly dividend payment by 2.1% to $0.6025 per share.

As in prior periods, dividend payments were a significant use of capital resources. During 2018, we paid $9.8 billion in dividends.

Asset-Backed Debt

As of December 31, 2019, the carrying value of our asset-backed debt was $12.4 billion. Our asset-backed debt includes Asset-Backed Notes (ABS Notes) issued to third-party investors (Investors) and loans (ABS Financing Facilities) received from banks and their conduit facilities (collectively, the Banks). Our consolidated asset-backed debt bankruptcy remote legal entities (each, an ABS Entity or collectively, the ABS Entities) issue the debt or are otherwise party to the transaction documentation in connection with our asset-backed debt transactions. Under the terms of our asset-backed debt, Cellco Partnership (Cellco) and certain other affiliates of Verizon (collectively, the Originators) transfer device payment plan agreement receivables to one of the ABS Entities, which in turn transfers such receivables to another ABS Entity that issues the debt. Verizon entities retain the equity interests in the ABS Entities, which represent the rights to all funds not needed to make required payments on the asset-backed debt and other related payments and expenses.

Our asset-backed debt is secured by the transferred device payment plan agreement receivables and future collections on such receivables. The device payment plan agreement receivables transferred to the ABS Entities and related assets, consisting primarily of restricted cash, will only be available for payment of asset-backed debt and expenses related thereto, payments to the Originators in respect of additional transfers of device payment plan agreement receivables, and other obligations arising from our asset-backed debt transactions, and will not be available to pay other obligations or claims of Verizon's creditors until the associated asset-backed debt and other obligations are satisfied. The Investors or Banks, as applicable, which hold our asset-backed debt have legal recourse to the assets securing the debt, but do not have any recourse to Verizon with respect to the payment of principal and interest on the debt. Under a parent support agreement, Verizon has agreed to guarantee certain of the payment obligations of Cellco and the Originators to the ABS Entities.

Cash collections on the device payment plan agreement receivables collateralizing our asset-backed debt securities are required at certain specified times to be placed into segregated accounts. Deposits to the segregated accounts are considered restricted cash and are included in Prepaid expenses and other, and Other assets in our consolidated balance sheets.

Proceeds from our asset-backed debt transactions are reflected in Cash flows from financing activities in our consolidated statements of cash flows. The asset-backed debt issued and the assets securing this debt are included in our consolidated balance sheets. See Note 7 to the consolidated financial statements for additional information.

In May 2018, we entered into an ABS financing facility with a number of financial institutions (2018 ABS Financing Facility). One loan agreement was entered into in connection with the 2018 ABS Financing Facility. In May 2019, the $540 million outstanding under the loan agreement was prepaid, and the loan agreement was terminated.

In September 2016, we entered into an ABS financing facility with a number of financial institutions (2016 ABS Financing Facility). Two loan agreements were entered into in connection with the 2016 ABS Financing Facility in September 2016 and May 2017. In April and May 2019, we paid off both the 2016 and 2017 loans for an aggregate of $671 million, and the loan agreements were terminated.

In May 2019, the 2016 ABS Financing Facility was amended and restated (2019 ABS Financing Facility). One loan agreement was entered into in connection with the 2019 ABS Financing Facility. Under the 2019 loan agreement, we have the right to prepay all or a portion of the advances at any time without penalty, but in certain cases, with breakage costs. During 2019, we received $4.8 billion of borrowings and prepaid $1.5 billion under the 2019 loan agreement.

Long-Term Credit Facilities

		At December 31, 2019		
(dollars in millions)	Maturities	Facility Capacity	Unused Capacity	Principal Amount Outstanding
Verizon revolving credit facility [1]	2022	$ 9,500	$ 9,390	N/A
Various export credit facilities [2]	2022-2027	5,500	—	4,471
Total		$ 15,000	$ 9,390	$ 4,471

[1] The revolving credit facility does not require us to comply with financial covenants or maintain specified credit ratings, and it permits us to borrow even if our business has incurred a material adverse change. The revolving credit facility provides for the issuance of letters of credit.
[2] During 2019 and 2018, we drew down $1.5 billion and $3.0 billion from these facilities, respectively. We use these credit facilities to finance equipment-related purchases.

Common Stock

Common stock has been used from time to time to satisfy some of the funding requirements of employee and shareholder plans. During the years ended December 31, 2019 and 2018, we issued 3.8 million and 3.5 million common shares from Treasury stock, respectively, which had an insignificant aggregate value.

In February 2020, the Verizon Board of Directors authorized a share buyback program to repurchase up to 100 million shares of the Company's common stock. The program will terminate when the aggregate number of shares purchased reaches 100 million, or a new share repurchase plan superseding the current plan is authorized, whichever is sooner. The program permits Verizon to repurchase shares over time, with the amount and timing of repurchases depending on market conditions and corporate needs. There were no repurchases of common stock during 2019 or 2018 under our previously authorized share buyback program.

Credit Ratings

Verizon's credit ratings did not change in 2019 or 2018.

Securities ratings assigned by rating organizations are expressions of opinion and are not recommendations to buy, sell or hold securities. A securities rating is subject to revision or withdrawal at any time by the assigning rating organization. Each rating should be evaluated independently of any other rating.

Covenants

Our credit agreements contain covenants that are typical for large, investment grade companies. These covenants include requirements to pay interest and principal in a timely fashion, pay taxes, maintain insurance with responsible and reputable insurance companies, preserve our corporate existence, keep appropriate books and records of financial transactions, maintain our properties, provide financial and other reports to our lenders, limit pledging and disposition of assets and mergers and consolidations, and other similar covenants.

We and our consolidated subsidiaries are in compliance with all of our restrictive covenants in our debt agreements.

Change In Cash, Cash Equivalents and Restricted Cash

Our Cash and cash equivalents at December 31, 2019 totaled $2.6 billion, a $151 million decrease compared to Cash and cash equivalents at December 31, 2018, primarily as a result of the factors discussed above.

Restricted cash at December 31, 2019 totaled $1.3 billion, a $152 million increase compared to restricted cash at December 31, 2018, primarily due to cash collections on the device payment plan agreement receivables that are required at certain specified times to be placed into segregated accounts.

Free Cash Flow

Free cash flow is a non-GAAP financial measure that reflects an additional way of viewing our liquidity that, when viewed with our GAAP results, provides a more complete understanding of factors and trends affecting our cash flows. Free cash flow is calculated by subtracting capital expenditures from net cash provided by operating activities. We believe it is a more conservative measure of cash flow since purchases of fixed assets are necessary for ongoing operations. Free cash flow has limitations due to the fact that it does not represent the residual cash flow available for discretionary expenditures. For example, free cash flow does not incorporate payments made on finance lease obligations or cash payments for business acquisitions or wireless licenses. Therefore, we believe it is important to view free cash flow as a complement to our entire consolidated statements of cash flows.

The following table reconciles net cash provided by operating activities to Free cash flow:

				(dollars in millions)
Years Ended December 31,		**2019**		2018
Net cash provided by operating activities	**$**	**35,746**	$	34,339
Less Capital expenditures (including capitalized software)		**17,939**		16,658
Free cash flow	**$**	**17,807**	$	17,681

The increase in free cash flow during 2019 is a reflection of the increase in operating cash flows, partially offset by the increase in capital expenditures discussed above.

Employee Benefit Plans Funded Status and Contributions

Employer Contributions

We operate numerous qualified and nonqualified pension plans and other postretirement benefit plans. These plans primarily relate to our domestic business units. During 2019 and 2018, contributions to our qualified pension plans were $300 million and $1.0 billion, respectively. We made contributions of $71 million in 2019 to our nonqualified pension plans.

The Company's overall investment strategy is to achieve a mix of assets that allows us to meet projected benefit payments while taking into consideration risk and return. In an effort to reduce the risk of our portfolio strategy and better align assets with liabilities, we have adopted a liability driven pension strategy that seeks to better match cash flows from investments with projected benefit payments. We expect that the strategy will reduce the likelihood that assets will decline at a time when liabilities increase (referred to as liability hedging), with the goal to reduce the risk of underfunding to the plan and its participants and beneficiaries; however, we also expect the strategy to result in lower asset returns. Nonqualified pension contributions are estimated to be approximately $70 million in 2020.

Contributions to our other postretirement benefit plans generally relate to payments for benefits on an as-incurred basis since these other postretirement benefit plans do not have funding requirements similar to the pension plans. We contributed $449 million to our other postretirement benefit plans in 2019 and $1.2 billion, including $679 million discretionary contributions, in 2018. Contributions to our other postretirement benefit plans are estimated to be approximately $700 million in 2020.

Leasing Arrangements

See Note 6 to the consolidated financial statements for a discussion of leasing arrangements.

Contractual Obligations

The following table provides a summary of our contractual obligations and commercial commitments at December 31, 2019. Additional detail about these items is included in the notes to the consolidated financial statements.

(dollars in millions)

	Payments Due By Period				
Contractual Obligations	Total	Less than 1 year	1 to 3 years	3 to 5 years	More than 5 years
Long-term debt[1]	$ 110,865	$ 10,470	$ 16,431	$ 9,803	$ 74,161
Finance lease obligations[2]	1,213	366	479	244	124
Total long-term debt, including current maturities	112,078	10,836	16,910	10,047	74,285
Interest on long-term debt[1]	62,450	4,578	8,383	7,426	42,063
Operating leases[2]	25,968	4,099	7,127	5,485	9,257
Purchase obligations[3]	18,769	8,384	7,448	1,441	1,496
Other long-term liabilities[4]	4,135	694	1,692	1,749	—
Finance obligations[5]	1,539	281	579	603	76
Total contractual obligations	$ 224,939	$ 28,872	$ 42,139	$ 26,751	$ 127,177

[1] Items included in long-term debt with variable coupon rates exclude unamortized debt issuance costs, and are described in Note 7 to the consolidated financial statements.

[2] See Note 6 to the consolidated financial statements for additional information.

[3] Items included in purchase obligations are primarily commitments to purchase content and network services, equipment, software and marketing services, which will be used or sold in the ordinary course of business. These amounts do not represent our entire anticipated purchases in the future, but represent only those items that are the subject of contractual obligations. We also purchase products and services as needed with no firm commitment. For this reason, the amounts presented in this table alone do not provide a reliable indicator of our expected future cash outflows or changes in our expected cash position. See Note 16 to the consolidated financial statements for additional information.

[4] Other long-term liabilities represent estimated postretirement benefit and qualified pension plan contributions. Estimated qualified pension plan contributions include expected minimum funding contributions, which commence in 2026 based on the plan's current funded status. Estimated postretirement benefit payments include expected future postretirement benefit payments. These estimated amounts: (1) are subject to change based on changes to assumptions and future plan performance, which could impact the timing or amounts of these payments; and (2) exclude expectations beyond 5 years due to uncertainty of the timing and amounts. See Note 11 to the consolidated financial statements for additional information.

[5] Represents future minimum payments under the sublease arrangement for our tower transaction. See Note 6 to the consolidated financial statements for additional information.

We are not able to make a reasonable estimate of when the unrecognized tax benefits balance of $2.9 billion and related interest and penalties will be settled with the respective taxing authorities until issues or examinations are further developed. See Note 12 to the consolidated financial statements for additional information.

Guarantees

We guarantee the debentures of our operating telephone company subsidiaries as well as the debt obligations of GTE LLC, as successor in interest to GTE Corporation, that were issued and outstanding prior to July 1, 2003. See Note 7 to the consolidated financial statements for additional information.

In connection with the execution of agreements for the sale of businesses and investments, Verizon ordinarily provides representations and warranties to the purchasers pertaining to a variety of nonfinancial matters, such as ownership of the securities being sold, as well as financial losses. See Note 16 to the consolidated financial statements for additional information.

As of December 31, 2019, letters of credit totaling approximately $632 million, which were executed in the normal course of business and support several financing arrangements and payment obligations to third parties, were outstanding. See Note 16 to the consolidated financial statements for additional information.

Market Risk

We are exposed to various types of market risk in the normal course of business, including the impact of interest rate changes, foreign currency exchange rate fluctuations, changes in investment, equity and commodity prices and changes in corporate tax rates. We employ risk management strategies, which may include the use of a variety of derivatives including cross currency swaps, forward starting interest rate swaps, interest rate swaps, interest rate caps and foreign exchange forwards. We do not hold derivatives for trading purposes.

It is our general policy to enter into interest rate, foreign currency and other derivative transactions only to the extent necessary to achieve our desired objectives in optimizing exposure to various market risks. Our objectives include maintaining a mix of fixed and variable rate debt to lower borrowing costs within reasonable risk parameters and to protect against earnings and cash flow volatility resulting from changes in market conditions. We do not hedge our market risk exposure in a manner that would completely eliminate the effect of changes in interest rates and foreign exchange rates on our earnings.

Counterparties to our derivative contracts are major financial institutions with whom we have negotiated derivatives agreements (ISDA master agreements) and credit support annex (CSA) agreements which provide rules for collateral exchange. Negotiations and executions of new ISDA master agreements and CSA agreements with our counterparties continued during 2018. The CSA agreements contain rating based thresholds such that we or our counterparties may be required to hold or post collateral based upon changes in outstanding positions as compared to established thresholds and changes in credit ratings. At December 31, 2019, we held an insignificant amount and at December 31, 2018, we posted approximately $0.1 billion of collateral related to derivative contracts under collateral exchange arrangements, which were recorded as Other current liabilities and Prepaid expenses and other, respectively, in our consolidated balance sheets. While we may be exposed to credit losses due to the nonperformance of our counterparties, we consider the risk remote and do not expect that any such nonperformance would result in a significant effect on our results of operations or financial condition due to our diversified pool of counterparties. See Note 9 to the consolidated financial statements for additional information regarding the derivative portfolio.

Interest Rate Risk

We are exposed to changes in interest rates, primarily on our short-term debt and the portion of long-term debt that carries floating interest rates. As of December 31, 2019, approximately 79% of the aggregate principal amount of our total debt portfolio consisted of fixed rate indebtedness, including the effect of interest rate swap agreements designated as hedges. The impact of a 100-basis-point change in interest rates affecting our floating rate debt would result in a change in annual interest expense, including our interest rate swap agreements that are designated as hedges, of approximately $248 million. The interest rates on our existing long-term debt obligations are unaffected by changes to our credit ratings.

Certain of our floating rate debt and our interest rate derivative transactions utilize interest rates that are linked to the London Inter-Bank Offered Rate (LIBOR) as the benchmark rate. LIBOR is the subject of recent U.S. and international regulatory guidance and proposals for reform. These reforms and other pressures may cause LIBOR to become unavailable or to perform or be reported differently than in the past. The consequences of these developments cannot be entirely predicted but could include an increase in the cost of our floating rate debt or exposure under our interest rate derivative transactions. We do not anticipate a significant impact to our financial position given our current mix of variable and fixed-rate debt, taking into account the impact of our interest rate hedging.

The table that follows summarizes the fair values of our long-term debt, including current maturities, and interest rate swap derivatives as of December 31, 2019 and 2018. The table also provides a sensitivity analysis of the estimated fair values of these financial instruments assuming 100-basis-point upward and downward shifts in the yield curve. Our sensitivity analysis does not include the fair values of our commercial paper and bank loans, if any, because they are not significantly affected by changes in market interest rates.

(dollars in millions)

Long-term debt and related derivatives	Fair Value	Fair Value assuming + 100 basis point shift	Fair Value assuming - 100 basis point shift
At December 31, 2019	**$ 128,633**	**$ 119,288**	**$ 139,980**
At December 31, 2018	119,195	111,250	128,957

Interest Rate Swaps

We enter into interest rate swaps to achieve a targeted mix of fixed and variable rate debt. We principally receive fixed rates and pay variable rates that are currently based on LIBOR, resulting in a net increase or decrease to Interest expense. These swaps are designated as fair value hedges and hedge against interest rate risk exposure of designated debt issuances. At December 31, 2019, the fair value of the asset and liability of these contracts were $568 million and $173 million, respectively. At December 31, 2018, the fair value of the asset and liability of these contracts were insignificant and $813 million, respectively. At December 31, 2019 and 2018, the total notional amount of the interest rate swaps was $17.0 billion and $19.8 billion, respectively.

Forward Starting Interest Rate Swaps

We have entered into forward starting interest rate swaps designated as cash flow hedges in order to manage our exposure to interest rate changes on future forecasted transactions. At December 31, 2019 and 2018, the fair value of the liability of these contracts was $604 million and $60 million, respectively. At December 31, 2019 and 2018, the total notional amount of the forward starting interest rate swaps was $3.0 billion and $4.0 billion, respectively.

Interest Rate Caps

We also have interest rate caps which we use as an economic hedge but for which we have elected not to apply hedge accounting. We enter into interest rate caps to mitigate our interest exposure to interest rate increases on our ABS Financing Facility and ABS Notes. The fair value of the asset and liability of these contracts was insignificant at both December 31, 2019 and 2018. At December 31, 2019 and 2018, the total notional value of these contracts was $679 million and $2.2 billion, respectively.

Foreign Currency Translation

The functional currency for our foreign operations is primarily the local currency. The translation of income statement and balance sheet amounts of our foreign operations into U.S. dollars is recorded as cumulative translation adjustments, which are included in Accumulated other comprehensive income in our consolidated balance sheets. Gains and losses on foreign currency transactions are recorded in the consolidated statements of income in Other income (expense), net. At December 31, 2019, our primary translation exposure was to the British Pound Sterling, Euro, Australian Dollar and Japanese Yen.

Cross Currency Swaps

We have entered into cross currency swaps designated as cash flow hedges to exchange our British Pound Sterling, Euro, Swiss Franc and Australian Dollar-denominated cash flows into U.S. dollars and to fix our cash payments in U.S. dollars, as well as to mitigate the impact of foreign currency transaction gains or losses. The fair value of the asset of these contracts was $211 million and $220 million at December 31, 2019 and 2018, respectively. At December 31, 2019 and 2018, the fair value of the liability of these contracts was $912 million and $536 million, respectively. At December 31, 2019 and 2018, the total notional amount of the cross currency swaps was $23.1 billion and $16.6 billion, respectively.

Foreign Exchange Forwards

We also have foreign exchange forwards which we use as an economic hedge but for which we have elected not to apply hedge accounting. We enter into British Pound Sterling and Euro foreign exchange forwards to mitigate our foreign exchange rate risk related to non-functional currency denominated monetary assets and liabilities of international subsidiaries. At December 31, 2019, the fair value of the asset of these contracts was insignificant. At December 31, 2019 and 2018, the total notional amount of the foreign exchange forwards was $1.1 billion and $600 million, respectively.

Critical Accounting Estimates and Recently Issued Accounting Standards

Critical Accounting Estimates

A summary of the critical accounting estimates used in preparing our financial statements is as follows:

Wireless Licenses and Goodwill

Wireless licenses and Goodwill are a significant component of our consolidated assets. Both our wireless licenses and goodwill are treated as indefinite-lived intangible assets and, therefore are not amortized, but rather are tested for impairment annually in the fourth fiscal quarter, unless there are events requiring an earlier assessment or changes in circumstances during an interim period providing impairment indicators are present. We believe our estimates and assumptions are reasonable and represent appropriate marketplace considerations as of the valuation date. Although we use consistent methodologies in developing the assumptions and estimates underlying the fair value calculations used in our impairment tests, these estimates and assumptions are uncertain by nature, may change over time and can vary from actual results. It is possible that in the future there may be changes in our estimates and assumptions, including the timing and amount of future cash flows, margins, growth rates, market participant assumptions, comparable benchmark companies and related multiples and discount rates, which could result in different fair value estimates. Significant and adverse changes to any one or more of the above-noted estimates and assumptions could result in a goodwill impairment for one or more of our reporting units.

Wireless Licenses

The carrying value of our wireless licenses was approximately $95.1 billion as of December 31, 2019. We aggregate our wireless licenses into one single unit of accounting, as we utilize our wireless licenses on an integrated basis as part of our nationwide wireless network. Our wireless licenses provide us with the exclusive right to utilize certain radio frequency spectrum to provide wireless communication services. There are currently no legal, regulatory, contractual, competitive, economic or other factors that limit the useful life of our wireless licenses.

In 2019, we performed a qualitative impairment assessment to determine whether it is more likely than not that the fair value of our wireless licenses was less than the carrying amount. As part of our assessment we considered several qualitative factors including the historical business enterprise value of our wireless business, macroeconomic conditions (including changes in interest rates and discount rates), industry and market considerations (including industry revenue and EBITDA margin projections), the recent and projected financial performance of our wireless business as a whole, as well as other factors.

In 2018, our quantitative impairment test consisted of comparing the estimated fair value of our aggregate wireless licenses to the aggregated carrying amount as of the test date.

Our impairment test in 2019 indicated that it is more likely than not that the fair value of our wireless licenses remained above their carrying value and, therefore, did not result in an impairment. Our impairment test in 2018 indicated that the fair value of our wireless licenses significantly exceeded their carrying value and, therefore, did not result in an impairment.

Under our quantitative assessment, we estimated the fair value of our wireless licenses using the Greenfield approach. The Greenfield approach is an income-based valuation approach that values the wireless licenses by calculating the cash flow generating potential of a hypothetical start-up company that goes into business with no assets except the wireless licenses to be valued. A discounted cash flow analysis is used to estimate what a marketplace participant would be willing to pay to purchase the aggregated wireless licenses as of the valuation date. As a result, we

were required to make significant estimates about future cash flows specifically associated with our wireless licenses, an appropriate discount rate based on the risk associated with those estimated cash flows and assumed terminal value and growth rates. We considered current and expected future economic conditions, current and expected availability of wireless network technology and infrastructure and related equipment and the costs thereof as well as other relevant factors in estimating future cash flows. The discount rate represented our estimate of the weighted-average cost of capital (WACC), or expected return, that a marketplace participant would have required as of the valuation date. We developed the discount rate based on our consideration of the cost of debt and equity of a group of guideline companies as of the valuation date. Accordingly, our discount rate incorporated our estimate of the expected return a marketplace participant would have required as of the valuation date, including the risk premium associated with the current and expected economic conditions as of the valuation date. The terminal value growth rate represented our estimate of the marketplace's long-term growth rate.

Goodwill

In November 2018, we announced a strategic reorganization of our business. The Company began reporting externally under the new structure as of April 1, 2019 which resulted in certain changes to our operating segments and reporting units. Upon the date of reorganization, the goodwill of our historical Wireless reporting unit, historical Wireline reporting unit and historical Verizon Connect reporting unit were reallocated to our new Consumer and Business reporting units using a relative fair value approach. At December 31, 2019, the balance of our goodwill was approximately $24.4 billion, of which $17.1 billion was in our Consumer reporting unit and $7.3 billion was in our Business reporting unit. To determine if goodwill is potentially impaired, we have the option to perform a qualitative assessment to determine whether it is more likely than not that the fair value of a reporting unit is less than its carrying value. If we elect not to conduct the qualitative assessment or if indications of a potential impairment exist, the determination of whether an impairment has occurred requires the determination of the fair value of each the reporting unit being assessed.

Under the qualitative assessment, we consider several qualitative factors, including the business enterprise value of the reporting unit from the last quantitative test and the excess of fair value over carrying value from this test, macroeconomic conditions (including changes in interest rates and discount rates), industry and market considerations (including industry revenue and EBITDA margin projections), the recent and projected financial performance of the reporting unit, as well as other factors.

Under our quantitative assessment, the fair value of the reporting unit is calculated using a market approach and a discounted cash flow method. The market approach includes the use of comparative multiples to corroborate discounted cash flow results. The discounted cash flow method is based on the present value of two components-projected cash flows and a terminal value. The terminal value represents the expected normalized future cash flows of the reporting unit beyond the cash flows from the discrete projection period. The fair value of the reporting unit is calculated based on the sum of the present value of the cash flows from the discrete period and the present value of the terminal value. The discount rate represented our estimate of the WACC, or expected return, that a marketplace participant would have required as of the valuation date. The application of our goodwill impairment test required key assumptions underlying our valuation model. The discounted cash flow analysis factored in assumptions on discount rates and terminal growth rates to reflect risk profiles of key strategic revenue and cost initiatives, as well as revenue and EBITDA growth relative to history and market trends and expectations. The market multiples approach incorporated significant judgment involved in the selection comparable public company multiples and benchmarks. The selection of companies was influenced by differences in growth and profitability, and volatility in market prices of peer companies. These valuation inputs are inherently uncertain, and an adverse change in one or a combination of these inputs could trigger a goodwill impairment loss in the future.

A projected sustained decline in a reporting unit's revenues and earnings could have a significant negative impact on its fair value and may result in impairment charges. Such a decline could be driven by, among other things: (1) further anticipated decreases in service pricing, sales volumes and long-term growth rate as a result of competitive pressures or other factors; or (2) the inability to achieve or delays in achieving the goals in strategic initiatives. Also, adverse changes to macroeconomic factors, such as increases to long-term interest rates, would also negatively impact the fair value of the reporting unit.

We performed impairment assessments of the impacted reporting units, specifically our historical Wireless, historical Wireline and historical Connect reporting units on March 31, 2019, immediately before our strategic reorganization became effective. Our impairment assessments indicated that the fair value for each of our historical Wireless, historical Wireline and historical Connect reporting units exceeded their respective carrying values, and therefore did not result in a goodwill impairment. We then performed quantitative assessments of our Consumer and Business reporting units on April 1, 2019, immediately following our strategic reorganization. Our impairment assessments indicated that the fair value for each of our Consumer and Business reporting units exceeded their respective carrying values and therefore, did not result in a goodwill impairment. Our Media reporting unit was not impacted by the strategic reorganization and there was no indicator of impairment as of the reorganization date.

We performed qualitative impairment assessments for our Consumer and Business reporting units during the fourth quarter of 2019. Our qualitative assessments indicated that it was more likely than not that the fair values for our Consumer and Business reporting units exceeded their respective carrying values and, therefore, did not result in an impairment. We performed quantitative impairment assessments for our Media reporting unit in 2019 and 2018. For details on our Media reporting unit, refer to the discussion below.

Our Media business, Verizon Media, experienced increased competitive and market pressures throughout 2018 that resulted in lower than expected revenues and earnings. These pressures were expected to continue and have resulted in a loss of market positioning to our competitors in the digital advertising business. Our Media business also achieved lower than expected benefits from the integration of the Yahoo Inc. and AOL Inc. (AOL) businesses.

As of August 2018, Hans Vestberg became Chief Executive Officer of Verizon, and as of October 2018, K. Guru Gowrappan was appointed Chief Executive Officer of our Media business. In connection with Verizon's annual budget process during the fourth quarter of 2019 and 2018, the leadership at both Verizon Media and Verizon completed a comprehensive five-year strategic planning review of Verizon Media's business prospects resulting in unfavorable adjustments to Verizon Media's financial projections. These revised projections were used as a key input into Verizon Media's annual goodwill impairment tests performed in the fourth quarter of 2019 and 2018.

During the fourth quarter of 2019 and 2018, consistent with our accounting policy, we applied a combination of a market approach and a discounted cash flow method reflecting current assumptions and inputs, including our revised projections, discount rate and expected growth rates, which resulted in the determination that the fair value of the Media reporting unit was less than its carrying amount. As a result, we recorded a non-cash goodwill impairment charge of approximately $186 million ($176 million after-tax) in the fourth quarter of 2019 and a charge of $4.6 billion ($4.5 billion after-tax) in the fourth quarter of 2018 in our consolidated statements of income. The goodwill balance of the Media reporting unit has been fully written off as a result of these impairment charges.

We performed a quantitative impairment assessment for all of the other reporting units in 2018. Our impairment tests indicated that the fair value for each of our historical Wireless, historical Wireline and historical Connect reporting units exceeded their respective carrying value and, therefore, did not result in an impairment.

Pension and Other Postretirement Benefit Plans

We maintain benefit plans for most of our employees, including, for certain employees, pension and other postretirement benefit plans. At December 31, 2019, in the aggregate, pension plan benefit obligations exceeded the fair value of pension plan assets, which will result in future pension plan expense. Other postretirement benefit plans have larger benefit obligations than plan assets, resulting in expense. Significant benefit plan assumptions, including the discount rate used, the long-term rate of return on plan assets, the determination of the substantive plan and health care trend rates are periodically updated and impact the amount of benefit plan income, expense, assets and obligations. Changes to one or more of these assumptions could significantly impact our accounting for pension and other postretirement benefits. A sensitivity analysis of the impact of changes in these assumptions on the benefit obligations and expense (income) recorded, as well as on the funded status due to an increase or a decrease in the actual versus expected return on plan assets as of December 31, 2019 and for the year then ended pertaining to Verizon's pension and postretirement benefit plans, is provided in the table below.

(dollars in millions)	Percentage point change	Increase/(decrease) at December 31, 2019*
Pension plans discount rate	+0.50 $	(1,137)
	-0.50	1,266
Rate of return on pension plan assets	+1.00	(167)
	-1.00	167
Postretirement plans discount rate	+0.50	(858)
	-0.50	948
Rate of return on postretirement plan assets	+1.00	(9)
	-1.00	9
Health care trend rates	+1.00	626
	-1.00	(696)

* In determining its pension and other postretirement obligation, the Company used a weighted-average discount rate of 3.3%. The rate was selected to approximate the composite interest rates available on a selection of high-quality bonds available in the market at December 31, 2019. The bonds selected had maturities that coincided with the time periods during which benefits payments are expected to occur, were non-callable and available in sufficient quantities to ensure marketability (at least $300 million par outstanding).

The annual measurement date for both our pension and other postretirement benefits is December 31. We use the full yield curve approach to estimate the interest cost component of net periodic benefit cost for pension and other postretirement benefits. The full yield curve approach refines our estimate of interest cost by applying the individual spot rates from a yield curve composed of the rates of return on several hundred high-quality fixed income corporate bonds available at the measurement date. These individual spot rates align with the timing of each future cash outflow for benefit payments and therefore provide a more precise estimate of interest cost.

Income Taxes

Our current and deferred income taxes and associated valuation allowances are impacted by events and transactions arising in the normal course of business as well as in connection with the adoption of new accounting standards, changes in tax laws and rates, acquisitions and dispositions of businesses and non-recurring items. As a global commercial enterprise, our income tax rate and the classification of income taxes can be affected by many factors, including estimates of the timing and realization of deferred income tax assets and the timing and amount of income tax payments. We account for tax benefits taken or expected to be taken in our tax returns in accordance with the accounting standard relating to the uncertainty in income taxes, which requires the use of a two-step approach for recognizing and measuring tax benefits taken or expected to be taken in a tax return. We review and adjust our liability for unrecognized tax benefits based on our best judgment given the facts, circumstances and information available at each reporting date. To the extent that the final outcome of these tax positions is different than the amounts recorded, such differences may impact income tax expense and actual tax payments. We recognize any interest and penalties accrued related to unrecognized tax benefits in income tax expense. Actual tax payments may materially differ from estimated liabilities as a result of changes in tax laws as well

as unanticipated transactions impacting related income tax balances. See Note 12 to the consolidated financial statements for additional information.

Property, Plant and Equipment

Our Property, plant and equipment balance represents a significant component of our consolidated assets. We record Property, plant and equipment at cost. We depreciate Property, plant and equipment on a straight-line basis over the estimated useful life of the assets. We expect that a one year increase in estimated useful lives of our Property, plant and equipment would result in a decrease to our 2019 depreciation expense of $2.7 billion and that a one year decrease would result in an increase of approximately $4.7 billion in our 2019 depreciation expense.

Accounts Receivable

We maintain allowances for uncollectible accounts receivable, including our direct-channel device payment plan agreement receivables, for estimated losses resulting from the failure or inability of our customers to make required payments. Indirect-channel device payment loans are considered financial instruments and are initially recorded at fair value net of imputed interest, and credit losses are recorded as incurred. However, loan balances are assessed quarterly for impairment and an allowance is recorded if the loan is considered impaired. Our allowance for uncollectible accounts receivable is based on management's assessment of the collectability of specific customer accounts and includes consideration of the credit worthiness and financial condition of those customers. We record an allowance to reduce the receivables to the amount that is reasonably believed to be collectible. We also record an allowance for all other receivables based on multiple factors including historical experience with bad debts, the general economic environment and the aging of such receivables. Similar to traditional service revenue, we record direct device payment plan agreement bad debt expense based on an estimate of the percentage of equipment revenue that will not be collected. This estimate is based on a number of factors including historical write-off experience, credit quality of the customer base and other factors such as macroeconomic conditions. If there is a deterioration of our customers' financial condition or if future actual default rates on receivables in general differ from those currently anticipated, we may have to adjust our allowance for doubtful accounts, which would affect earnings in the period the adjustments are made.

Recently Issued Accounting Standards

See Note 1 to the consolidated financial statements for a discussion of recently issued accounting standard updates not yet adopted as of December 31, 2019.

Acquisitions and Divestitures

Acquisition of AOL Inc.

In May 2015, we entered into an Agreement and Plan of Merger with AOL Inc. pursuant to which we commenced a tender offer to acquire all of the outstanding shares of common stock of AOL at a price of $50.00 per share, net to the seller in cash, without interest and less any applicable withholding taxes.

On June 23, 2015, we completed the tender offer and merger, and AOL became a wholly-owned subsidiary of Verizon. The aggregate cash consideration paid by Verizon at the closing of these transactions was approximately $3.8 billion. Holders of approximately 6.6 million shares exercised appraisal rights under Delaware law. In September 2018, we obtained court approval to settle this matter for total cash consideration of $219 million, of which an insignificant amount relates to interest, resulting in an insignificant gain. We paid the cash consideration in October 2018.

XO Holdings

In February 2016, we entered into a purchase agreement to acquire XO Holdings' wireline business (XO), which owned and operated one of the largest fiber-based IP and Ethernet networks in the U.S. Concurrently, we entered into a separate agreement to utilize certain wireless spectrum from a wholly-owned subsidiary of XO Holdings, NextLink, that held XO's millimeter-wave wireless spectrum. The agreement included an option, subject to certain conditions, to acquire NextLink. In February 2017, we completed our acquisition of XO for total cash consideration of approximately $1.5 billion, of which $100 million was paid in 2015, and we prepaid $320 million in connection with the NextLink option which represented the fair value of the option.

In April 2017, we exercised our option to buy NextLink for approximately $493 million, subject to certain adjustments, of which $320 million was prepaid in the first quarter of 2017. The transaction closed in January 2018. The acquisition of NextLink was accounted for as an asset acquisition, as substantially all of the value related to the acquired spectrum. Upon closing, we recorded approximately $657 million of wireless licenses, $110 million of a deferred tax liability and $58 million of other liabilities. See Note 3 to the consolidated financial statements for additional information.

Straight Path

In May 2017, we entered into a purchase agreement to acquire Straight Path, a holder of millimeter wave spectrum configured for 5G wireless services, for total consideration reflecting an enterprise value of approximately $3.1 billion. Under the terms of the purchase agreement, we agreed to pay: (1) Straight Path shareholders $184.00 per share, payable in Verizon shares; and (2) certain transaction costs payable in cash of approximately $736 million, consisting primarily of a fee to be paid to the FCC. The transaction closed in February 2018 at which time we issued

approximately 49 million shares of Verizon common stock, valued at approximately $2.4 billion, and paid the associated cash consideration. See Note 3 to the consolidated financial statements for additional information.

Spectrum License Transactions

From time to time, we enter into agreements to buy, sell or exchange spectrum licenses. We believe these spectrum license transactions have allowed us to continue to enhance the reliability of our wireless network while also resulting in a more efficient use of spectrum. See Note 3 to the consolidated financial statements for additional information regarding our spectrum license transactions.

Other

From time to time, we enter into strategic agreements to acquire various other businesses and investments. See Note 3 to the consolidated financial statements for additional information.

Cautionary Statement Concerning Forward-Looking Statements

In this report we have made forward-looking statements. These statements are based on our estimates and assumptions and are subject to risks and uncertainties. Forward-looking statements include the information concerning our possible or assumed future results of operations. Forward-looking statements also include those preceded or followed by the words "anticipates," "believes," "estimates," "expects," "hopes" or similar expressions. For those statements, we claim the protection of the safe harbor for forward-looking statements contained in the Private Securities Litigation Reform Act of 1995. We undertake no obligation to revise or publicly release the results of any revision to these forward-looking statements, except as required by law. Given these risks and uncertainties, readers are cautioned not to place undue reliance on such forward-looking statements.

The following important factors, along with those discussed elsewhere in this report and in other filings with the SEC, could affect future results and could cause those results to differ materially from those expressed in the forward-looking statements:

- cyber attacks impacting our networks or systems and any resulting financial or reputational impact;
- natural disasters, terrorist attacks or acts of war or significant litigation and any resulting financial or reputational impact;
- disruption of our key suppliers' or vendors' provisioning of products or services;
- material adverse changes in labor matters and any resulting financial or operational impact;
- the effects of competition in the markets in which we operate;
- failure to take advantage of developments in technology and address changes in consumer demand;
- performance issues or delays in the deployment of our 5G network resulting in significant costs or a reduction in the anticipated benefits of the enhancement to our networks;
- the inability to implement our business strategy;
- adverse conditions in the U.S. and international economies;
- changes in the regulatory environment in which we operate, including any increase in restrictions on our ability to operate our networks;
- our high level of indebtedness;
- an adverse change in the ratings afforded our debt securities by nationally accredited ratings organizations or adverse conditions in the credit markets affecting the cost, including interest rates, and/or availability of further financing;
- significant increases in benefit plan costs or lower investment returns on plan assets;
- changes in tax laws or treaties, or in their interpretation; and
- changes in accounting assumptions that regulatory agencies, including the SEC, may require or that result from changes in the accounting rules or their application, which could result in an impact on earnings.

Report of Management on Internal Control Over Financial Reporting

We, the management of Verizon Communications Inc., are responsible for establishing and maintaining adequate internal control over financial reporting of the company. Management has evaluated internal control over financial reporting of the company using the criteria for effective internal control established in Internal Control–Integrated Framework issued by the Committee of Sponsoring Organizations of the Treadway Commission in 2013.

Management has assessed the effectiveness of the company's internal control over financial reporting as of December 31, 2019. Based on this assessment, we believe that the internal control over financial reporting of the company is effective as of December 31, 2019. In connection with this assessment, there were no material weaknesses in the company's internal control over financial reporting identified by management.
The company's financial statements included in this Annual Report have been audited by Ernst & Young LLP, independent registered public accounting firm. Ernst & Young LLP has also provided an attestation report on the company's internal control over financial reporting.

/s/ **Hans E. Vestberg**

Hans E. Vestberg

Chairman and Chief Executive Officer

/s/ **Matthew D. Ellis**

Matthew D. Ellis

Executive Vice President and Chief Financial Officer

/s/ **Anthony T. Skiadas**

Anthony T. Skiadas

Senior Vice President and Controller

Report of Independent Registered Public Accounting Firm

To the Shareholders and the Board of Directors of Verizon Communications Inc.:

Opinion on Internal Control Over Financial Reporting

We have audited Verizon Communications Inc. and subsidiaries' (Verizon) internal control over financial reporting as of December 31, 2019, based on criteria established in Internal Control-Integrated Framework issued by the Committee of Sponsoring Organizations of the Treadway Commission (2013 framework) (the COSO criteria). In our opinion, Verizon maintained, in all material respects, effective internal control over financial reporting as of December 31, 2019, based on the COSO criteria.

We also have audited, in accordance with the standards of the Public Company Accounting Oversight Board (United States) (PCAOB), the consolidated balance sheets of Verizon as of December 31, 2019 and 2018, the related consolidated statements of income, comprehensive income, cash flows, and changes in equity for each of the three years in the period ended December 31, 2019, and the related notes and our report dated February 21, 2020 expressed an unqualified opinion thereon.

Basis for Opinion

Verizon's management is responsible for maintaining effective internal control over financial reporting and for its assessment of the effectiveness of internal control over financial reporting included in the accompanying Report of Management on Internal Control Over Financial Reporting. Our responsibility is to express an opinion on Verizon's internal control over financial reporting based on our audit. We are a public accounting firm registered with the PCAOB and are required to be independent with respect to Verizon in accordance with the U.S. federal securities laws and the applicable rules and regulations of the Securities and Exchange Commission and the PCAOB.

We conducted our audit in accordance with the standards of the PCAOB. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether effective internal control over financial reporting was maintained in all material respects.

Our audit included obtaining an understanding of internal control over financial reporting, assessing the risk that a material weakness exists, testing and evaluating the design and operating effectiveness of internal control based on the assessed risk, and performing such other procedures as we considered necessary in the circumstances. We believe that our audit provides a reasonable basis for our opinion.

Definition and Limitations of Internal Control Over Financial Reporting

A company's internal control over financial reporting is a process designed to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles. A company's internal control over financial reporting includes those policies and procedures that (1) pertain to the maintenance of records that, in reasonable detail, accurately and fairly reflect the transactions and dispositions of the assets of the company; (2) provide reasonable assurance that transactions are recorded as necessary to permit preparation of financial statements in accordance with generally accepted accounting principles, and that receipts and expenditures of the company are being made only in accordance with authorizations of management and directors of the company; and (3) provide reasonable assurance regarding prevention or timely detection of unauthorized acquisition, use, or disposition of the company's assets that could have a material effect on the financial statements.

Because of its inherent limitations, internal control over financial reporting may not prevent or detect misstatements. Also, projections of any evaluation of effectiveness to future periods are subject to the risk that controls may become inadequate because of changes in conditions, or that the degree of compliance with the policies or procedures may deteriorate.

/s/ **Ernst & Young LLP**

Ernst & Young LLP

New York, New York

February 21, 2020

Report of Independent Registered Public Accounting Firm

To the Shareholders and the Board of Directors of Verizon Communications Inc.:

Opinion on the Financial Statements

We have audited the accompanying consolidated balance sheets of Verizon Communications Inc. and subsidiaries (Verizon) as of December 31, 2019 and 2018, the related consolidated statements of income, comprehensive income, cash flows, and changes in equity for each of the three years in the period ended December 31, 2019, and the related notes (collectively referred to as the "consolidated financial statements"). In our opinion, the consolidated financial statements present fairly, in all material respects, the financial position of Verizon at December 31, 2019 and 2018, and the results of its operations and its cash flows for each of the three years in the period ended December 31, 2019, in conformity with U.S. generally accepted accounting principles.

We also have audited, in accordance with the standards of the Public Company Accounting Oversight Board (United States) (PCAOB), Verizon's internal control over financial reporting as of December 31, 2019, based on criteria established in Internal Control–Integrated Framework issued by the Committee of Sponsoring Organizations of the Treadway Commission (2013 framework) and our report dated February 21, 2020 expressed an unqualified opinion thereon.

Adoption of New Accounting Standards

ASU No. 2016-02
As discussed in Note 1 to the consolidated financial statements, effective January 1, 2019, Verizon changed its method of accounting for leases due to the adoption of Accounting Standards Update (ASU) No. 2016-02, Leases (Topic 842), and the related amendments, using the modified retrospective method.

ASU No. 2014-09
As discussed in Note 1 to the consolidated financial statements, effective January 1, 2018 Verizon changed its method for recognizing revenue as a result of the adoption of ASU No. 2014-09, Revenue from Contracts with Customers (Topic 606), and the amendments in ASUs 2015-14, 2016-08, 2016-10 and 2016-12 using the modified retrospective method.

Basis for Opinion

These financial statements are the responsibility of Verizon's management. Our responsibility is to express an opinion on Verizon's financial statements based on our audits. We are a public accounting firm registered with the PCAOB and are required to be independent with respect to Verizon in accordance with the U.S. federal securities laws and the applicable rules and regulations of the Securities and Exchange Commission and the PCAOB.

We conducted our audits in accordance with the standards of the PCAOB. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement, whether due to error or fraud. Our audits included performing procedures to assess the risks of material misstatement of the financial statements, whether due to error or fraud, and performing procedures that respond to those risks. Such procedures included examining, on a test basis, evidence regarding the amounts and disclosures in the financial statements. Our audits also included evaluating the accounting principles used and significant estimates made by management, as well as evaluating the overall presentation of the financial statements. We believe that our audits provide a reasonable basis for our opinion.

Critical Audit Matters

The critical audit matters communicated below are matters arising from the current period audit of the financial statements that were communicated or required to be communicated to the audit committee and that: (1) relate to accounts or disclosures that are material to the financial statements and (2) involved our especially challenging, subjective or complex judgments. The communication of critical audit matters does not alter in any way our opinion on the consolidated financial statements, taken as a whole, and we are not, by communicating the critical audit matters below, providing separate opinions on the critical audit matters or on the accounts or disclosures to which they relate.

Impairment Evaluation for Wireline Goodwill

Description of the Matter

At March 31, 2019, the Company's goodwill related to its historical Wireline reporting unit was $3.9 billion and represented 1.4% of total assets. As discussed in Notes 1 and 4 of the consolidated financial statements, goodwill is not amortized but rather is tested for impairment at the reporting unit level at least annually, or more frequently if impairment indicators are present. The impairment test compares the fair value of the reporting unit (calculated using a combination of a market approach and a discounted cash flow method) to its carrying amount. Effective April 1, 2019, the Company transitioned to its new segment reporting structure, which resulted in certain changes to its operating segments and reporting units. On March 31, 2019 the Company performed an impairment assessment of the impacted reporting units, including the Wireline reporting unit, immediately before the segment reorganization became effective.

Auditing management's goodwill impairment test was complex and highly judgmental due to the inherent subjectivity of developing an estimate of the fair value of the reporting unit, which is based on assumptions about future conditions, transactions, or events whose outcome is uncertain and will therefore be subject to change over time. In particular, the fair value estimate was sensitive to significant assumptions such as the weighted average cost of capital, revenue growth rate and operating margin, which are affected by expected future market or economic conditions.

How We Addressed the Matter in Our Audit

We obtained an understanding, evaluated the design and tested the operating effectiveness of controls over the Company's goodwill impairment review process. For example, we tested controls over the Company's development of prospective financial information and management's review of other key assumptions.

To test the estimated fair value of the Company's Wireline reporting unit prior to segment reorganization, our audit procedures included, among others, assessing the suitability and application of the valuation methodologies selected and evaluating the significant assumptions discussed above and underlying data used by the Company in its analysis. We compared the significant assumptions used by management to current industry and economic trends, changes in the Company's business model, customer base or product mix and other relevant factors. We performed sensitivity analyses of significant assumptions to determine what changes in assumptions are particularly sensitive when assessing the likelihood of impairment, or when calculating the amount of an impairment. In addition, we involved a valuation specialist to assist in the evaluation of the assumptions and other relevant information that are most significant to the fair value estimate. We also assessed the historical accuracy of management's forecasts of financial results used in developing prior fair value estimates to assist in evaluating the reliability of the current forecasts.

Valuation of Employee Benefit Obligations

Description of the Matter

The Company sponsors several pension plans and other post-employment benefit plans. At December 31, 2019, the Company's aggregate defined benefit pension obligation was $21.2 billion and exceeded the fair value of pension plan assets of $19.4 billion, resulting in an unfunded defined benefit pension obligation of $1.8 billion. Also, at December 31, 2019, the other postretirement benefits obligation was approximately $15.7 billion. As explained in Note 11 of the consolidated financial statements, the Company updates the estimates used to measure employee benefit obligations and plan assets in the fourth quarter and upon a remeasurement event to reflect the actual return on plan assets and updated actuarial assumptions.

Auditing the employee benefit obligations was complex due to the highly judgmental nature of the actuarial assumptions (e.g., discount rate, health care cost trends, per capita claims cost trends and mortality rates) used in the measurement process. These assumptions had a significant effect on the projected benefit obligation.

How We Addressed the Matter in Our Audit

We obtained an understanding, evaluated the design and tested the operating effectiveness of controls over the employee benefits obligation valuation process. For example, we tested controls over management's review of the employee benefit obligation calculations, the significant actuarial assumptions and the data inputs provided to the actuary.

To test the employee benefit obligations, our audit procedures included, among others, evaluating the methodologies used, the significant actuarial assumptions discussed above and the underlying data used by the Company. We compared the actuarial assumptions used by management to historical trends, current economic factors and evaluated the change in the employee benefit obligations from prior year due to the change in service cost, interest cost, actuarial gains and losses, benefit payments, contributions and other activities. In addition, we involved an actuarial specialist to assist in evaluating management's methodology for determining the discount rate that reflects the maturity and duration of the benefit payments and is used to measure the employee benefit obligations. As part of this assessment, we compared the projected cash flows to prior year projections and compared the current year benefits paid to the prior year projected cash flows. To evaluate the health care cost trends, per capita claims cost trends and the mortality rates, we involved an actuarial specialist to assist in evaluating the assumptions and assessed whether the information is consistent with publicly available information, and whether any market data adjusted for entity-specific adjustments were applied. We also tested the completeness and accuracy of the underlying data, including the participant data provided to management's actuarial specialists.

Income Taxes - Benefit from the disposition of stock of a foreign affiliate

Description of the Matter

As described in Note 12 to the consolidated financial statements, during the fourth quarter of 2019 the Company sold a minority interest in a foreign affiliate to unrelated parties resulting in the recognition of a tax benefit of approximately $2.2 billion.

Auditing the recognition and measurement of this income tax benefit required significant auditor judgment because the determination of whether the tax positions' technical merits are more likely than not to be sustained in an audit by a taxing authority is based on the application and interpretation of the relevant tax laws to the facts of the specific transaction.

How We Addressed the Matter in Our Audit	We obtained an understanding, evaluated the design and tested the operating effectiveness of controls over the Company's income tax processes. For example, we tested controls over management's review of the income tax technical merits of the transaction and the related recognition and measurement of the income tax benefit. To test the income tax benefit related to this transaction, our audit procedures included, among others, assessing the suitability and application of tax laws and legal rulings and evaluating the related conclusions. In addition, we involved our tax professionals to assist in the review and evaluation of management's third-party tax opinions and memoranda and other relevant agreements. We tested the completeness and accuracy of the data and calculations used to determine the amount of the income tax benefit recognized.

/s/ **Ernst & Young LLP**

Ernst & Young LLP

We have served as Verizon's auditor since 2000.

New York, New York

February 21, 2020

Consolidated Statements of Income Verizon Communications Inc. and Subsidiaries

(dollars in millions, except per share amounts)

Years Ended December 31,	2019	2018	2017
Operating Revenues			
Service revenues and other	**$ 110,305**	$ 108,605	$ 107,145
Wireless equipment revenues	**21,563**	22,258	18,889
Total Operating Revenues	**131,868**	130,863	126,034
Operating Expenses			
Cost of services (exclusive of items shown below)	**31,772**	32,185	30,916
Cost of wireless equipment	**22,954**	23,323	22,147
Selling, general and administrative expense (including net gain/(loss) on sale of divested businesses of $(94), $0 and $1,774, respectively)	**29,896**	31,083	28,592
Depreciation and amortization expense	**16,682**	17,403	16,954
Media goodwill impairment	**186**	4,591	—
Total Operating Expenses	**101,490**	108,585	98,609
Operating Income	**30,378**	22,278	27,425
Equity in losses of unconsolidated businesses	**(15)**	(186)	(77)
Other income (expense), net	**(2,900)**	2,364	(2,021)
Interest expense	**(4,730)**	(4,833)	(4,733)
Income Before (Provision) Benefit For Income Taxes	**22,733**	19,623	20,594
(Provision) benefit for income taxes	**(2,945)**	(3,584)	9,956
Net Income	**$ 19,788**	$ 16,039	$ 30,550
Net income attributable to noncontrolling interests	**$ 523**	$ 511	$ 449
Net income attributable to Verizon	**19,265**	15,528	30,101
Net Income	**$ 19,788**	$ 16,039	$ 30,550
Basic Earnings Per Common Share			
Net income attributable to Verizon	**$ 4.66**	$ 3.76	$ 7.37
Weighted-average shares outstanding (in millions)	**4,138**	4,128	4,084
Diluted Earnings Per Common Share			
Net income attributable to Verizon	**$ 4.65**	$ 3.76	$ 7.36
Weighted-average shares outstanding (in millions)	**4,140**	4,132	4,089

See Notes to Consolidated Financial Statements

Consolidated Statements of Comprehensive Income Verizon Communications Inc. and Subsidiaries

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Net Income	**$ 19,788**	$ 16,039	$ 30,550
Other Comprehensive Loss, Net of Tax (Expense) Benefit			
Foreign currency translation adjustments, net of tax of $(21), $(11) and $30	**16**	(117)	245
Unrealized gain (loss) on cash flow hedges, net of tax of $265, $(19) and $20	**(736)**	55	(31)
Unrealized gain (loss) on marketable securities, net of tax of $(2), $0 and $10	**7**	1	(14)
Defined benefit pension and postretirement plans, net of tax of $219, $284 and $144	**(659)**	(858)	(214)
Other comprehensive loss attributable to Verizon	**(1,372)**	(919)	(14)
Total Comprehensive Income	**$ 18,416**	$ 15,120	$ 30,536
Comprehensive income attributable to noncontrolling interests	**$ 523**	$ 511	$ 449
Comprehensive income attributable to Verizon	**17,893**	14,609	30,087
Total Comprehensive Income	**$ 18,416**	$ 15,120	$ 30,536

See Notes to Consolidated Financial Statements

Consolidated Balance Sheets Verizon Communications Inc. and Subsidiaries

(dollars in millions, except per share amounts)

At December 31,	2019	2018
Assets		
Current assets		
Cash and cash equivalents	**$ 2,594**	$ 2,745
Accounts receivable, net of allowances of $733 and $765	**25,429**	25,102
Inventories	**1,422**	1,336
Prepaid expenses and other	**8,028**	5,453
Total current assets	**37,473**	34,636
Property, plant and equipment	**265,734**	252,835
Less accumulated depreciation	**173,819**	163,549
Property, plant and equipment, net	**91,915**	89,286
Investments in unconsolidated businesses	**558**	671
Wireless licenses	**95,059**	94,130
Goodwill	**24,389**	24,614
Other intangible assets, net	**9,498**	9,775
Operating lease right-of-use assets	**22,694**	—
Other assets	**10,141**	11,717
Total assets	**$ 291,727**	$ 264,829
Liabilities and Equity		
Current liabilities		
Debt maturing within one year	**$ 10,777**	$ 7,190
Accounts payable and accrued liabilities	**21,806**	22,501
Current operating lease liabilities	**3,261**	—
Other current liabilities	**9,024**	8,239
Total current liabilities	**44,868**	37,930
Long-term debt	**100,712**	105,873
Employee benefit obligations	**17,952**	18,599
Deferred income taxes	**34,703**	33,795
Non-current operating lease liabilities	**18,393**	—
Other liabilities	**12,264**	13,922
Total long-term liabilities	**184,024**	172,189
Commitments and Contingencies (Note 16)		
Equity		
Series preferred stock ($0.10 par value; 250,000,000 shares authorized; none issued)	**—**	—
Common stock ($0.10 par value; 6,250,000,000 shares authorized in each period; 4,291,433,646 issued in each period)	**429**	429
Additional paid in capital	**13,419**	13,437
Retained earnings	**53,147**	43,542
Accumulated other comprehensive income	**998**	2,370
Common stock in treasury, at cost (155,605,527 and 159,400,267 shares outstanding)	**(6,820)**	(6,986)
Deferred compensation – employee stock ownership plans and other	**222**	353
Noncontrolling interests	**1,440**	1,565
Total equity	**62,835**	54,710
Total liabilities and equity	**$ 291,727**	$ 264,829

See Notes to Consolidated Financial Statements

Consolidated Statements of Cash Flows Verizon Communications Inc. and Subsidiaries

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Cash Flows from Operating Activities			
Net Income	**$ 19,788**	$ 16,039	$ 30,550
Adjustments to reconcile net income to net cash provided by operating activities:			
Depreciation and amortization expense	**16,682**	17,403	16,954
Employee retirement benefits	**(284)**	(2,657)	440
Deferred income taxes	**1,232**	389	(14,463)
Provision for uncollectible accounts	**1,588**	980	1,167
Equity in losses of unconsolidated businesses, net of dividends received	**74**	231	117
Net loss (gain) on sale of divested businesses	**94**	—	(1,774)
Media goodwill impairment	**186**	4,591	—
Changes in current assets and liabilities, net of effects from acquisition/disposition of businesses:			
Accounts receivable	**(1,471)**	(2,667)	(5,674)
Inventories	**(76)**	(324)	168
Prepaid expenses and other	**(2,807)**	37	27
Accounts payable and accrued liabilities and Other current liabilities	**(2,359)**	1,777	(459)
Discretionary employee benefits contributions	**(300)**	(1,679)	(3,411)
Other, net	**3,399**	219	676
Net cash provided by operating activities	**35,746**	34,339	24,318
Cash Flows from Investing Activities			
Capital expenditures (including capitalized software)	**(17,939)**	(16,658)	(17,247)
Acquisitions of businesses, net of cash acquired	**(29)**	(230)	(5,880)
Acquisitions of wireless licenses	**(898)**	(1,429)	(583)
Proceeds from dispositions of businesses	**28**	—	3,614
Other, net	**1,257**	383	1,640
Net cash used in investing activities	**(17,581)**	(17,934)	(18,456)
Cash Flows from Financing Activities			
Proceeds from long-term borrowings	**10,079**	5,967	27,707
Proceeds from asset-backed long-term borrowings	**8,576**	4,810	4,290
Repayments of long-term borrowings and finance lease obligations	**(17,584)**	(10,923)	(23,837)
Repayments of asset-backed long-term borrowings	**(6,302)**	(3,635)	(400)
Dividends paid	**(10,016)**	(9,772)	(9,472)
Other, net	**(2,917)**	(1,824)	(4,439)
Net cash used in financing activities	**(18,164)**	(15,377)	(6,151)
Increase (decrease) in cash, cash equivalents and restricted cash	**1**	1,028	(289)
Cash, cash equivalents and restricted cash, beginning of period	**3,916**	2,888	3,177
Cash, cash equivalents and restricted cash, end of period (Note 1)	**$ 3,917**	$ 3,916	$ 2,888

See Notes to Consolidated Financial Statements

Consolidated Statements of Changes in Equity Verizon Communications Inc. and Subsidiaries

(dollars in millions, except per share amounts, and shares in thousands)

Years Ended December 31,	2019		2018		2017	
	Shares	**Amount**	Shares	Amount	Shares	Amount
Common Stock						
Balance at beginning of year	**4,291,434**	**$ 429**	4,242,374	$ 424	4,242,374	$ 424
Common shares issued	**—**	**—**	49,060	5	—	—
Balance at end of year	**4,291,434**	**429**	4,291,434	429	4,242,374	424
Additional Paid In Capital						
Balance at beginning of year		**13,437**		11,101		11,182
Other		**(18)**		2,336		(81)
Balance at end of year		**13,419**		13,437		11,101
Retained Earnings						
Balance at beginning of year		**43,542**		35,635		15,059
Opening balance sheet adjustment (Note 1)		**410**		2,232		—
Adjusted opening balance		**43,952**		37,867		15,059
Net income attributable to Verizon		**19,265**		15,528		30,101
Dividends declared ($2.435, $2.385, $2.335 per share)		**(10,070)**		(9,853)		(9,525)
Balance at end of year		**53,147**		43,542		35,635
Accumulated Other Comprehensive Income						
Balance at beginning of year attributable to Verizon		**2,370**		2,659		2,673
Opening balance sheet adjustment (Note 1)		**—**		630		—
Adjusted opening balance		**2,370**		3,289		2,673
Foreign currency translation adjustments		**16**		(117)		245
Unrealized gain (loss) on cash flow hedges		**(736)**		55		(31)
Unrealized gain (loss) on marketable securities		**7**		1		(14)
Defined benefit pension and postretirement plans		**(659)**		(858)		(214)
Other comprehensive loss		**(1,372)**		(919)		(14)
Balance at end of year attributable to Verizon		**998**		2,370		2,659
Treasury Stock						
Balance at beginning of year	**(159,400)**	**(6,986)**	(162,898)	(7,139)	(165,690)	(7,263)
Employee plans (Note 14)	**3,790**	**166**	3,494	153	2,787	124
Shareholder plans (Note 14)	**4**	**—**	4	—	5	—
Balance at end of year	**(155,606)**	**(6,820)**	(159,400)	(6,986)	(162,898)	(7,139)
Deferred Compensation-ESOPs and Other						
Balance at beginning of year		**353**		416		449
Restricted stock equity grant		**140**		162		157
Amortization		**(271)**		(225)		(190)
Balance at end of year		**222**		353		416
Noncontrolling Interests						
Balance at beginning of year		**1,565**		1,591		1,508
Opening balance sheet adjustment (Note 1)		**1**		44		—
Adjusted opening balance		**1,566**		1,635		1,508
Total comprehensive income		**523**		511		449
Distributions and other		**(649)**		(581)		(366)

Balance at end of year	**1,440**	1,565	1,591
Total Equity	**$ 62,835**	$ 54,710	$ 44,687

See Notes to Consolidated Financial Statements

Notes to Consolidated Financial Statements Verizon Communications Inc. and Subsidiaries

Note 1. Description of Business and Summary of Significant Accounting Policies

Description of Business

Verizon Communications Inc. (Verizon or the Company) is a holding company that, acting through its subsidiaries, is one of the world's leading providers of communications, information and entertainment products and services to consumers, businesses and government entities. With a presence around the world, we offer voice, data and video services and solutions on our networks that are designed to meet customers' demand for mobility, reliable network connectivity, security and control.

In November 2018, we announced a strategic reorganization of our business. Under the new structure, effective April 1, 2019, there are two reportable segments that we operate and manage as strategic business units - Verizon Consumer Group (Consumer) and Verizon Business Group (Business).

Our Consumer segment provides consumer-focused wireless and wireline communications services and products. Our wireless services are provided across one of the most extensive wireless networks in the United States (U.S.) under the Verizon brand and through wholesale and other arrangements. Our wireline services are provided in nine states in the Mid-Atlantic and Northeastern U.S., as well as Washington D.C., over our 100% fiber-optic network under the Fios brand and over a traditional copper-based network to customers who are not served by Fios. Our Consumer segment's wireless and wireline products and services are available to our retail customers, as well as resellers that purchase wireless network access from us on a wholesale basis.

Our Business segment provides wireless and wireline communications services and products, video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various Internet of Things (IoT) services and products. We provide these products and services to businesses, government customers and wireless and wireline carriers across the U.S. and select products and services to customers around the world.

Consolidation

The method of accounting applied to investments, whether consolidated or equity, involves an evaluation of all significant terms of the investments that explicitly grant or suggest evidence of control or influence over the operations of the investee. The consolidated financial statements include our controlled subsidiaries, as well as variable interest entities (VIE) where we are deemed to be the primary beneficiary. For controlled subsidiaries that are not wholly-owned, the noncontrolling interests are included in Net income and Total equity. Investments in businesses that we do not control, but have the ability to exercise significant influence over operating and financial policies, are accounted for using the equity method. Equity method investments are included in Investments in unconsolidated businesses in our consolidated balance sheets. All significant intercompany accounts and transactions have been eliminated.

Use of Estimates

We prepare our financial statements using U.S. generally accepted accounting principles (GAAP), which requires management to make estimates and assumptions that affect reported amounts and disclosures. Actual results could differ from those estimates.

Examples of significant estimates include the allowance for doubtful accounts, the recoverability of property, plant and equipment, the incremental borrowing rate for the lease liability, the recoverability of intangible assets and other long-lived assets, fair value measurements, including those related to financial instruments, goodwill, spectrum licenses and intangible assets, unrecognized tax benefits, valuation allowances on tax assets, pension and postretirement benefit obligations, contingencies and the identification and valuation of assets acquired and liabilities assumed in connection with business combinations.

Revenue Recognition

We earn revenue from contracts with customers, primarily through the provision of telecommunications and other services and through the sale of wireless equipment. These services include a variety of communication and connectivity services for our Consumer and Business customers including other carriers that use our facilities to provide services to their customers, as well as professional and integrated managed services for our large enterprises and government customers. We account for these revenues under Accounting Standards Update (ASU) 2014-09, "Revenue from Contracts with Customers" (Topic 606), which we adopted on January 1, 2018, using the modified retrospective approach. This standard update, along with related subsequently issued updates, clarifies the principles for recognizing revenue and develops a common revenue standard for GAAP. The standard update also amends current guidance for the recognition of costs to obtain and fulfill contracts with customers such that incremental costs of obtaining and direct costs of fulfilling contracts with customers are deferred and amortized consistent with the transfer of the related good or service.

We also earn revenues that are not accounted for under Topic 606 from leasing arrangements (such as those for towers and equipment), captive reinsurance arrangements primarily related to wireless device insurance and the interest on equipment financed under a device payment plan agreement when sold to the customer by an authorized agent.

Nature of Products and Services

Telecommunications

Service

We offer wireless services through a variety of plans on a postpaid or prepaid basis. For wireless service, we recognize revenue using an output method, either as the service allowance units are used or as time elapses, because it reflects the pattern by which we satisfy our performance obligation through the transfer of service to the customer. Monthly service is generally billed in advance, which results in a contract liability. See Note 2 for additional information. For postpaid plans, where monthly usage exceeds the allowance, the overage usage represents options held by the customer for incremental services and the usage-based fee is recognized when the customer exercises the option (typically on a month-to-month basis).

For our contracts related to wireline communication and connectivity services, in general, fixed monthly fees for service are billed one month in advance, which results in a contract liability, and service revenue is recognized over the enforceable contract term as the service is rendered, as the customer simultaneously receives and consumes the benefits of the services through network access and usage. While substantially all of our wireline service revenue contracts are the result of providing access to our networks, revenue from services that are not fixed in amount and, instead, are based on usage are generally billed in arrears and recognized as the usage occurs.

Equipment

We sell wireless devices and accessories under the Verizon brand. Equipment revenue is generally recognized when the products are delivered to and accepted by the customer, as this is when control passes to the customer. In addition to offering the sale of equipment on a standalone basis, we have two primary offerings through which customers pay for a wireless device, in connection with a service contract: fixed-term plans and device payment plans.

Under a fixed-term plan, the customer is sold the wireless device without any upfront charge or at a discounted price in exchange for entering into a fixed-term service contract (typically for a term of 24 months or less).

Under a device payment plan, the customer is sold the wireless device in exchange for a non-interest-bearing installment note, which is repaid by the customer, typically over a 24-month term, and concurrently enters into a month-to-month contract for wireless service. We may offer certain promotions that provide billing credits applied over a specified term, contingent upon the customer maintaining service. The credits are included in the transaction price, which are allocated to the performance obligations based on their relative selling price and are recognized when earned.

A financing component exists in both our fixed-term plans and device payment plans because the timing of the payment for the device, which occurs over the contract term, differs from the satisfaction of the performance obligation, which occurs at contract inception upon transfer of the device to the customer. We periodically assess, at the contract level, the significance of the financing component inherent in our fixed-term and device payment plan receivable based on qualitative and quantitative considerations related to our customer classes. These considerations include assessing the commercial objective of our plans, the term and duration of financing provided, interest rates prevailing in the marketplace, and credit risks of our customer classes, all of which impact our selection of appropriate discount rates. Based on current facts and circumstances, we determined that the financing component in our existing wireless device payments and fixed-term contracts sold through the direct channel is not significant and therefore is not accounted for separately. See Note 8 for additional information on the interest on equipment financed on a device payment plan agreement when sold to the customer by an authorized agent in our indirect channel.

Wireless Contracts

For our wireless contracts, total contract revenue, which represents the transaction price for wireless service and wireless equipment, is allocated between service and equipment revenue based on their estimated standalone selling prices. We estimate the standalone selling price of the device or accessory to be its retail price excluding subsidies or conditional purchase discounts. We estimate the standalone selling price of wireless service to be the price that we offer to customers on month-to-month contracts that can be cancelled at any time without penalty (i.e., when there is no fixed-term for service) or when service is procured without the concurrent purchase of a wireless device. In addition, we also assess whether the service term is impacted by certain legally enforceable rights and obligations in our contract with customers, such as penalties that a customer would have to pay to early terminate a fixed-term contract or billing credits that would cease if the month-to-month wireless service is canceled. The assessment of these legally enforceable rights and obligations involves judgment and impacts our determination of the transaction price and related disclosures.

From time to time, we may offer certain promotions that provide our customers on device payment plans with the right to upgrade to a new device after paying a specified portion of their device payment plan agreement amount and trading in their device in good working order. We account for this trade-in right as a guarantee obligation. The full amount of the trade-in right's fair value is recognized as a guarantee liability and results in a reduction to the revenue recognized upon the sale of the device. The guarantee liability was insignificant at December 31, 2019 and 2018. The total transaction price is reduced by the guarantee, which is accounted for outside the scope of Topic 606, and the remaining transaction price is allocated between the performance obligations within the contract.

Our fixed-term plans generally include the sale of a wireless device at subsidized prices. This results in the creation of a contract asset at the time of sale, which represents the recognition of equipment revenue in excess of amounts billed.

For our device payment plans, billing credits are accounted for as consideration payable to a customer and are included in the determination of total transaction price, resulting in a contract liability.

We may provide a right of return on our products and services for a short time period after a sale. These rights are accounted for as variable consideration when determining the transaction price, and accordingly we recognize revenue based on the estimated amount to which we expect to be entitled after considering expected returns. Returns and credits are estimated at contract inception and updated at the end of each reporting period as additional information becomes available. We also may provide credits or incentives on our products and services for contracts with resellers, which are accounted for as variable consideration when estimating the amount of revenue to recognize.

Wireline Contracts

Total consideration for wireline services that are bundled in a single contract is allocated to each performance obligation based on our standalone selling price for each service. While many contracts include one or more service performance obligations, the revenue recognition pattern is generally not impacted by the allocation since the services are generally satisfied over the same period of time. We estimate the standalone selling price to be the price of the services when sold on a standalone basis without any promotional discount. In addition, we also assess whether the service term is impacted by certain legally enforceable rights and obligations in our contract with customers such as penalties that a customer would have to pay to early terminate a fixed-term contract. The assessment of these legally enforceable rights and obligations involves judgment and impacts our determination of transaction price and related disclosures.

We may provide performance-based credits or incentives on our products and services for contracts with our Business customers, which are accounted for as variable consideration when estimating the transaction price. Credits are estimated at contract inception and are updated at the end of each reporting period as additional information becomes available.

Wireless and Wireline Contracts

For offers that include third-party providers, we evaluate whether we are acting as the principal or as the agent with respect to the goods or services provided to the customer. This principal-versus-agent assessment involves judgment and focuses on whether the facts and circumstances of the arrangement indicate that the goods or services were controlled by us prior to transferring them to the customer. To evaluate if we have control, we consider various factors including whether we are primarily responsible for fulfillment, bear risk of loss and have discretion over pricing.

Other

Advertising revenues are generated through display advertising and search advertising. Display advertising revenue is generated by the display of graphical advertisements and other performance-based advertising. Search advertising revenue is generated when a consumer clicks on a text-based advertisement on the search results page. Our Media business, Verizon Media, primarily earns revenue through display advertising on Verizon Media properties, as well as on third-party properties through our advertising platforms, search advertising and subscription arrangements. Revenue for display and search advertising contracts is recognized as ads are delivered, while subscription contracts are recognized over time. We are generally the principal in transactions carried out through our advertising platforms, and therefore report gross revenue based on the amount billed to our customers. The control and transfer of digital advertising inventory occurs in a rapid, real-time environment, where our proprietary technology enables us to identify, enhance, verify and solely control digital advertising inventory that we then sell to our customers. Our control is further supported by us being primarily responsible to our customers for fulfillment and the fact that we can exercise a level of discretion over pricing.

We offer telematics services including smart fleet management and optimization software. Telematics service revenue is generated primarily through subscription contracts. We recognize revenue over time for our subscription contracts.

We report taxes collected from customers on behalf of governmental authorities on revenue-producing transactions on a net basis.

Maintenance and Repairs

We charge the cost of maintenance and repairs, including the cost of replacing minor items not constituting substantial betterments, principally to Cost of services as these costs are incurred.

Advertising Costs

Costs for advertising products and services, as well as other promotional and sponsorship costs, are charged to Selling, general and administrative expense in the periods in which they are incurred. See Note 15 for additional information.

Earnings Per Common Share

Basic earnings per common share are based on the weighted-average number of shares outstanding during the period. Where appropriate, diluted earnings per common share include the dilutive effect of shares issuable under our stock-based compensation plans.

There were a total of approximately 2 million, 4 million and 5 million outstanding dilutive securities, primarily consisting of restricted stock units, included in the computation of diluted earnings per common share for the years ended December 31, 2019, 2018 and 2017, respectively.

Cash, Cash Equivalents and Restricted Cash

We consider all highly liquid investments with an original maturity of 90 days or less when purchased to be cash equivalents. Cash equivalents are stated at cost, which approximates quoted market value and includes amounts held in money market funds.

Cash collections on the device payment plan agreement receivables collateralizing asset-backed debt securities are required at certain specified times to be placed into segregated accounts. Deposits to the segregated accounts are considered restricted cash and are included in Prepaid expenses and other and Other assets in our consolidated balance sheets.

Cash, cash equivalents and restricted cash are included in the following line items in the consolidated balance sheets:

(dollars in millions)

At December 31,	**2019**	2018	**Increase / (Decrease)**
Cash and cash equivalents	**$ 2,594**	$ 2,745	$ (151)
Restricted cash:			
Prepaid expenses and other	**1,221**	1,047	174
Other assets	**102**	124	(22)
Cash, cash equivalents and restricted cash	**$ 3,917**	$ 3,916	$ 1

Investments in Debt and Equity Securities

Investments in equity securities that are not accounted for under equity method accounting or result in consolidation are to be measured at fair value. For investments in equity securities without readily determinable fair values, Verizon elects the measurement alternative permitted under GAAP to measure these investments at cost, less any impairment, plus or minus changes resulting from observable price changes in orderly transactions for an identical or similar investment of the same issuer. For investments in debt securities without quoted prices, Verizon uses an alternative matrix pricing method. Investments in equity securities that do not result in consolidation of the investee are included in Investments in unconsolidated businesses and debt securities are included in Other assets in our consolidated balance sheets.

Allowance for Doubtful Accounts

Accounts receivable are recorded in the consolidated financial statements at cost net of an allowance for credit losses, with the exception of indirect-channel device payment plan loans. We maintain allowances for uncollectible accounts receivable, including our direct-channel device payment plan agreement receivables, for estimated losses resulting from the failure or inability of our customers to make required payments. Indirect-channel device payment loans are considered financial instruments and are initially recorded at fair value net of imputed interest, and credit losses are recorded as incurred. However, loan balances are assessed quarterly for impairment and an allowance is recorded if the loan is considered impaired. Our allowance for uncollectible accounts receivable is based on management's assessment of the collectability of specific customer accounts and includes consideration of the credit worthiness and financial condition of those customers. We record an allowance to reduce the receivables to the amount that is reasonably believed to be collectible. We also record an allowance for all other receivables based on multiple factors including historical experience with bad debts, the general economic environment and the aging of such receivables. Similar to traditional service revenue, we record direct device payment plan agreement bad debt expense based on an estimate of the percentage of equipment revenue that will not be collected. This estimate is based on a number of factors including historical write-off experience, credit quality of the customer base and other factors such as macroeconomic conditions. We monitor the aging of our accounts with device payment plan agreement receivables and write-off account balances if collection efforts are unsuccessful and future collection is unlikely.

Inventories

Inventory consists of wireless and wireline equipment held for sale, which is carried at the lower of cost (determined principally on either an average cost or first-in, first-out basis) or net realizable value.

Plant and Depreciation

We record property, plant and equipment at cost. Property, plant and equipment are generally depreciated on a straight-line basis.

Leasehold improvements are amortized over the shorter of the estimated life of the improvement or the remaining term of the related lease, calculated from the time the asset was placed in service.

When depreciable assets are retired or otherwise disposed of, the related cost and accumulated depreciation are deducted from the property, plant and equipment accounts and any gains or losses on disposition are recognized in income.

We capitalize and depreciate network software purchased or developed within property, plant and equipment assets. We also capitalize interest associated with the acquisition or construction of network-related assets. Capitalized interest is reported as a reduction in interest expense and depreciated as part of the cost of the network-related assets.

In connection with our ongoing review of the estimated useful lives of property, plant and equipment during 2018, we determined that the average useful lives of certain assets would be increased. These changes in estimates were applied prospectively in 2018 and resulted in a decrease to

depreciation expense of $271 million for the year ended December 31, 2018. While the timing and extent of current deployment plans are subject to ongoing analysis and modification, we believe that the current estimates of useful lives are reasonable.

Computer Software Costs

We capitalize the cost of internal-use network and non-network software that has a useful life in excess of one year. Subsequent additions, modifications or upgrades to internal-use network and non-network software are capitalized only to the extent that they allow the software to perform a task it previously did not perform. Planning, software maintenance and training costs are expensed in the period in which they are incurred. Also, we capitalize interest associated with the development of internal-use network and non-network software. Capitalized non-network internal-use software costs are amortized using the straight-line method over a period of 3 to 7 years and are included in Other intangible assets, net in our consolidated balance sheets. For a discussion of our impairment policy for capitalized software costs, see "Goodwill and Other Intangible Assets" below. Also, see Note 4 for additional information of internal-use non-network software reflected in our consolidated balance sheets.

Goodwill and Other Intangible Assets

Goodwill

Goodwill is the excess of the acquisition cost of businesses over the fair value of the identifiable net assets acquired. Impairment testing for goodwill is performed annually in the fourth quarter or more frequently if impairment indicators are present.

To determine if goodwill is potentially impaired, we have the option to perform a qualitative assessment. However, we may elect to bypass the qualitative assessment and perform a quantitative impairment test even if no indications of a potential impairment exist. The quantitative impairment test for goodwill is performed at the reporting unit level and compares the fair value of the reporting unit (calculated using a combination of a market approach and a discounted cash flow method) to its carrying value. Estimated fair values of reporting units are Level 3 measures in the fair value hierarchy, see Fair Value Measurements discussion below for additional information.

Under the qualitative assessment, we consider several qualitative factors, including the business enterprise value of the reporting unit from the last quantitative test and the excess of fair value over carrying value from this test, macroeconomic conditions (including changes in interest rates and discount rates), industry and market considerations (including industry revenue and Earnings before interest, taxes, depreciation and amortization (EBITDA) margin projections), the recent and projected financial performance of the reporting unit, as well as other factors.

The market approach includes the use of comparative multiples of guideline companies to corroborate discounted cash flow results. The discounted cash flow method is based on the present value of two components, a projected cash flows and a terminal value. The terminal value represents the expected normalized future cash flows of the reporting unit beyond the cash flows from the discrete projection period. The fair value of the reporting unit is calculated based on the sum of the present value of the cash flows from the discrete period and the present value of the terminal value. The discount rate represents our estimate of the weighted-average cost of capital, or expected return, that a marketplace participant would have required as of the valuation date. If the carrying value exceeds the fair value, an impairment charge is booked for the excess carrying value over fair value, limited to the total amount of goodwill of that reporting unit. During the fourth quarter each year, we update our five-year strategic planning review for each of our reporting units. Those plans consider current economic conditions and trends, estimated future operating results, our view of growth-rates and-anticipated future economic and regulatory conditions.

See Note 4 for additional information regarding our goodwill impairment testing.

Intangible Assets Not Subject to Amortization

A significant portion of our intangible assets are wireless licenses that provide our wireless operations with the exclusive right to utilize designated radio frequency spectrum to provide wireless communication services. While licenses are issued for only a fixed time, generally ten years, such licenses are subject to renewal by the Federal Communications Commission (FCC). License renewals have occurred routinely and at nominal cost. Moreover, we have determined that there are currently no legal, regulatory, contractual, competitive, economic or other factors that limit the useful life of our wireless licenses. As a result, we treat the wireless licenses as an indefinite-lived intangible asset. We re-evaluate the useful life determination for wireless licenses each year to determine whether events and circumstances continue to support an indefinite useful life. We aggregate our wireless licenses into one single unit of accounting, as we utilize our wireless licenses on an integrated basis as part of our nationwide wireless network.

We test our wireless licenses for potential impairment annually or more frequently if impairment indicators are present. We have the option to first perform a qualitative assessment to determine whether it is necessary to perform a quantitative impairment test. However, we may elect to bypass the qualitative assessment in any period and proceed directly to performing the quantitative impairment test. Our quantitative assessment consists of comparing the estimated fair value of our aggregate wireless licenses to the aggregated carrying amount as of the test date. Using a quantitative assessment, we estimate the fair value of our aggregate wireless licenses using the Greenfield approach. The Greenfield approach is an income based valuation approach that values the wireless licenses by calculating the cash flow generating potential of a hypothetical start-up company that goes into business with no assets except the wireless licenses to be valued. A discounted cash flow analysis is used to estimate what a marketplace participant would be willing to pay to purchase the aggregated wireless licenses as of the valuation date. If the estimated fair value of the aggregated wireless licenses is less than the aggregated carrying amount of the wireless licenses, then an impairment charge is recognized. As part of our qualitative assessment, we consider several qualitative factors including the business enterprise value of our historical Wireless segment, macroeconomic conditions (including changes in interest rates and discount rates), industry and market considerations

(including industry revenue and EBITDA margin projections), the recent and projected financial performance of our historical Wireless segment, as well as other factors. See Note 4 for additional information regarding our impairment tests.

Interest expense incurred while qualifying activities are performed to ready wireless licenses for their intended use is capitalized as part of wireless licenses. The capitalization period ends when the development is discontinued or substantially completed and the license is ready for its intended use.

Wireless licenses can be purchased through public auctions conducted by the FCC. Deposits required to participate in these auctions and purchase licenses are recorded as other non-current assets until the corresponding licenses are received and within Net cash used in investing activities in our consolidated statements of cash flows.

Intangible Assets Subject to Amortization and Long-Lived Assets

Our intangible assets that do not have indefinite lives (primarily customer lists and non-network internal-use software) are amortized over their estimated useful lives. All of our intangible assets subject to amortization and other long-lived assets are reviewed for impairment whenever events or changes in circumstances indicate that the carrying amount of the asset may not be recoverable. If any indications of impairment are present, we would test for recoverability by comparing the carrying amount of the asset group to the net undiscounted cash flows expected to be generated from the asset group. If those net undiscounted cash flows do not exceed the carrying amount, we would perform the next step, which is to determine the fair value of the asset and record an impairment, if any. We re-evaluate the useful life determinations for these intangible assets each year to determine whether events and circumstances warrant a revision to their remaining useful lives.

For information related to the carrying amount of goodwill, wireless licenses and other intangible assets, as well as the major components and average useful lives of our other acquired intangible assets, see Note 4.

Fair Value Measurements

Fair value of financial and non-financial assets and liabilities is defined as an exit price, representing the amount that would be received to sell an asset or paid to transfer a liability in an orderly transaction between market participants. The three-tier hierarchy for inputs used in measuring fair value, which prioritizes the inputs used in the methodologies of measuring fair value for assets and liabilities, is as follows:

Level 1—Quoted prices in active markets for identical assets or liabilities
Level 2—Observable inputs other than quoted prices in active markets for identical assets and liabilities
Level 3—Unobservable pricing inputs in the market

Financial assets and financial liabilities are classified in their entirety based on the lowest level of input that is significant to the fair value measurements. Our assessment of the significance of a particular input to the fair value measurements requires judgment and may affect the valuation of the assets and liabilities being measured and their categorization within the fair value hierarchy.

Income Taxes

Our effective tax rate is based on pre-tax income, statutory tax rates, tax laws and regulations and tax planning strategies available to us in the various jurisdictions in which we operate.

Deferred income taxes are provided for temporary differences in the basis between financial statement and income tax assets and liabilities. Deferred income taxes are recalculated annually at tax rates in effect for the years in which those tax assets and liabilities are expected to be realized or settled. We record valuation allowances to reduce our deferred tax assets to the amount that is more likely than not to be realized.

We use a two-step approach for recognizing and measuring tax benefits taken or expected to be taken in a tax return. The first step is recognition: we determine whether it is more likely than not that a tax position will be sustained upon examination, including resolution of any related appeals or litigation processes, based on the technical merits of the position. In evaluating whether a tax position has met the more-likely-than-not recognition threshold, we presume that the position will be examined by the appropriate taxing authority that has full knowledge of all relevant information. The second step is measurement: a tax position that meets the more-likely-than-not recognition threshold is measured to determine the amount of benefit to recognize in the financial statements. The tax position is measured at the largest amount of benefit that is greater than 50 percent likely of being realized upon ultimate settlement. Differences between tax positions taken in a tax return and amounts recognized in the financial statements will generally result in one or more of the following: an increase in a liability for income taxes payable, a reduction of an income tax refund receivable, a reduction in a deferred tax asset or an increase in a deferred tax liability.

Significant management judgment is required in evaluating our tax positions and in determining our effective tax rate.

Stock-Based Compensation

We measure and recognize compensation expense for all stock-based compensation awards made to employees and directors based on estimated fair values. See Note 10 for additional information.

Foreign Currency Translation and Transactions

The functional currency of our foreign operations is generally the local currency. For these foreign entities, we translate their financial statements into U.S. dollars using average exchange rates for the period for income statement amounts and using end-of-period exchange rates for assets and liabilities. We record these translation adjustments in Accumulated other comprehensive income, a separate component of Equity, in our consolidated balance sheets. We record exchange gains and losses resulting from the conversion of transaction currency to functional currency as a component of Other income (expense), net.

Employee Benefit Plans

Pension and postretirement health care and life insurance benefits earned during the year, as well as interest on projected benefit obligations, are accrued. Prior service costs and credits resulting from changes in plan benefits are generally amortized over the average remaining service period of the employees expected to receive benefits. Expected return on plan assets is determined by applying the return on assets assumption to the actual fair value of plan assets. Actuarial gains and losses are recognized in Other income (expense), net in the year in which they occur. These gains and losses are measured annually as of December 31 or upon a remeasurement event. Verizon management employees no longer earn pension benefits or earn service towards the Company retiree medical subsidy. See Note 11 for additional information.

We recognize a pension or a postretirement plan's funded status as either an asset or liability in the consolidated balance sheets. Also, we measure any unrecognized prior service costs and credits that arise during the period as a component of Accumulated other comprehensive income, net of applicable income tax.

Derivative Instruments

We enter into derivative transactions primarily to manage our exposure to fluctuations in foreign currency exchange rates and interest rates. We employ risk management strategies, which may include the use of a variety of derivatives including cross currency swaps, forward starting interest rate swaps, interest rate swaps, interest rate caps and foreign exchange forwards. We do not hold derivatives for trading purposes.

We measure all derivatives at fair value and recognize them as either assets or liabilities in our consolidated balance sheets. Our derivative instruments are valued primarily using models based on readily observable market parameters for all substantial terms of our derivative contracts and thus are classified as Level 2. Changes in the fair values of derivative instruments not qualifying for hedge accounting are recognized in earnings in the current period. For fair value hedges, the change in the fair value of the derivative instruments is recognized in earnings, along with the change in the fair value of the hedged item. For cash flow hedges, the change in the fair value of the derivative instruments is reported in Other comprehensive income (loss) and recognized in earnings when the hedged item is recognized in earnings. For net investment hedges of certain of our foreign operations, the change in the fair value of the derivative instruments is reported in Other comprehensive income (loss) as part of the cumulative translation adjustment and partially offset the impact of foreign currency changes on the value of our net investment. See Note 9 for additional information.

Variable Interest Entities

VIEs are entities that lack sufficient equity to permit the entity to finance its activities without additional subordinated financial support from other parties, have equity investors that do not have the ability to make significant decisions relating to the entity's operations through voting rights, do not have the obligation to absorb the expected losses, or do not have the right to receive the residual returns of the entity. We consolidate the assets and liabilities of VIEs when we are deemed to be the primary beneficiary. The primary beneficiary is the party that has the power to make the decisions that most significantly affect the economic performance of the VIE and has the obligation to absorb losses or the right to receive benefits that could potentially be significant to the VIE.

Recently Adopted Accounting Standards

The following ASUs were issued by Financial Accounting Standards Board (FASB), and have been recently adopted by Verizon.

Description	Date of Adoption	Effect on Financial Statements
ASU 2016-02, ASU 2018-01, ASU 2018-10, ASU 2018-11, ASU 2018-20 and ASU 2019-01, Leases (Topic 842)		
The FASB issued Topic 842 requiring entities to recognize assets and liabilities on the balance sheet for all leases, with certain exceptions. In addition, Topic 842 enables users of financial statements to further understand the amount, timing and uncertainty of cash flows arising from leases. Topic 842 allowed for a modified retrospective application and was effective as of the first quarter of 2019. Entities were allowed to apply the modified retrospective approach: (1) retrospectively to each prior reporting period presented in the financial statements with the cumulative-effect adjustment recognized at the beginning of the earliest comparative period presented; or (2) retrospectively at the beginning of the period of adoption (January 1, 2019) through a cumulative-effect adjustment. The modified retrospective approach includes a number of optional practical expedients that entities may elect to apply.	1/1/2019	We adopted Topic 842 beginning on January 1, 2019, using the modified retrospective approach with a cumulative-effect adjustment to opening retained earnings recorded at the beginning of the period of adoption. Therefore, upon adoption, we have recognized and measured leases without revising comparative period information or disclosure. We recorded an increase of $410 million (net of tax) to retained earnings on January 1, 2019 which related to deferred sale leaseback gains recognized from prior transactions. Additionally, the adoption of the standard had a significant impact in our consolidated balance sheet due to the recognition of $22.1 billion of operating lease liabilities, along with $23.2 billion of operating lease right-of-use-assets.

The cumulative after-tax effect of the changes made to our consolidated balance sheet for the adoption of Topic 842 were as follows:

(dollars in millions)	At December 31, 2018	Adjustments due to Topic 842	At January 1, 2019
Prepaid expenses and other	$ 5,453	$ (329)	$ 5,124
Operating lease right-of-use assets	—	23,241	23,241
Other assets	11,717	(2,048)	9,669
Accounts payable and accrued liabilities	22,501	(3)	22,498
Other current liabilities	8,239	(2)	8,237
Current operating lease liabilities	—	2,931	2,931
Deferred income taxes	33,795	139	33,934
Non-current operating lease liabilities	—	19,203	19,203
Other liabilities	13,922	(1,815)	12,107
Retained earnings	43,542	410	43,952
Noncontrolling interests	1,565	1	1,566

In addition to the increase to the operating lease liabilities and right-of-use assets and the derecognition of deferred sale leaseback gains through opening retained earnings, Topic 842 also resulted in reclassifying the presentation of prepaid and deferred rent to operating lease right-of-use assets. The operating lease right-of-use assets amount also includes the balance of any prepaid lease payments, unamortized initial direct costs and lease incentives.

We elected the package of practical expedients permitted under the transition guidance within the new standard. Accordingly, we have adopted these practical expedients and did not reassess: (1) whether an expired or existing contract is a lease or contains an embedded lease; (2) lease classification of an expired or existing lease; or (3) capitalization of initial direct costs for an expired or existing lease. In addition, we have elected the land easement transition practical expedient, and did not reassess whether an existing or expired land easement is a lease or contains a lease if it has not historically been accounted for as a lease.

We lease network equipment including towers, distributed antenna systems, small cells, real estate, connectivity mediums which include dark fiber, equipment, and other various types of assets for use in our operations under both operating and finance leases. We assess whether an arrangement is a lease or contains a lease at inception. For arrangements considered leases or that contain a lease that is accounted for separately, we determine the classification and initial measurement of the right-of-use asset and lease liability at the lease commencement date, which is the date that the underlying asset becomes available for use.

For both operating and finance leases, we recognize a right-of-use asset, which represents our right to use the underlying asset for the lease term, and a lease liability, which represents the present value of our obligation to make payments arising over the lease term. The present value of the lease payments is calculated using the incremental borrowing rate for operating and finance leases. The incremental borrowing rate is determined using a portfolio approach based on the rate of interest that the Company would have to pay to borrow an amount equal to the lease payments on a collateralized basis over a similar term. Management uses the unsecured borrowing rate and risk-adjusts that rate to approximate a collateralized rate, which is updated on a quarterly basis.

In those circumstances where the Company is the lessee, we have elected to account for non-lease components associated with our leases (e.g., common area maintenance costs) and lease components as a single lease component for substantially all of our asset classes. Additionally, in arrangements where we are the lessor, we have customer premise equipment for which we apply the lease and non-lease component practical expedient and account for non-lease components (e.g., service revenue) and lease components as combined components under the revenue recognition guidance in Topic 606 as the service revenues are the predominant components in the arrangements.

Rent expense for operating leases is recognized on a straight-line basis over the term of the lease and is included in either Cost of services or Selling, general and administrative expense in our consolidated statements of income, based on the use of the facility or equipment on which rent is being paid. Variable rent payments related to both operating and finance leases are expensed in the period incurred. Our variable lease payments consist of payments dependent on various external indicators, including real estate taxes, common area maintenance charges and utility usage.

Operating leases with a term of 12 months or less are not recorded on the balance sheet; we recognize rent expense for these leases on a straight-line basis over the lease term.

We recognize the amortization of the right-of-use asset for our finance leases on a straight-line basis over the shorter of the lease term or the useful life of the right-of-use asset in Depreciation and amortization expense in our consolidated statements of income. The interest expense related to finance leases is recognized using the effective interest method based on the discount rate determined at lease commencement and is included within Interest expense in our consolidated statements of income.

See Note 6 for additional information related to leases, including disclosure required under Topic 842.

Opening Equity Balance Sheet Adjustments from Accounting Standards Adopted in 2018

On January 1, 2018, we adopted Topic 606, ASU 2018-02, Income Statement-Reporting Comprehensive Income and other ASUs. We adopted Topic 606 using the modified retrospective method. We early adopted ASU 2018-02, which allows a reclassification from accumulated other comprehensive income to retained earnings for stranded tax effects resulting from Tax Cuts and Jobs Act (TCJA). The cumulative after-tax effect of the changes made to our consolidated balance sheet for the adoption of Topic 606, ASU 2018-02 and other ASUs was as follows:

		Adjustments due to			
(dollars in millions)	At December 31, 2017	Topic 606	ASU 2018-02	Other ASUs	At January 1, 2018
Retained earnings	35,635	2,890	(652)	(6)	37,867
Accumulated other comprehensive income	2,659	—	652	(22)	3,289
Noncontrolling interests	1,591	44	—	—	1,635

Recently Issued Accounting Standards

The following ASUs have been recently issued by the FASB.

Description	Date of Adoption	Effect on Financial Statements
ASU 2016-13, ASU 2018-19, ASU 2019-04, ASU 2019-05, Financial Instruments - Credit Losses (Topic 326)		
In June 2016, the FASB issued this standard update which requires certain financial assets be measured at amortized cost net of an allowance for estimated credit losses such that the net receivable represents the present value of expected cash collection. In addition, this standard update requires that certain financial assets be measured at amortized cost reflecting an allowance for estimated credit losses expected to occur over the life of the assets. The estimate of credit losses must be based on all relevant information including historical information, current conditions and reasonable and supportable forecasts that affect the collectability of the amounts. An entity will apply the update through a cumulative effect adjustment to retained earnings as of the beginning of the first reporting period in which the guidance is effective (January 1, 2020). A prospective transition approach is required for debt securities for which an other-than-temporary impairment has been recognized before the effective date. Early adoption of this standard is permitted.	1/1/2020	We established a cross-functional coordinated team to implement the standard update. We have completed our assessment of the expected impacts and updated our processes to meet the standards reporting and disclosure requirements. Upon adoption of this standard on January 1, 2020, we expect the cumulative effect of initially applying the new standard to result in a decrease to the opening balance of retained earnings ranging from approximately $200 million to $300 million on a pre-tax basis ($150 million to $225 million net of tax), primarily related to the expected impact on certain device payment plan agreement receivables. We do not expect our operating results to be significantly impacted by this standard update.

Note 2. Revenue and Contract Costs

We earn revenue from contracts with customers, primarily through the provision of telecommunications and other services and through the sale of wireless equipment. These services include a variety of communication and connectivity services for our Consumer and Business customers including other carriers that use our facilities to provide services to their customers, as well as professional and integrated managed services for our large enterprises and government customers. We account for these revenues under Topic 606, which we adopted on January 1, 2018, using the modified retrospective approach. We also earn revenues that are not accounted for under Topic 606 from leasing arrangements (such as those for towers and equipment), captive reinsurance arrangements primarily related to wireless device insurance and the interest on equipment financed under a device payment plan agreement when sold to the customer by an authorized agent.

We applied the new revenue recognition standard to customer contracts not completed at the date of initial adoption. For incomplete contracts that were modified before the date of adoption, the Company elected to use the practical expedient available under the modified retrospective method, which allows us to aggregate the effect of all modifications when identifying satisfied and unsatisfied performance obligations, determining the transaction price and allocating transaction price to the satisfied and unsatisfied performance obligations for the modified contract at transition. Results for reporting periods beginning after January 1, 2018 are presented under Topic 606, while amounts reported for prior periods have not been adjusted and continue to be reported under accounting standards in effect for those periods.

Prior to the adoption of Topic 606, we were required to limit the revenue recognized when a wireless device was sold to the amount of consideration that was not contingent on the provision of future services, which was typically limited to the amount of consideration received from the customer at the time of sale. Under Topic 606, the total consideration in the contract is allocated between wireless equipment and service based on their relative standalone selling prices. This change primarily impacts our arrangements that include sales of wireless devices at subsidized prices in conjunction with a fixed-term plan, also known as the subsidy model, for service. Accordingly, under Topic 606, generally more equipment revenue is recognized upon sale of the equipment to the customer and less service revenue is recognized over the contract term than was previously recognized under the prior "Revenue Recognition" (Topic 605) standard. At the time the equipment is sold, this allocation results in the recognition

of a contract asset equal to the difference between the amount of revenue recognized and the amount of consideration received from the customer. As of January 2017, we no longer offer Consumer customers new fixed-term plans with subsidized equipment pricing; however, we continue to offer fixed-term plans to our Business customers. At December 31, 2019 and December 31, 2018, approximately 12% and 14% of retail postpaid connections were under fixed-term plans, respectively.

Topic 606 also requires the deferral of incremental costs incurred to obtain a customer contract, which are then amortized to expense, as a component of Selling, general and administrative expense, over the respective periods of expected benefit. As a result, a significant amount of our sales commission costs, which were historically expensed as incurred under our previous accounting, relating to our contracts to provide wireless and wireline services, are now deferred and amortized under Topic 606.

Finally, under Topic 605, at the time of the sale of a device, we imputed risk adjusted interest on the device payment plan agreement receivables. We recorded the imputed interest as a reduction to the related accounts receivable and interest income was recognized over the financed device payment term. Under Topic 606, while there continues to be a financing component in both the fixed-term plans and device payment plans, also known as the installment model, we have determined that this financing component for our customer classes in the direct channels for wireless devices are not significant and therefore we no longer impute interest for these contracts. This change results in additional revenue recognized upon the sale of wireless devices and no interest income recognized over the device payment term.

A reconciliation of the adjustments from the adoption of Topic 606 relative to Topic 605 on certain impacted financial statement line items in our consolidated statements of income is as follows:

	Year Ended December 31, 2018		
(dollars in millions)	As reported	Balances without adoption of Topic 606	Adjustments
Operating Revenues			
Service revenues and other	$ 108,605	$ 109,964	$ (1,359)
Wireless equipment revenues	22,258	20,474	1,784
Total Operating Revenues	130,863	130,438	425
Cost of services (exclusive of items shown below)	32,185	32,240	(55)
Cost of wireless equipment	23,323	23,189	134
Selling, general and administrative expense	31,083	32,588	(1,505)
Equity in losses of unconsolidated businesses	(186)	(187)	1
Income Before Provision For Income Taxes	19,623	17,771	1,852
Provision for income taxes	(3,584)	(3,104)	(480)
Net Income	$ 16,039	$ 14,667	$ 1,372
Net income attributable to noncontrolling interests	$ 511	$ 481	$ 30
Net income attributable to Verizon	15,528	14,186	1,342
Net Income	$ 16,039	$ 14,667	$ 1,372

Revenue by Category

We have two reportable segments that we operate and manage as strategic business units, Consumer and Business. Revenue is disaggregated by products and services within Consumer, and customer groups (Global Enterprise, Small and Medium Business, Public Sector and Other, and Wholesale) within Business. See Note 13 for additional information on revenue by segment.

Corporate and other includes the results of our media business, Verizon Media, and other businesses. Verizon Media generated revenues from contracts with customers under Topic 606 of approximately $7.5 billion and $7.7 billion for the years ended December 31, 2019 and 2018, respectively.

We also earn revenues that are not accounted for under Topic 606 from leasing arrangements (such as those for towers and equipment), captive reinsurance arrangements primarily related to wireless device insurance and the interest on equipment financed under a device payment plan agreement when sold to the customer by an authorized agent. As allowed by the practical expedient within Topic 842, we have elected to combine the lease and non-lease components for those arrangements of customer premise equipment where we are the lessor as components accounted for under Topic 606. Revenues from arrangements that were not accounted for under Topic 606 were approximately $3.1 billion and $4.5 billion for the years ended December 31, 2019 and 2018, respectively.

Remaining Performance Obligations

When allocating the total contract transaction price to identified performance obligations, a portion of the total transaction price may relate to service performance obligations which were not satisfied or are partially satisfied as of the end of the reporting period. Below we disclose

information relating to these unsatisfied performance obligations. Upon adoption, we elected to apply the practical expedient available under Topic 606 that provides the option to exclude the expected revenues arising from unsatisfied performance obligations related to contracts that have an original expected duration of one year or less. This situation primarily arises with respect to certain month-to-month service contracts. At December 31, 2019, month-to-month service contracts represented approximately 88% of our wireless postpaid contracts and 61% of our wireline Consumer and Small and Medium Business contracts, compared to December 31, 2018, for which month-to-month service contracts represented approximately 86% of our wireless postpaid contracts and 56% of our wireline Consumer and Small and Medium Business contracts.

Additionally, certain contracts provide customers the option to purchase additional services. The fees related to these additional services are recognized when the customer exercises the option (typically on a month-to-month basis).

Contracts for wireless services are generally either month-to-month and cancellable at any time (typically under a device payment plan) or contain terms ranging from greater than one month to up to two years (typically under a fixed-term plan). Additionally, customers may incur charges based on usage or additional optional services purchased in conjunction with entering into a contract that can be cancelled at any time and therefore are not included in the transaction price. The transaction price allocated to service performance obligations, which are not satisfied or are partially satisfied as of the end of the reporting period, are generally related to our fixed-term plans.

Our Consumer group customers also include traditional wholesale resellers that purchase and resell wireless service under their own brands to their respective customers. Reseller arrangements generally include a stated contract term, which typically extends longer than two years and, in some cases, include a periodic minimum revenue commitment over the contract term for which revenues will be recognized in future periods.

Consumer customer contracts for wireline services generally have a service term of two years; however, this term may be shorter than twelve months or may be month-to-month. Certain contracts with Business customers for wireline services extend into future periods, contain fixed monthly fees and usage-based fees, and can include annual commitments in each year of the contract or commitments over the entire specified contract term; however, a significant number of contracts for wireline services with our Business customers have a contract term that is twelve months or less.

Additionally, there are certain contracts with Business customers for wireline and telematics services and certain Media contracts with customers that have a contractual minimum fee over the total contract term. We cannot predict the time period when revenue will be recognized related to those contracts; thus, they are excluded from the time bands below. These contracts have varying terms spanning over approximately five years ending in November 2024 and have aggregate contract minimum payments totaling $3.4 billion.

At December 31, 2019, the transaction price related to unsatisfied performance obligations for total Verizon that is expected to be recognized for 2020, 2021 and thereafter was $20.2 billion, $9.4 billion and $1.6 billion, respectively. Remaining performance obligation estimates are subject to change and are affected by several factors, including terminations and changes in the timing and scope of contracts, arising from contract modifications.

Accounts Receivable and Contract Balances

The timing of revenue recognition may differ from the time of billing to our customers. Receivables presented in our consolidated balance sheet represent an unconditional right to consideration. Contract balances represent amounts from an arrangement when either Verizon has performed, by transferring goods or services to the customer in advance of receiving all or partial consideration for such goods and services from the customer, or the customer has made payment to Verizon in advance of obtaining control of the goods and/or services promised to the customer in the contract.

The following table presents information about receivables from contracts with customers:

(dollars in millions)	**At December 31, 2019**	At December 31, 2018	At January 1, 2018
Receivables[1]	**$ 12,078**	$ 12,104	$ 12,073
Device payment plan agreement receivables[2]	**11,741**	8,940	1,461

[1] Balances do not include receivables related to the following contracts: leasing arrangements (such as those for towers and equipment), captive reinsurance arrangements primarily related to wireless device insurance and the interest on equipment financed under a device payment plan agreement when sold to the customer by an authorized agent.

[2] Included in device payment plan agreement receivables presented in Note 8. Balances do not include receivables related to contracts completed prior to January 1, 2018 and receivables derived from the sale of equipment on a device payment plan through an authorized agent.

The following table presents information about contract balances:

(dollars in millions)	At December 31, 2019	At December 31, 2018	At January 1, 2018
Contract asset	$ 1,150	$ 1,003	$ 1,170
Contract liability	5,307	4,943	4,452

Contract assets primarily relate to our rights to consideration for goods or services provided to customers but for which we do not have an unconditional right at the reporting date. Under a fixed-term plan, total contract revenue is allocated between wireless service and equipment revenues, as discussed above. In conjunction with these arrangements, a contract asset is created, which represents the difference between the amount of equipment revenue recognized upon sale and the amount of consideration received from the customer when the performance obligation related to the transfer of control of the equipment is satisfied. The contract asset is reclassified to accounts receivable as wireless services are provided and billed. We have the right to bill the customer as service is provided over time, which results in our right to the payment being unconditional. The contract asset balances are presented in our consolidated balance sheet as Prepaid expenses and other and Other assets. We assess our contract assets for impairment on a quarterly basis and will recognize an impairment charge to the extent their carrying amount is not recoverable.

Contract assets increased $147 million during the year ended December 31, 2019. The change in the contract asset balance was primarily due to new contracts and increases in sales promotions recognized upfront, driven by customer activity related to wireless and Fios services, partially offset by reclassifications to accounts receivable due to billings on existing contracts and impairment charges of $113 million. Contract assets decreased $167 million during the year ended December 31, 2018. The change in the contract asset balance was primarily due to reclassifications to accounts receivable due to billings on existing contracts and impairment charges of $116 million, offset by new contracts related to wireless and Fios services.

Contract liabilities arise when we bill our customers and receive consideration in advance of providing the goods or services promised in the contract. We typically bill service one month in advance, which is the primary component of the contract liability balance. Contract liabilities are recognized as revenue when services are provided to the customer. The contract liability balances are presented in our consolidated balance sheet as Other current liabilities and Other liabilities.

Contract liabilities increased $364 million during the year ended December 31, 2019. The change in contract liabilities was primarily due to increases in sales promotions recognized over time and upfront fees, as well as increases in deferred revenue related to advanced billings, partially offset by the satisfaction of performance obligations related to wireless and Fios services. Contract liabilities increased $491 million during the year ended December 31, 2018. The change in contract liabilities was primarily due to increases in sales promotions, as well as increases in deferred revenue related to advanced billings, partially offset by the satisfaction of performance obligations related to wireless and Fios services.

Revenue recognized during the years ended December 31, 2019 and 2018 related to contract liabilities existing at January 1, 2019 and 2018 were $4.2 billion and $3.9 billion, respectively, as performance obligations related to services were satisfied.

The balance of contract assets and contract liabilities recorded in our consolidated balance sheets were as follows:

(dollars in millions)	At December 31, 2019	At December 31, 2018
Assets		
Prepaid expenses and other	$ 848	$ 757
Other assets	302	246
Total	$ 1,150	$ 1,003
Liabilities		
Other current liabilities	$ 4,651	$ 4,207
Other liabilities	656	736
Total	$ 5,307	$ 4,943

Contract Costs

As discussed in Note 1, Topic 606 requires the recognition of an asset for incremental costs to obtain a customer contract, which is then amortized to expense over the respective period of expected benefit. We recognize an asset for incremental commission costs paid to internal and external sales personnel and agents in conjunction with obtaining customer contracts. We only defer these costs when we have determined the commissions are incremental costs that would not have been incurred absent the customer contract and are expected to be recoverable. Costs to obtain a contract are amortized and recorded ratably as commission expense over the period representing the transfer of goods or services to which the assets relate. Costs to obtain wireless contracts are amortized over both of our Consumer and Business customers' estimated device upgrade cycles, as such costs are typically incurred each time a customer upgrades. Costs to obtain wireline contracts are amortized as expense over the estimated customer relationship period for our Consumer customers. Incremental costs to obtain wireline contracts for our Business customers are insignificant. Costs to obtain contracts are recorded in Selling, general and administrative expense.

We also defer costs incurred to fulfill contracts that: (1) relate directly to the contract; (2) are expected to generate resources that will be used to satisfy our performance obligation under the contract; and (3) are expected to be recovered through revenue generated under the contract. Contract fulfillment costs are expensed as we satisfy our performance obligations and recorded to Cost of services. These costs principally relate to direct costs that enhance our wireline business resources, such as costs incurred to install circuits.

We determine the amortization periods for our costs incurred to obtain or fulfill a customer contract at a portfolio level due to the similarities within these customer contract portfolios.

Other costs, such as general costs or costs related to past performance obligations, are expensed as incurred.

Collectively, costs to obtain a contract and costs to fulfill a contract are referred to as deferred contract costs, and amortized over a 2 to 5-year period. Deferred contract costs are classified as current or non-current within Prepaid expenses and other and Other assets, respectively.

The balances of deferred contract costs included in our consolidated balance sheets were as follows:

(dollars in millions)		**At December 31, 2019**		At December 31, 2018
Assets				
Prepaid expenses and other	**$**	**2,578**	$	2,083
Other assets		**1,911**		1,812
Total	**$**	**4,489**	$	3,895

For the years ended December 31, 2019 and 2018, we recognized expense of $2.7 billion and $2.0 billion, respectively, associated with the amortization of deferred contract costs, primarily within Selling, general and administrative expense in our consolidated statements of income.

We assess our deferred contract costs for impairment on a quarterly basis. We recognize an impairment charge to the extent the carrying amount of a deferred cost exceeds the remaining amount of consideration we expect to receive in exchange for the goods and services related to the cost, less the expected costs related directly to providing those goods and services that have not yet been recognized as expenses. There have been no impairment charges recognized for the years ended December 31, 2019 and 2018.

Note 3. Acquisitions and Divestitures

Spectrum License Transactions

Since 2017, we have entered into or completed several strategic spectrum transactions including:

- During the fourth quarter of 2016, we entered into a license exchange agreement with affiliates of AT&T Inc. (AT&T) to exchange certain Advanced Wireless Services (AWS) and Personal Communication Services (PCS) spectrum licenses. This non-cash exchange was completed in February 2017. As a result, we received $1.0 billion of AWS and PCS spectrum licenses at fair value and recorded a pre-tax gain of $126 million in Selling, general and administrative expense in our consolidated statement of income for the year ended December 31, 2017.

- During the first quarter of 2017, we entered into a license exchange agreement with affiliates of Sprint Corporation to exchange certain PCS spectrum licenses. This non-cash exchange was completed in May 2017. As a result, we received $132 million of PCS spectrum licenses at fair value and recorded an insignificant gain in Selling, general and administrative expense in our consolidated statement of income for the year ended December 31, 2017.

- During the third quarter of 2017, we entered into a license exchange agreement with affiliates of T-Mobile USA Inc. to exchange certain AWS and PCS spectrum licenses. This non-cash exchange was completed in December 2017. As a result, we received $414 million of AWS and PCS spectrum licenses at fair value and recorded a pre-tax gain of $143 million in Selling, general and administrative expense in our consolidated statement of income for the year ended December 31, 2017.

- During 2018, we entered into and completed various wireless license transactions, including the purchase of Straight Path Communications Inc. (Straight Path) and NextLink Wireless LLC (NextLink).

- During 2019, the FCC completed two millimeter wave spectrum license auctions. Verizon participated in these auctions and was the high bidder on 9 and 1,066 licenses, respectively, in the 24 Gigahertz (GHz) and 28 GHz bands. We submitted an application to the FCC and paid cash of approximately $521 million for the licenses. We received the licenses during the fourth quarter of 2019.

- During 2019, we entered into and completed various other wireless license acquisitions for an insignificant amount of cash consideration.

In December 2019, the FCC incentive auction for spectrum licenses in the upper 37 GHz, 39 GHz, and 47 GHz bands commenced. As an incumbent licensee, Verizon received vouchers related to our existing 39 GHz licenses. These vouchers can be converted into cash, the amount of which will not be known until the conclusion of the auction, or applied toward the purchase price of spectrum in the auction. At the conclusion

of the auction, all existing licenses will be cancelled and new reconfigured licenses or cash will be distributed depending on the results of the auction. Due to the FCC's rules restricting communications regarding the auction, we will not disclose our financial plans for the auction during the quiet period for this auction unless legally required. In addition, as of this time, until the completion of the auction process, we cannot determine the resulting financial outcome, including a potential gain or loss. Such gain or loss, if any, may be material.

Acquisition of AOL Inc.

In May 2015, we entered into an Agreement and Plan of Merger with AOL Inc. (AOL) pursuant to which we commenced a tender offer to acquire all of the outstanding shares of common stock of AOL at a price of $50.00 per share, net to the seller in cash, without interest and less any applicable withholding taxes.

On June 23, 2015, we completed the tender offer and merger, and AOL became a wholly-owned subsidiary of Verizon. The aggregate cash consideration paid by Verizon at the closing of these transactions was approximately $3.8 billion. Holders of approximately 6.6 million shares exercised appraisal rights under Delaware law. In September 2018, we obtained court approval to settle this matter for total cash consideration of $219 million of which an insignificant amount relates to interest, resulting in an insignificant gain. We paid the cash consideration in October 2018.

XO Holdings

In February 2016, we entered into a purchase agreement to acquire XO Holdings' wireline business (XO), which owned and operated one of the largest fiber-based Internet Protocol and Ethernet networks in the U.S. Concurrently, we entered into a separate agreement to utilize certain wireless spectrum from a wholly-owned subsidiary of XO Holdings, NextLink, that held XO's millimeter-wave wireless spectrum. The agreement included an option, subject to certain conditions, to acquire NextLink. In February 2017, we completed our acquisition of XO for total cash consideration of approximately $1.5 billion, of which $100 million was paid in 2015, and we prepaid $320 million in connection with the NextLink option which represented the fair value of the option.

In April 2017, we exercised our option to buy NextLink for approximately $493 million, subject to certain adjustments, of which $320 million was prepaid in the first quarter of 2017. The transaction closed in January 2018. The acquisition of NextLink was accounted for as an asset acquisition, as substantially all of the value related to the acquired spectrum. Upon closing, we recorded approximately $657 million of wireless licenses, $110 million of a deferred tax liability and $58 million of other liabilities.

The consolidated financial statements include the results of XO's operations from the date the acquisition closed. If the acquisition of XO had been completed as of January 1, 2016, the results of operations of Verizon would not have been significantly different than our previously reported results of operations.

The acquisition of XO was accounted for as a business combination. The consideration was allocated to the assets acquired and liabilities assumed based on their fair values as of the close of the acquisition. We recorded approximately $1.2 billion of property, plant and equipment, $120 million of goodwill and $194 million of other intangible assets. Goodwill is calculated as the difference between the acquisition date fair value of the consideration transferred and the fair value of the net assets acquired. The goodwill represents future economic benefits that we expect to achieve as a result of the acquisition.

Acquisition of Yahoo! Inc.'s Operating Business

In July 2016, Verizon entered into a stock purchase agreement (the Purchase Agreement) with Yahoo! Inc. (Yahoo). Pursuant to the Purchase Agreement, upon the terms and subject to the conditions thereof, we agreed to acquire the stock of one or more subsidiaries of Yahoo holding all of Yahoo's operating business for approximately $4.83 billion in cash, subject to certain adjustments (the Transaction).

In February 2017, Verizon and Yahoo entered into an amendment to the Purchase Agreement, pursuant to which the Transaction purchase price was reduced by $350 million to approximately $4.48 billion in cash, subject to certain adjustments. Subject to certain exceptions, the parties also agreed that certain user security and data breaches incurred by Yahoo (and the losses arising therefrom) were to be disregarded: (1) for purposes of specified conditions to Verizon's obligations to close the Transaction; and (2) in determining whether a "Business Material Adverse Effect" under the Purchase Agreement had occurred.

Concurrently with the amendment of the Purchase Agreement, Yahoo and Yahoo Holdings, Inc., a wholly-owned subsidiary of Yahoo that Verizon agreed to purchase pursuant to the Transaction, also entered into an amendment to the related reorganization agreement, pursuant to which Yahoo (which changed its name to Altaba Inc. following the closing of the Transaction) retains 50% of certain post-closing liabilities arising out of governmental or third-party investigations, litigations or other claims related to certain user security and data breaches incurred by Yahoo prior to its acquisition by Verizon, including an August 2013 data breach disclosed by Yahoo on December 14, 2016. At that time, Yahoo disclosed that more than one billion of the approximately three billion accounts existing in 2013 had likely been affected. In accordance with the original Transaction agreements, Yahoo will continue to retain 100% of any liabilities arising out of any shareholder lawsuits (including derivative claims) and investigations and actions by the SEC.

In June 2017, we completed the Transaction. The aggregate purchase consideration at the closing of the Transaction was approximately $4.7 billion, including cash acquired of $230 million.

Prior to the closing of the Transaction, pursuant to a related reorganization agreement, Yahoo transferred all of the assets and liabilities constituting Yahoo's operating business to the subsidiaries that we acquired in the Transaction. The assets that we acquired did not include Yahoo's ownership interests in Alibaba, Yahoo! Japan and certain other investments, certain undeveloped land recently divested by Yahoo, certain non-core intellectual property or its cash, other than the cash from its operating business we acquired. We received for our benefit and that of our current and certain future affiliates a non-exclusive, worldwide, perpetual, royalty-free license to all of Yahoo's intellectual property that was not conveyed with the business.

In October 2017, based upon information that we received in connection with our integration of Yahoo's operating business, we disclosed that we believe that the August 2013 data breach previously disclosed by Yahoo affected all of its accounts.

The acquisition of Yahoo's operating business has been accounted for as a business combination. The fair values of the assets acquired and liabilities assumed were determined using the income, cost, market and multiple period excess earnings approaches. The fair value measurements were primarily based on significant inputs that are not observable in the market and thus represent a Level 3 measurement as defined in Accounting Standards Codification 820, Fair Value Measurements and Disclosures, other than long-term debt assumed in the acquisition. The income approach was primarily used to value the intangible assets, consisting primarily of acquired technology and customer relationships. The income approach indicates value for an asset based on the present value of cash flow projected to be generated by the asset. Projected cash flow is discounted at a required rate of return that reflects the relative risk of achieving the cash flow and the time value of money. The cost approach, which estimates value by determining the current cost of replacing an asset with another of equivalent economic utility, was used, as appropriate, for property, plant and equipment. The cost to replace a given asset reflects the estimated reproduction or replacement cost for the property, less an allowance for loss in value due to depreciation.

In June 2018, we finalized the accounting for the Yahoo acquisition. The following table summarizes the final accounting for the assets acquired, including cash acquired of $230 million, and liabilities assumed as of the close of the acquisition, as well as the fair value at the acquisition date of Yahoo's noncontrolling interests:

(dollars in millions)	As of December 31, 2017	Measurement-period adjustments [1]	Adjusted Fair Value
Cash payment to Yahoo's equity holders	$ 4,673	$ —	$ 4,673
Estimated liabilities to be paid	38	—	38
Total consideration	$ 4,711	$ —	$ 4,711
Assets acquired:			
Goodwill	$ 1,929	$ 215	$ 2,144
Intangible assets subject to amortization	1,873	1	1,874
Property, plant, and equipment	1,805	(6)	1,799
Other	1,332	128	1,460
Total assets acquired	6,939	338	7,277
Liabilities assumed:			
Total liabilities assumed	2,178	338	2,516
Net assets acquired:	4,761	—	4,761
Noncontrolling interest	(50)	—	(50)
Total consideration	$ 4,711	$ —	$ 4,711

[1] Adjustments to the fair value measurements to reflect new information obtained about facts and circumstances that existed as of the acquisition date that, if known, would have affected the measurement of the amounts recognized as of that date. The most significant adjustments related to an increase in goodwill and the recognition of liabilities per certain pre-acquisition contingencies.

On the closing date of the Transaction, each unvested and outstanding Yahoo restricted stock unit award that was held by an employee who became an employee of Verizon was replaced with a Verizon restricted stock unit award, which is generally payable in cash upon the applicable vesting date. The value of those outstanding restricted stock units on the acquisition date was approximately $1.0 billion.

Goodwill is calculated as the difference between the acquisition date fair value of the consideration transferred and the fair value of the net assets acquired. The goodwill was primarily attributable to increased synergies that were expected to be achieved from the integration of Yahoo's operating business into our Media business. The goodwill related to this acquisition is included within Corporate and other.

The consolidated financial statements include the results of Yahoo's operating business from the date the acquisition closed. If the acquisition of Yahoo's operating business had been completed as of January 1, 2016, the results of operations of Verizon would not have been significantly different than our previously reported results of operations.

Acquisition and Integration Related Charges

Related to the Yahoo Transaction, we recorded $473 million of acquisition and integration related charges during the year ended December 31, 2018, of which $273 million, $195 million and an insignificant amount are related to Severance, Integration costs and Transaction costs, respectively. In connection with the Yahoo Transaction, we recorded acquisition and integration related charges of approximately $762 million during the year ended December 31, 2017, of which $526 million, $166 million and $70 million related to Severance, Integration costs and Transaction costs, respectively. These charges were recorded in Selling, general and administrative expense in our consolidated statements of income.

Data Center Sale

In December 2016, we entered into a definitive agreement, which was subsequently amended in March 2017, with Equinix, Inc. (Equinix) pursuant to which we agreed to sell 23 customer-facing data center sites in the U.S. and Latin America for approximately $3.6 billion, subject to certain adjustments (Data Center Sale) . The transaction closed in May 2017.

For the year ended December 31, 2017, these sites generated an insignificant amount of revenues and earnings.

In connection with the Data Center Sale and other insignificant divestitures, we recorded a net gain on sale of divested businesses of approximately $1.8 billion in Selling, general and administrative expense in our consolidated statement of income for the year ended December 31, 2017.

Straight Path

In May 2017, we entered into a purchase agreement to acquire Straight Path, a holder of millimeter wave spectrum configured for fifth-generation (5G) wireless services, for total consideration reflecting an enterprise value of approximately $3.1 billion. Under the terms of the purchase agreement, we agreed to pay: (1) Straight Path shareholders $184.00 per share, payable in Verizon shares; and (2) certain transaction costs payable in cash of approximately $736 million, consisting primarily of a fee to be paid to the FCC. The transaction closed in February 2018 at which time we issued approximately 49 million shares of Verizon common stock, valued at approximately $2.4 billion, and paid the associated cash consideration.

The acquisition of Straight Path was accounted for as an asset acquisition, as substantially all of the value related to the acquired spectrum. Upon closing, we recorded approximately $4.5 billion of wireless licenses and $1.4 billion of a deferred tax liability. The spectrum acquired as part of the transaction is being used for our 5G technology deployment. See Note 4 for additional information.

WideOpenWest, Inc.

In August 2017, we entered into a definitive agreement to purchase certain fiber-optic network assets in the Chicago market from WideOpenWest, Inc. (WOW!), a leading provider of communications services. The transaction closed in December 2017. In addition, the parties entered into a separate agreement pursuant to which WOW! was to complete the build-out of the network assets in 2019. This build-out was completed in 2019. The total cash consideration for the transactions was approximately $275 million, of which $226 million was paid in December 2017. During 2019 and 2018, the remaining cash consideration was paid.

Other

In July 2019, Verizon completed a sale-leaseback transaction for buildings and real estate. See Note 6 for additional information related to the transaction. In connection with this transaction and other insignificant transactions, we recorded a pre-tax net gain from dispositions of assets and businesses of $261 million in Selling, general and administrative expense in our consolidated statement of income for the year ended December 31, 2019.

During 2019, 2018 and 2017, we completed various other acquisitions for an insignificant amount of cash consideration.

Note 4. Wireless Licenses, Goodwill and Other Intangible Assets

Wireless Licenses

The carrying amounts of Wireless licenses are as follows:

(dollars in millions)

At December 31,		**2019**		2018
Wireless licenses	**$**	**95,059**	$	94,130

At December 31, 2019 and 2018, approximately $6.2 billion and $8.6 billion, respectively, of wireless licenses were under development for commercial service for which we were capitalizing interest costs. We recorded approximately $321 million and $515 million of capitalized interest on wireless licenses for each of the years ended December 31, 2019 and 2018, respectively.

For the year ended December 31, 2018, we recorded approximately $4.5 billion of wireless licenses in connection with the Straight Path acquisition and $657 million in connection with the NextLink acquisition. See Note 3 for additional information regarding spectrum license transactions in 2019 and 2018.

The average remaining renewal period of our wireless license portfolio was 4.6 years as of December 31, 2019. See Note 1 for additional information.

As discussed in Note 1, we test our wireless licenses for potential impairment annually or more frequently if impairment indicators are present. In 2019, we performed a qualitative assessment to determine whether it was more likely than not that the fair value of our wireless licenses was less than the carrying amount. In 2018, our quantitative impairment test consisted of comparing the estimated fair value of our aggregate wireless licenses estimated using the Greenfield approach to the aggregated carrying amount of the licenses as of the test date. In 2017, we performed a qualitative assessment to determine whether it was more likely than not that the fair value of our wireless licenses was less than the carrying amount. Our assessments in 2019, 2018 and 2017 indicated that the fair value of our wireless licenses exceeded the carrying value and, therefore, did not result in impairment.

Goodwill

The Company transitioned into our new reporting structure as of April 1, 2019, which resulted in certain changes to our operating segments and reporting units. Upon the date of reorganization, the goodwill of our historical Wireless reporting unit, historical Wireline reporting unit and historical Verizon Connect reporting unit were reallocated to our new Consumer and Business reporting units using a relative fair value approach.

Changes in the carrying amount of Goodwill are as follows:

(dollars in millions)

	Consumer	Business	Wireless	Wireline	Other [2]	Total
Balance at January 1, 2018	$ —	$ —	$ 18,397	$ 3,955	$ 6,820	$ 29,172
Acquisitions (Note 3)	—	—	—	(77)	225	148
Reclassifications, adjustments and other	—	—	—	(7)	(108)	(115)
Media goodwill impairment	—	—	—	—	(4,591)	(4,591)
Balance at December 31, 2018	—	—	18,397	3,871	2,346	24,614
Acquisitions	—	—	—	**20**	—	**20**
Reclassifications, adjustments and other	—	—	—	**1**	—	**1**
Balance at March 31, 2019	—	—	**18,397**	**3,892**	**2,346**	**24,635**
Reporting Unit reallocation [1]	**17,104**	**7,269**	**(18,397)**	**(3,892)**	**(2,084)**	—
Balance at April 1, 2019	**17,104**	**7,269**	—	—	**262**	**24,635**
Acquisitions	—	**2**	—	—	—	**2**
Media goodwill impairment	—	—	—	—	**(186)**	**(186)**
Reclassifications, adjustments and other	—	**(2)**	—	—	**(60)**	**(62)**
Balance at December 31, 2019	**$ 17,104**	**$ 7,269**	**$ —**	**$ —**	**$ 16**	**$ 24,389**

[1] Represents the reallocation of goodwill as a result of the Company reorganizing its segments as described in Note 1.
[2] Goodwill is net of accumulated impairment charges of $4.6 billion as of December 31, 2018 and $4.8 billion as of December 31, 2019, related to our Media reporting unit.

We performed impairment assessments of the impacted reporting units, specifically our historical Wireless, historical Wireline and historical Connect reporting units on March 31, 2019, immediately before our strategic reorganization became effective. Our impairment assessments indicated that the fair value for each of our historical Wireless, historical Wireline and historical Connect reporting units exceeded their respective carrying values, and therefore did not result in a goodwill impairment. We then performed quantitative assessments of our Consumer and Business reporting units on April 1, 2019, immediately following our strategic reorganization. Our impairment assessments indicated that the fair value for each of our Consumer and Business reporting units exceeded their respective carrying values and therefore, did not result in a goodwill impairment. Our Media reporting unit was not impacted by the strategic reorganization and there was no indicator of impairment as of the reorganization date.

We performed qualitative impairment assessments for our Consumer and Business reporting units during the fourth quarter of 2019. Our qualitative assessments indicated that it was more likely than not that the fair values for our Consumer and Business reporting units exceeded their respective carrying values and, therefore, did not result in an impairment. We performed quantitative impairment assessments for our Media reporting unit in 2019 and 2018. For details on our Media reporting unit, refer to the discussion below.

Our Media business, Verizon Media, experienced increased competitive and market pressures throughout 2018 that resulted in lower than expected revenues and earnings. These pressures were expected to continue and have resulted in a loss of market positioning to our competitors in the digital advertising business. Our Media business also achieved lower than expected benefits from the integration of the Yahoo and AOL businesses.

In connection with Verizon's annual budget process during the fourth quarter of 2019 and 2018, the leadership at both Verizon Media and Verizon completed a comprehensive five-year strategic planning review of Verizon Media's business prospects resulting in unfavorable adjustments to Verizon Media's financial projections. These revised projections were used as a key input into Verizon Media's annual goodwill impairment tests performed in the fourth quarter of 2019 and 2018.

During the fourth quarter of 2019 and 2018, consistent with our accounting policy, we applied a combination of a market approach and a discounted cash flow method reflecting current assumptions and inputs, including our revised projections, discount rate and expected growth rates, which resulted in the determination that the fair value of the Media reporting unit was less than its carrying amount. As a result, we recorded a non-cash goodwill impairment charge of approximately $186 million ($176 million after-tax) in the fourth quarter of 2019 and a charge of $4.6 billion ($4.5 billion after-tax) in the fourth quarter of 2018 in our consolidated statements of income. The goodwill balance of the Media reporting unit has been fully written off as a result of these impairment charges.

We performed a quantitative impairment assessment for all of the other reporting units in 2018. Our impairment tests indicated that the fair value for each of our historical Wireless, historical Wireline and historical Connect reporting units exceeded their respective carrying value and, therefore, did not result in an impairment.

For 2017, we performed a quantitative impairment assessment for all of our reporting units, except for our historical Wireless reporting unit, for which a qualitative assessment was completed. For 2017, our impairment tests indicated that the fair value for each of our reporting units exceeded their respective carrying value and therefore, did not result in goodwill impairment.

Other Intangible Assets

The following table displays the composition of Other intangible assets, net as well as the respective amortization period:

(dollars in millions)

			2019					2018
At December 31,	**Gross Amount**	**Accumulated Amortization**	**Net Amount**	Gross Amount	Accumulated Amortization	Net Amount
Customer lists (8 to 13 years)	**$ 3,896**	**$ (1,511)**	**$ 2,385**	$ 3,951	$ (1,121)	$ 2,830
Non-network internal-use software (3 to 7 years)	**20,530**	**(14,418)**	**6,112**	18,603	(12,785)	5,818
Other (2 to 25 years)	**1,967**	**(966)**	**1,001**	1,988	(861)	1,127
Total	**$ 26,393**	**$ (16,895)**	**$ 9,498**	$ 24,542	$ (14,767)	$ 9,775

The amortization expense for Other intangible assets was as follows:

Years	(dollars in millions)
2019	**$ 2,311**
2018	2,217
2017	2,213

Estimated annual amortization expense for Other intangible assets is as follows:

Years	(dollars in millions)
2020	**$ 2,235**
2021	**1,931**
2022	**1,651**
2023	**1,317**
2024	**968**

Note 5. Property, Plant and Equipment

The following table displays the details of Property, plant and equipment, which is stated at cost:

(dollars in millions)

At December 31,	Lives (years)	2019	2018
Land	-	**$ 594**	$ 807
Buildings and equipment	**7 to 45**	**31,216**	30,468
Central office and other network equipment	**3 to 50**	**152,733**	147,250
Cable, poles and conduit	**7 to 50**	**52,658**	49,859
Leasehold improvements	**5 to 20**	**9,072**	8,580
Work in progress	-	**9,234**	6,362
Furniture, vehicles and other	**3 to 20**	**10,227**	9,509
		265,734	252,835
Less accumulated depreciation		**173,819**	163,549
Property, plant and equipment, net		**$ 91,915**	$ 89,286

Note 6. Leasing Arrangements

We enter into various lease arrangements for network equipment including towers, distributed antenna systems, small cells, real estate and connectivity mediums including dark fiber, equipment, and other various types of assets for use in our operations. Our leases have remaining lease terms ranging from 1 year to 28 years, some of which include options that we can elect to extend the leases term for up to 25 years, and some of which include options to terminate the leases. For the majority of leases entered into during the current period, we have concluded it is not reasonably certain that we would exercise the options to extend the lease or terminate the lease. Therefore, as of the lease commencement date, our lease terms generally do not include these options. We include options to extend the lease when it is reasonably certain that we will exercise that option.

During March 2015, we completed a transaction with American Tower Corporation (American Tower) pursuant to which American Tower acquired the exclusive rights to lease and operate approximately 11,300 of our wireless towers for an upfront payment of $5.0 billion. We have subleased capacity on the towers from American Tower for a minimum of 10 years at current market rates in 2015, with options to renew. We continue to include the towers in Property, plant and equipment, net in our consolidated balance sheets and depreciate them accordingly. In addition to the rights to lease and operate the towers, American Tower assumed the interest in the underlying ground leases related to these towers. While American Tower can renegotiate the terms of and is responsible for paying the ground leases, we are still the primary obligor for these leases and accordingly, the present value of these ground leases are included in our operating lease right-of-use assets and operating lease liabilities. We do not expect to be required to make ground lease payments unless American Tower defaults, which we determined to be remote.

The components of net lease cost were as follows:

(dollars in millions)

Year Ended December 31,	Classification	2019
Operating lease cost [1]	Cost of services Selling, general and administrative expense	**$ 4,746**
Finance lease cost:		
Amortization of right-of-use assets	Depreciation and amortization expense	**330**
Interest on lease liabilities	Interest expense	**38**
Short-term lease cost [1]	Cost of services Selling, general and administrative expense	**40**
Variable lease cost [1]	Cost of services Selling, general and administrative expense	**218**
Sublease income	Service revenues and other	**(275)**
Total net lease cost		**$ 5,097**
Gain on sale and leaseback transaction, net	Selling, general and administrative expense	**$ (391)**

[1] All operating lease costs, including short-term and variable lease costs, are split between Cost of services and Selling, general and administrative expense in the consolidated statements of income based on the use of the facility or equipment that the rent is being paid on. See Note 1 for additional information. Variable lease costs represent payments that are dependent on a rate or index, or on usage of the asset.

Supplemental disclosure for the statement of cash flows related to operating and finance leases were as follows:

	(dollars in millions)
Year Ended December 31,	**2019**
Cash Flows from Operating Activities	
Cash paid for amounts included in the measurement of lease liabilities	
Operating cash flows for operating leases	**$ (4,392)**
Operating cash flows for finance leases	**(38)**
Cash Flows from Financing Activities	
Financing cash flows for finance leases	**(352)**
Supplemental lease cash flow disclosures	
Operating lease right-of-use assets obtained in exchange for new operating lease liabilities	**3,510**
Right-of-use assets obtained in exchange for new finance lease liabilities	**564**

Supplemental disclosures for the balance sheet related to finance leases were as follows:

	(dollars in millions)
At December 31,	**2019**
Assets	
Property, plant and equipment, net	**$ 939**
Liabilities	
Debt maturing within one year	**$ 336**
Long-term debt	**780**
Total Finance lease liabilities	**$ 1,116**

The weighted-average remaining lease term and the weighted-average discount rate of our leases were as follows:

At December 31,	**2019**
Weighted-average remaining lease term (years)	
Operating Leases	**9**
Finance Leases	**5**
Weighted-average discount rate	
Operating Leases	**4.0%**
Finance Leases	**3.2%**

The Company's maturity analysis of operating and finance lease liabilities as of December 31, 2019 were as follows:

		(dollars in millions)
Years	**Operating Leases**	**Finance Leases**
2020	**$ 4,099**	**$ 366**
2021	**3,764**	**271**
2022	**3,363**	**208**
2023	**3,001**	**152**
2024	**2,484**	**92**
Thereafter	**9,257**	**124**
Total lease payments	**25,968**	**1,213**
Less interest	**4,314**	**97**
Present value of lease liabilities	**21,654**	**1,116**
Less current obligation	**3,261**	**336**
Long-term obligation at December 31, 2019	**$ 18,393**	**$ 780**

As of December 31, 2019, we have contractually obligated lease payments amounting to $1.9 billion for office facility operating leases and small cell colocation and fiber operating leases that have not yet commenced. We have legally obligated lease payments for various other operating leases that have not yet commenced for which the total obligation was not significant. We have certain rights and obligations for these leases, but have not recognized an operating lease right-of-use asset or an operating lease liability since they have not yet commenced.

Real Estate Transaction

On July 23, 2019, Verizon completed a sale-leaseback transaction for buildings and real estate. We received total gross proceeds of approximately $1.0 billion. We leased back a portion of the buildings and real estate sold and accounted for it as an operating lease. The term of the leaseback is for two years with four options to renew for an additional three months each. The proceeds received as a result of this transaction have been classified in Other, net within Cash Flows from Investing Activities in our consolidated statement of cash flows for the year ended December 31, 2019. The net gain as a result of this transaction is included in the components of net lease cost table above.

Disclosures Related to Periods Prior to Adoption of Topic 842

Total rent expense under operating leases amounted to $4.1 billion in 2018 and $3.8 billion in 2017.

Amortization of capital leases was included in Depreciation and amortization expense in the consolidated statements of income. Capital lease amounts included in Property, plant and equipment were as follows:

(dollars in millions)

At December 31,	2018
Capital leases	$ 1,756
Less accumulated amortization	998
Total	$ 758

Note 7. Debt

Outstanding long-term debt obligations as of December 31, 2019 and 2018 are as follows:

(dollars in millions)

At December 31,	Maturities	Interest Rates %		2019	2018
Verizon Communications	**2019-2024**	**1.38 – 5.51**		**$ 19,885**	$ 24,242
	2025-2029	**1.38 – 6.80**		**30,038**	23,711
	2030-2055	**2.65 – 8.95**		**47,777**	54,662
	2019-2024	**Floating**	(1)	**2,210**	2,868
	2025-2029	**Floating**	(1)	**1,789**	1,789
Alltel Corporation	**2025-2029**	**6.80**		**38**	116
	2030-2055	**7.88**		**58**	118
Operating telephone company subsidiaries—debentures	**2019-2024**	**7.88 – 8.00**		**141**	147
	2025-2029	**6.00 – 8.38**		**286**	288
	2030-2055	**5.13 – 8.75**		**339**	361
GTE LLC	**2019-2024**	**8.75**		**141**	178
	2025-2029	**6.94**		**250**	266
Other subsidiaries—asset-backed debt	**2019-2024**	**1.42 – 3.56**		**8,116**	7,962
	2019-2024	**Floating**	(1)	**4,277**	2,139
Finance lease obligations (average rate of 3.2% and 4.1% in 2019 and 2018, respectively)				**1,116**	905
Unamortized discount, net of premium				**(4,480)**	(6,298)
Unamortized debt issuance costs				**(492)**	(541)
Total long-term debt, including current maturities				**111,489**	112,913
Less long-term debt maturing within one year				**10,777**	7,040
Total long-term debt				**$ 100,712**	$ 105,873
Total long-term debt, including current maturities				**$ 111,489**	$ 112,913
Plus short-term notes payable				**—**	150
Total debt				**$ 111,489**	$ 113,063

(1) The debt obligations bore interest at a floating rate based on the London Interbank Offered Rate (LIBOR) plus an applicable interest margin per annum.

Maturities of long-term debt (secured and unsecured) outstanding, including current maturities, excluding unamortized debt issuance costs, at December 31, 2019 are as follows:

Years	(dollars in millions)
2020	**$ 10,470**
2021	**7,269**
2022	**9,162**
2023	**5,591**
2024	**4,212**
Thereafter	**74,161**

During 2019, we received $18.7 billion of proceeds from long-term borrowings, which included $8.6 billion of proceeds from asset-backed debt transactions. The net proceeds were used for general corporate purposes including the repayment of debt. We used $23.9 billion of cash to repay, redeem and repurchase long-term borrowings and finance lease obligations, including $6.3 billion to prepay and repay asset-backed, long-term borrowings.

During 2018, we received $10.8 billion of proceeds from long-term borrowings, which included $4.8 billion of proceeds from asset-backed debt transactions. The net proceeds were used for general corporate purposes including the repayment of debt. We used $14.6 billion of cash to repay, redeem and repurchase long-term borrowings and finance lease obligations, including $3.6 billion to prepay and repay asset-backed, long-term borrowings.

2019 Significant Debt Transactions

The following tables show the significant transactions involving the senior unsecured debt securities of Verizon and its subsidiaries that occurred during the year ended December 31, 2019.

Exchange Offers

(dollars in millions)	**Principal Amount Exchanged**	**Principal Amount Issued**
Verizon 1.750% - 5.150% notes and floating rate notes, due 2021 - 2025	**$ 3,892**	**$ —**
GTE LLC 8.750% debentures, due 2021	**21**	**—**
Verizon 4.016% notes due 2029 [1]	**—**	**4,000**
Total	**$ 3,913**	**$ 4,000**

[1] The principal amount issued in exchange does not include either an insignificant amount of cash paid in lieu of the issuance of fractional new notes or accrued and unpaid interest paid on the old notes accepted for exchange to the date of exchange.

Tender Offers

(dollars in millions)	**Principal Amount Purchased**	**Cash Consideration[1]**
Verizon 4.672% - 5.012% notes due 2054 - 2055	**$ 4,500**	**$ 5,030**
Verizon 3.850% - 6.550% notes due 2039 - 2055	**3,816**	**4,828**
Verizon and other subsidiaries 5.050% - 8.950% notes and debentures due 2021 - 2041	**593**	**837**
Total	**$ 8,909**	**$ 10,695**

[1] The total cash consideration includes the tender offer consideration, plus any accrued and unpaid interest to the date of purchase.

Redemptions, Repurchases and Repayments

(dollars in millions)		Principal Redeemed/ Repurchased/ Repaid	Amount Paid as % of Principal [1]
Verizon 5.900% notes due 2054	$	500	100.000%
Verizon 1.375% notes due 2019		206	100.000%
Verizon 1.750% notes due 2021		621	100.000%
Verizon 3.000% notes due 2021		930	101.061%
Verizon 3.500% notes due 2021		315	102.180%
Verizon 2.625% notes due 2020		831	100.037%
Verizon 3.500% notes due 2021		736	102.238%
Verizon floating rate (LIBOR + 0.770%) notes due 2019		229	100.000%
Verizon 4.200% notes due 2046		2,059	100.000%
Verizon floating rate (LIBOR + 0.370%) notes due 2019		306	100.000%
Verizon 2.600% - 4.300% Internotes due 2022 - 2029		201	100.000%
Open market repurchases of various Verizon notes		543	Various
Total	$	7,477	

[1] Percentages represent price paid to redeem, repurchase and repay.

In February 2020, we redeemed, in whole, approximately $1.5 billion aggregate principal amount of 4.95% Notes due 2047.

Issuances

(dollars in millions)		Principal Amount Issued		Net Proceeds [1]
Verizon 3.875% notes due 2029 [2]	$	1,000	$	994
Verizon 5.000% notes due 2051	$	510		506
Verizon 0.875% notes due 2027	€	1,250		1,391
Verizon 1.250% notes due 2030	€	1,250		1,385
Verizon 2.500% notes due 2031	£	500		647
Verizon 0.875% notes due 2032	€	800		882
Verizon 1.500% notes due 2039	€	500		545
Verizon 1.875% notes due 2030	£	550		672
Verizon 2.100% notes due 2026	A$	450		307
Verizon 2.650% notes due 2030	A$	300		205
Verizon 3.500% notes due 2039	A$	500		341
Total			$	7,875

[1] Net proceeds were net of discount and issuance costs.
[2] An amount equal to the net proceeds from this green bond will be used to fund, in whole or in part, "Eligible Green Investments." "Eligible Green Investments" include new and existing investments made by us during the period from two years prior to the issuance of the green bond through the maturity date of the green bond, in the following categories: (1) renewable energy; (2) energy efficiency; (3) green buildings; (4) sustainable water management; and (5) biodiversity and conservation.

Short-Term Borrowing and Commercial Paper Program

In July 2018, we entered into a short-term uncommitted credit facility with the ability to borrow up to $700 million. As of December 31, 2019 and 2018, there was no outstanding balance.

As of December 31, 2019 and 2018, we had no commercial paper outstanding.

Asset-Backed Debt

As of December 31, 2019, the carrying value of our asset-backed debt was $12.4 billion. Our asset-backed debt includes Asset-Backed Notes (ABS Notes) issued to third-party investors (Investors) and loans (ABS Financing Facilities) received from banks and their conduit facilities (collectively, the Banks). Our consolidated asset-backed debt bankruptcy remote legal entities (each, an ABS Entity or collectively, the ABS Entities) issue the debt or are otherwise party to the transaction documentation in connection with our asset-backed debt transactions. Under the terms of our asset-backed debt, Cellco Partnership (Cellco) and certain other affiliates of Verizon (collectively, the Originators) transfer device payment plan agreement receivables to one of the ABS Entities, which in turn transfers such receivables to another ABS Entity that issues the debt. Verizon entities retain the equity interests in the ABS Entities, which represent the rights to all funds not needed to make required payments on the asset-backed debt and other related payments and expenses.

Our asset-backed debt is secured by the transferred device payment plan agreement receivables and future collections on such receivables. The device payment plan agreement receivables transferred to the ABS Entities and related assets, consisting primarily of restricted cash, will only be available for payment of asset-backed debt and expenses related thereto, payments to the Originators in respect of additional transfers of device payment plan agreement receivables, and other obligations arising from our asset-backed debt transactions, and will not be available to pay other obligations or claims of Verizon's creditors until the associated asset-backed debt and other obligations are satisfied. The Investors or Banks, as applicable, which hold our asset-backed debt have legal recourse to the assets securing the debt, but do not have any recourse to Verizon with respect to the payment of principal and interest on the debt. Under a parent support agreement, Verizon has agreed to guarantee certain of the payment obligations of Cellco and the Originators to the ABS Entities.

Cash collections on the device payment plan agreement receivables collateralizing our asset-backed debt securities are required at certain specified times to be placed into segregated accounts. Deposits to the segregated accounts are considered restricted cash and are included in Prepaid expenses and other, and Other assets in our consolidated balance sheets.

Proceeds from our asset-backed debt transactions are reflected in Cash flows from financing activities in our consolidated statements of cash flows. The asset-backed debt issued and the assets securing this debt are included in our consolidated balance sheets.

ABS Notes

During the year ended December 31, 2019, we completed the following ABS Notes transactions:

(dollars in millions)	Interest Rates %	Expected Weighted-average Life to Maturity (in years)	Principal Amount Issued
March 2019			
A-1a Senior class notes	**2.930**	**2.50**	**$ 900**
A-1b Senior floating rate class notes	**LIBOR + 0.330** [(1)]	**2.50**	**100**
B Junior class notes	**3.020**	**3.22**	**69**
C Junior class notes	**3.220**	**3.40**	**53**
March 2019 total			**1,122**
June 2019			
A-1a Senior class notes	**2.330**	**2.52**	**855**
A-1b Senior floating rate class notes	**LIBOR + 0.450** [(1)]	**2.52**	**145**
B Junior class notes	**2.400**	**3.28**	**69**
C Junior class notes	**2.600**	**3.47**	**53**
June 2019 total			**1,122**
October 2019			
A-1a Senior class notes	**1.940**	**2.51**	**1,276**
A-1b Senior floating rate class notes	**LIBOR + 0.420** [(1)]	**2.51**	**150**
B Junior class notes	**2.060**	**3.23**	**98**
C Junior class notes	**2.160**	**3.41**	**76**
October 2019 total			**1,600**
Total			**$ 3,844**

[(1)] The one-month LIBOR at December 31, 2019 was 1.763%.

Under the terms of each series of ABS Notes, there is a two year revolving period during which we may transfer additional receivables to the ABS Entity. In April, July and November 2019, the two year revolving period of the ABS Notes we issued in March, June and October 2017, respectively, ended, and we began to repay principal on the 2017-1, 2017-2 and 2017-3 Class A senior ABS Notes. In October 2019, in connection with an optional acquisition of receivables and redemption of 2016-1 Notes, we made a principal payment, in whole, for an insignificant amount. During the year ended December 31, 2019, we made aggregate principal repayments of $3.3 billion, for all ABS Notes.

In January 2020, we issued $1.6 billion aggregate principal amount of senior and junior Asset-Backed Notes through an ABS Entity.

ABS Financing Facility

In May 2018, we entered into an ABS financing facility with a number of financial institutions (2018 ABS Financing Facility). One loan agreement was entered into in connection with the 2018 ABS Financing Facility. In May 2019, the $540 million outstanding under the loan agreement was prepaid, and the loan agreement was terminated.

In September 2016, we entered into an ABS financing facility with a number of financial institutions (2016 ABS Financing Facility). Two loan agreements were entered into in connection with the 2016 ABS Financing Facility in September 2016 and May 2017. In April and May 2019, we paid off both the 2016 and 2017 loans for an aggregate of $671 million, and the loan agreements were terminated.

In May 2019, the 2016 ABS Financing Facility was amended and restated (2019 ABS Financing Facility). Under the terms of the 2019 ABS Financing Facility, which is an uncommitted facility, the financial institutions make advances under asset-backed loans backed by device payment plan agreement receivables of both consumer and business customers. One loan agreement was entered into in connection with the 2019 ABS Financing Facility. The 2019 loan agreement has a final maturity date in May 2023 and bears interest at floating rates. There is a one year revolving period until May 2020, which may be extended with the approval of the financial institutions. Under the 2019 loan agreement, we have the right to prepay all or a portion of the advances at any time without penalty, but in certain cases, with breakage costs. Subject to certain conditions, we may also remove receivables from the ABS Entity. In May 2019, we borrowed $1.8 billion under the 2019 loan agreement. In August 2019, we prepaid $1.5 billion of the loan made in May 2019 under the 2019 loan agreement. In November 2019, we borrowed an additional $1.5 billion under the 2019 loan agreement. In December 2019, the 2019 loan agreement was amended to increase the facility by an additional $1.5 billion, and an additional $1.5 billion was borrowed under the 2019 loan agreement. The aggregate outstanding balance under the 2019 ABS Financing Facility was $3.3 billion as of December 31, 2019. In January 2020, we prepaid $1.3 billion of the loan under the 2019 loan agreement.

Variable Interest Entities

The ABS Entities meet the definition of a VIE for which we have determined that we are the primary beneficiary as we have both the power to direct the activities of the entity that most significantly impact the entity's performance and the obligation to absorb losses or the right to receive benefits of the entity. Therefore, the assets, liabilities and activities of the ABS Entities are consolidated in our financial results and are included in amounts presented on the face of our consolidated balance sheets.

The assets and liabilities related to our asset-backed debt arrangements included in our consolidated balance sheets were as follows:

(dollars in millions)	At December 31, 2019	At December 31, 2018
Assets		
Accounts receivable, net	**$ 10,525**	$ 8,861
Prepaid expenses and other	**1,180**	989
Other assets	**3,856**	2,725
Liabilities		
Accounts payable and accrued liabilities	**11**	7
Debt maturing within one year	**5,578**	5,352
Long-term debt	**6,791**	4,724

See Note 8 for additional information on device payment plan agreement receivables used to secure asset-backed debt.

Long-Term Credit Facilities

			At December 31, 2019	
(dollars in millions)	**Maturities**	**Facility Capacity**	**Unused Capacity**	**Principal Amount Outstanding**
Verizon revolving credit facility [1]	**2022**	**$ 9,500**	**$ 9,390**	**N/A**
Various export credit facilities [2]	**2022-2027**	**5,500**	**—**	**4,471**
Total		**$ 15,000**	**$ 9,390**	**$ 4,471**

[1] The revolving credit facility does not require us to comply with financial covenants or maintain specified credit ratings, and it permits us to borrow even if our business has incurred a material adverse change. The revolving credit facility provides for the issuance of letters of credit.
[2] During 2019 and 2018, we drew down $1.5 billion and $3.0 billion from these facilities, respectively. We use these credit facilities to finance equipment-related purchases.

Non-Cash Transaction

During the years ended December 31, 2019, 2018 and 2017, we financed, primarily through vendor financing arrangements, the purchase of approximately $563 million, $1.1 billion, and $501 million, respectively, of long-lived assets consisting primarily of network equipment. At both December 31, 2019 and 2018, $1.1 billion relating to these financing arrangements, including those entered into in prior years and liabilities assumed through acquisitions, remained outstanding. These purchases are non-cash financing activities and therefore are not reflected within Capital expenditures in our consolidated statements of cash flows.

Early Debt Redemptions

During 2019, 2018 and 2017, we recorded losses on early debt redemptions of $3.7 billion, $681 million, and $2.0 billion, respectively.

We recognize losses on early debt redemptions in Other income (expense), net, in our consolidated statements of income. The total losses are reflected as an adjustment to reconcile net income to Net cash used in operating activities and the portion of the losses representing cash payments are reflected within Net cash used in financing activities in our consolidated statements of cash flows.

Guarantees

We guarantee the debentures of our operating telephone company subsidiaries. As of December 31, 2019, $765 million aggregate principal amount of these obligations remained outstanding. Each guarantee will remain in place for the life of the obligation unless terminated pursuant to its terms, including the operating telephone company no longer being a wholly-owned subsidiary of Verizon.

We also guarantee the debt obligations of GTE LLC as successor in interest to GTE Corporation that were issued and outstanding prior to July 1, 2003. As of December 31, 2019, $391 million aggregate principal amount of these obligations remain outstanding.

Debt Covenants

We and our consolidated subsidiaries are in compliance with all of our restrictive covenants in our debt agreements.

Note 8. Wireless Device Payment Plans

Under the Verizon device payment program, our eligible wireless customers purchase wireless devices under a device payment plan agreement. Customers that activate service on devices purchased under the device payment program pay lower service fees as compared to those under our fixed-term service plans, and their device payment plan charge is included on their wireless monthly bill. As of January 2017, we no longer offer Consumer customers new fixed-term, subsidized service plans for phones; however, we continue to offer subsidized plans to our Business customers. We also continue to service existing plans for customers who have not yet purchased and activated devices under the Verizon device payment program.

Wireless Device Payment Plan Agreement Receivables

The following table displays device payment plan agreement receivables, net, that are recognized in our consolidated balance sheets:

(dollars in millions)

At December 31,		**2019**		2018
Device payment plan agreement receivables, gross	**$**	**19,493**	$	19,313
Unamortized imputed interest		**(454)**		(546)
Device payment plan agreement receivables, net of unamortized imputed interest		**19,039**		18,767
Allowance for credit losses		**(472)**		(597)
Device payment plan agreement receivables, net	**$**	**18,567**	$	18,170
Classified in our consolidated balance sheets:				
Accounts receivable, net	**$**	**13,045**	$	12,624
Other assets		**5,522**		5,546
Device payment plan agreement receivables, net	**$**	**18,567**	$	18,170

Included in our device payment plan agreement receivables, net at December 31, 2019 and December 31, 2018, are net device payment plan agreement receivables of $14.3 billion and $11.5 billion, respectively, which have been transferred to ABS Entities and continue to be reported in our consolidated balance sheets. See Note 7 for additional information. We believe the carrying value of our installment loans receivables approximate their fair value using a Level 3 expected cash flow model.

We may offer certain promotions that allow a customer to trade in their owned device in connection with the purchase of a new device. Under these types of promotions, the customer receives a credit for the value of the trade-in device. In addition, we may provide the customer with additional future credits that will be applied against the customer's monthly bill as long as service is maintained. We recognize a liability for the customer's right to trade-in the device measured at fair value, which is determined by considering several factors, including the weighted-average selling prices obtained in recent resales of similar devices eligible for trade-in. Future credits are recognized when earned by the customer. Device payment plan agreement receivables, net does not reflect the trade-in device liability. At December 31, 2019 and December 31, 2018, the amount of trade-in liability was $103 million and $64 million, respectively.

From time to time, we offer certain marketing promotions that allow our customers to upgrade to a new device after paying down a certain specified portion of the required device payment plan agreement amount, as well as trading in their device in good working order. When a customer enters into a device payment plan agreement with the right to upgrade to a new device, we account for this trade-in right as a guarantee obligation.

For indirect channel wireless contracts with customers, we impute risk adjusted interest on the device payment plan agreement receivables. We record the imputed interest as a reduction to the related accounts receivable. Interest income, which is included within Service revenues and

other in our consolidated statements of income, is recognized over the financed device payment term. See Note 2 for additional information on financing considerations with respect to wireless direct channel contracts with customers.

When originating device payment plan agreements for Consumer customers, we use internal and external data sources to create a credit risk score to measure the credit quality of a customer and to determine eligibility for the device payment program. If a customer is either new to Verizon or has 45 days or less of customer tenure with Verizon, the credit decision process relies more heavily on external data sources. If the customer has more than 45 days of customer tenure with Verizon (an existing customer), the credit decision process relies on a combination of internal and external data sources. External data sources include obtaining a credit report from a national consumer credit reporting agency, if available. Verizon uses its internal data and/or credit data obtained from the credit reporting agencies to create a custom credit risk score. The custom credit risk score is generated automatically (except with respect to a small number of applications where the information needs manual intervention) from the applicant's credit data using Verizon's proprietary custom credit models, which are empirically derived and demonstrably and statistically sound. The credit risk score measures the likelihood that the potential customer will become severely delinquent and be disconnected for non-payment. For a small portion of new customer applications, a traditional credit report is not available from one of the national credit reporting agencies because the potential customer does not have sufficient credit history. In those instances, alternate credit data is used for the risk assessment.

Based on the custom credit risk score, we assign each customer to a credit class, each of which has specified offers of credit including an account level spending limit and either a maximum amount of credit allowed per device or a required down payment percentage. During the fourth quarter of 2018, Verizon moved all Consumer customers, new and existing, from a required down payment percentage, between zero and 100%, to a maximum amount of credit per device.

Subsequent to origination, Verizon monitors delinquency and write-off experience as key credit quality indicators for its portfolio of device payment plan agreements and fixed-term service plans. The extent of our collection efforts with respect to a particular customer are based on the results of proprietary custom empirically derived internal behavioral scoring models that analyze the customer's past performance to predict the likelihood of the customer falling further delinquent. These customer scoring models assess a number of variables, including origination characteristics, customer account history and payment patterns. Based on the score derived from these models, accounts are grouped by risk category to determine the collection strategy to be applied to such accounts. We continuously monitor collection performance results and the credit quality of our device payment plan agreement receivables based on a variety of metrics, including aging. Verizon considers an account to be delinquent and in default status if there are unpaid charges remaining on the account on the day after the bill's due date.

The balance and aging of the device payment plan agreement receivables on a gross basis were as follows:

			(dollars in millions)
At December 31,		**2019**	2018
Unbilled	**$**	**18,203**	$ 18,043
Billed:			
Current		**1,002**	986
Past due		**288**	284
Device payment plan agreement receivables, gross	**$**	**19,493**	$ 19,313

Activity in the allowance for credit losses for the device payment plan agreement receivables was as follows:

			(dollars in millions)
		2019	2018
Balance at January 1,	**$**	**597**	$ 848
Bad debt expense		**915**	459
Write-offs		**(1,040)**	(710)
Balance at December 31,	**$**	**472**	$ 597

Sales of Wireless Device Payment Plan Agreement Receivables

In 2015 and 2016, we established programs pursuant to a Receivables Purchase Agreement (RPA) to sell from time to time, on an uncommitted basis, eligible device payment plan agreement receivables to a group of primarily relationship banks (Purchasers) on both a revolving and non-revolving basis, collectively the Programs. Under the Programs, eligible device payment plan agreement receivables were transferred to the Purchasers for upfront cash proceeds and additional consideration upon settlement of the receivables, referred to as the deferred purchase price. In December 2017, the RPA and all other related transaction documents were terminated and as of December 31, 2017 we had no further continuing involvement with any of the receivables sold under the RPA program.

There were no sales of device payment plan agreement receivables under the Programs during 2017.

Deferred Purchase Price

Collections of deferred purchase price were $1.4 billion during 2017. During 2017, we repurchased all outstanding receivables previously sold to the Purchasers in exchange for the obligation to pay the associated deferred purchase price to the wholly-owned subsidiaries that were bankruptcy remote special purpose entities (Sellers). At December 31, 2017, our deferred purchase price receivable was fully satisfied. Collections

following the repurchase of receivables were insignificant, $195 million and $238 million during 2019, 2018 and 2017, respectively. Collections of both deferred purchase price and repurchased receivables were recorded in Cash flows used in investing activities in our consolidated statement of cash flows.

Note 9. Fair Value Measurements and Financial Instruments

Recurring Fair Value Measurements

The following table presents the balances of assets and liabilities measured at fair value on a recurring basis as of December 31, 2019:

(dollars in millions)

	Level 1[1]	Level 2[2]	Level 3[3]	Total
Assets:				
Other assets:				
Fixed income securities	$ —	$ 442	$ —	$ 442
Interest rate swaps	—	568	—	568
Cross currency swaps	—	211	—	211
Foreign exchange forwards	—	5	—	5
Total	$ —	$ 1,226	$ —	$ 1,226
Liabilities:				
Other liabilities:				
Interest rate swaps	$ —	$ 173	$ —	$ 173
Cross currency swaps	—	912	—	912
Forward starting interest rate swaps	—	604	—	604
Total	$ —	$ 1,689	$ —	$ 1,689

The following table presents the balances of assets and liabilities measured at fair value on a recurring basis as of December 31, 2018:

(dollars in millions)

	Level 1 [1]	Level 2 [2]	Level 3 [3]	Total
Assets:				
Other assets:				
Fixed income securities	$ —	$ 405	$ —	$ 405
Interest rate swaps	—	3	—	3
Cross currency swaps	—	220	—	220
Interest rate caps	—	14	—	14
Total	$ —	$ 642	$ —	$ 642
Liabilities:				
Other liabilities:				
Interest rate swaps	$ —	$ 813	$ —	$ 813
Cross currency swaps	—	536	—	536
Forward starting interest rate swaps	—	60	—	60
Interest rate caps	—	4	—	4
Total	$ —	$ 1,413	$ —	$ 1,413

[1] Quoted prices in active markets for identical assets or liabilities
[2] Observable inputs other than quoted prices in active markets for identical assets and liabilities
[3] Unobservable pricing inputs in the market

Certain of our equity investments do not have readily determinable fair values and are excluded from the tables above. Such investments are measured at cost, less any impairment, plus or minus changes resulting from observable price changes in orderly transactions for an identical or similar investment of the same issuer and are included in Investments in unconsolidated businesses in our consolidated balance sheets. As of December 31, 2019 and December 31, 2018, the carrying amount of our investments without readily determinable fair values was $284 million and $248 million, respectively. During 2019, there were insignificant adjustments due to observable price changes and we recognized an insignificant impairment charge. Cumulative adjustments due to observable price changes and impairment charges were insignificant.

Fixed income securities consist primarily of investments in municipal bonds. For fixed income securities that do not have quoted prices in active markets, we use alternative matrix pricing resulting in these debt securities being classified as Level 2.

Derivative contracts are valued using models based on readily observable market parameters for all substantial terms of our derivative contracts and thus are classified within Level 2. We use mid-market pricing for fair value measurements of our derivative instruments. Our derivative instruments are recorded on a gross basis.

We recognize transfers between levels of the fair value hierarchy as of the end of the reporting period. There were no transfers between Level 1 and Level 2 during 2019 and 2018.

Fair Value of Short-term and Long-term Debt

The fair value of our debt is determined using various methods, including quoted prices for identical terms and maturities, which is a Level 1 measurement, as well as quoted prices for similar terms and maturities in inactive markets and future cash flows discounted at current rates, which are Level 2 measurements. The fair value of our short-term and long-term debt, excluding finance leases, was as follows:

(dollars in millions)

At December 31,	2019		2018	
	Carrying Amount	**Fair Value**	Carrying Amount	Fair Value
Short- and long-term debt, excluding finance leases	**$ 110,373**	**$ 129,200**	$ 112,159	$ 118,535

Derivative Instruments

The following table sets forth the notional amounts of our outstanding derivative instruments:

(dollars in millions)

At December 31,	2019	2018
Interest rate swaps	**$ 17,004**	$ 19,813
Cross currency swaps	**23,070**	16,638
Forward starting interest rate swaps	**3,000**	4,000
Interest rate caps	**679**	2,218
Foreign exchange forwards	**1,130**	600

Interest Rate Swaps

We enter into interest rate swaps to achieve a targeted mix of fixed and variable rate debt. We principally receive fixed rates and pay variable rates that are currently based on LIBOR, resulting in a net increase or decrease to Interest expense. These swaps are designated as fair value hedges and hedge against interest rate risk exposure of designated debt issuances. We record the interest rate swaps at fair value in our consolidated balance sheets as assets and liabilities. Changes in the fair value of the interest rate swaps are recorded to Interest expense, which are offset by changes in the fair value of the hedged debt due to changes in interest rates.

During 2019, we entered into interest rate swaps with a total notional value of $510 million and settled interest rate swaps with a total notional value of $3.3 billion. During 2018, we entered into interest rate swaps with a total notional value of $730 million and settled interest rate swaps with a total notional value of $1.1 billion.

The ineffective portion of these interest rate swaps was $54 million and insignificant for the years ended December 31, 2019 and 2018, respectively.

The following amounts were recorded in Long-term debt in our consolidated balance sheets related to cumulative basis adjustments for fair value hedges:

(dollars in millions)

At December 31,	2019	2018
Carrying amount of hedged liabilities	**$ 17,337**	$ 18,903
Cumulative amount of fair value hedging adjustment included in the carrying amount of the hedged liabilities	**433**	(785)

Cross Currency Swaps

We have entered into cross currency swaps designated as cash flow hedges to exchange our British Pound Sterling, Euro, Swiss Franc and Australian Dollar-denominated cash flows into U.S. dollars and to fix our cash payments in U.S. dollars, as well as to mitigate the impact of foreign currency transaction gains or losses.

During 2019, we entered into cross currency swaps with a total notional value of $6.4 billion and did not settle any cross currency swaps. A pre-tax loss of $385 million was recognized in Other comprehensive loss with respect to these swaps.

During 2018, we did not enter into or settle any cross currency swaps. A pre-tax loss of $720 million was recognized in Other comprehensive loss with respect to these swaps.

A portion of the losses recognized in Other comprehensive loss was reclassified to Other income (expense), net to offset the related pre-tax foreign currency transaction gain or loss on the underlying hedged item.

Forward Starting Interest Rate Swaps

We have entered into forward starting interest rate swaps designated as cash flow hedges in order to manage our exposure to interest rate changes on future forecasted transactions.

During 2019, we did not enter into any forward starting interest rate swaps and we settled forward starting interest rate swaps with a total notional value of $1.0 billion. A pre-tax loss of $565 million, resulting from interest rate movements was recognized in Other comprehensive loss with respect to these swaps.

During 2018, we entered into forward starting interest rate swaps with a total notional value of $4.0 billion. A pre-tax loss of $60 million was recognized in Other comprehensive loss with respect to these swaps.

We hedge our exposure to the variability in future cash flows of based on the expected maturities of the related forecasted debt issuance.

Net Investment Hedges

We have designated certain foreign currency instruments as net investment hedges to mitigate foreign exchange exposure related to non-U.S. dollar net investments in certain foreign subsidiaries against changes in foreign exchange rates. The notional amount of the Euro-denominated debt as a net investment hedge was €750 million as of both December 31, 2019 and 2018, respectively.

Undesignated Derivatives

We also have the following derivative contracts which we use as economic hedges but for which we have elected not to apply hedge accounting.

Interest Rate Caps

We enter into interest rate caps to mitigate our interest exposure to interest rate increases on our ABS Financing Facility and ABS Notes. During both 2019 and 2018, we recognized an insignificant amount in Interest expense related to interest rate caps.

Foreign Exchange Forwards

We enter into British Pound Sterling and Euro foreign exchange forwards to mitigate our foreign exchange rate risk related to non-functional currency denominated monetary assets and liabilities of international subsidiaries. During 2019, we entered into foreign exchange forwards with a total notional value of $12.0 billion and settled foreign exchange forwards with a total notional value of $11.5 billion. During 2018, we entered into foreign exchange forwards with a total notional value of $2.8 billion and settled foreign exchange forwards with a total notional value of $2.2 billion. During 2019 and 2018, a pre-tax loss of insignificant amount was recognized in Other income (expense), net.

Treasury Rate Locks

During 2019, we entered into treasury rate locks with a total notional value of $1.5 billion to hedge the tender offers conducted in May 2019 for fifteen series of notes issued by Verizon with coupon rates ranging from 4.672% to 5.012% and maturity dates ranging from 2054 to 2055 (May Tender offers). In addition, we entered into treasury rate locks with a total notional value of $1.5 billion to hedge the tender offers conducted in November and December 2019 for eleven and twenty series of notes and debentures, respectively, issued by Verizon and other subsidiaries with coupon rates ranging from 3.850% to 8.950% and maturity dates ranging from 2021 to 2055 (November and December Tender offers). Upon the early settlement of the May, November and December Tender Offers, we settled these hedges and recognized an insignificant gain in Other income (expense), net.

During 2018, we entered into treasury rate locks with a total notional value of $2.0 billion to hedge the tender offers conducted in September 2018 for eight series of notes issued by Verizon with coupon rates ranging from 3.850% to 5.012% and maturity dates ranging from 2039 to 2055 (September Tender Offers). Upon the early settlement of the September Tender Offers, we settled these hedges and recognized an insignificant loss in Other income (expense), net.

Concentrations of Credit Risk

Financial instruments that subject us to concentrations of credit risk consist primarily of temporary cash investments, short-term and long-term investments, trade receivables, including device payment plan agreement receivables, certain notes receivable, including lease receivables, and derivative contracts.

Counterparties to our derivative contracts are major financial institutions with whom we have negotiated derivatives agreements (ISDA master agreements) and credit support annex (CSA) agreements which provide rules for collateral exchange. Negotiations and executions of new ISDA master agreements and CSA agreements with our counterparties continued during 2018. The CSA agreements contain rating based thresholds such that we or our counterparties may be required to hold or post collateral based upon changes in outstanding positions as compared to established thresholds and changes in credit ratings. At December 31, 2019, we held an insignificant amount and at December 31, 2018, we posted approximately $0.1 billion of collateral related to derivative contracts under collateral exchange arrangements, which were recorded as Other current liabilities and Prepaid expenses and other, respectively, in our consolidated balance sheets. While we may be exposed to credit

losses due to the nonperformance of our counterparties, we consider the risk remote and do not expect that any such nonperformance would result in a significant effect on our results of operations or financial condition due to our diversified pool of counterparties.

Note 10. Stock-Based Compensation

Verizon Long-Term Incentive Plan

In May 2017, Verizon's shareholders approved the 2017 Long-Term Incentive Plan (the 2017 Plan) and terminated Verizon's authority to grant new awards under the Verizon 2009 Long-Term Incentive Plan (the 2009 Plan). The 2017 Plan provides for broad-based equity grants to employees, including executive officers, and permits the granting of stock options, stock appreciation rights, restricted stock, restricted stock units, performance shares, performance stock units and other awards. Upon approval of the 2017 Plan, Verizon reserved for issuance under the 2017 Plan the number of shares that were remaining but not issued under the 2009 Plan. Shares subject to outstanding awards under the 2009 Plan that expire, are canceled or otherwise terminated will also be available for awards under the 2017 Plan. As of December 31, 2019, 89 million shares are reserved for future issuance under the 2017 Plan.

Restricted Stock Units

Restricted Stock Units (RSUs) granted under the 2017 Plan generally vest in three equal installments on each anniversary of the grant date. The RSUs that are paid in stock upon vesting and are thus classified as equity awards are measured using the grant date fair value of Verizon common stock and are not remeasured at the end of each reporting period. The RSUs that are settled in cash are classified as liability awards and the liability is measured at its fair value at the end of each reporting period. All RSUs granted under the 2017 Plan have dividend equivalent units, which will be paid to participants at the time the RSU award is paid, and in the same proportion as the RSU award.

In February 2018, Verizon announced a broad-based employee special award of RSUs under the 2017 Plan to eligible full-time and part-time employees. These RSUs are vested in two equal installments on each anniversary of the grant date and paid in cash. The first installment of the restricted stock units was vested and paid in February 2019 and the remaining restricted stock units will be vested and paid in February 2020.

In connection with our acquisition of Yahoo's operating business, on the closing date of the Transaction each unvested and outstanding Yahoo RSU award that was held by an employee who became an employee of Verizon was replaced with a Verizon RSU award, which is generally payable in cash upon the applicable vesting date. These awards are classified as liability awards and are measured at fair value at the end of each reporting period.

We estimate forfeitures at the time of grant and revise those estimates in subsequent periods if actual forfeitures differ from those estimates. We use historical data to estimate forfeitures and recognize that estimated compensation cost of restricted stock units, net of estimated forfeitures, on a straight-line basis over the vesting period.

Performance Stock Units

The 2017 Plan also provides for grants of Performance Stock Units (PSUs) that generally vest at the end of the third year after the grant. As defined by the 2017 Plan, the Human Resources Committee of the Board of Directors determines the number of PSUs a participant earns based on the extent to which the corresponding performance goals have been achieved over the three-year performance cycle. The PSUs are classified as liability awards because the PSU awards are paid in cash upon vesting. The PSU award liability is measured at its fair value at the end of each reporting period and, therefore, will fluctuate based on the price of Verizon common stock as well as performance relative to the targets. All PSUs granted under the 2017 Plan have dividend equivalent units, which will be paid to participants at the time that PSU award is determined and paid, and in the same proportion as the PSU award. The granted and cancelled activity for the PSU award includes adjustments for the performance goals achieved.

The following table summarizes Verizon's Restricted Stock Unit and Performance Stock Unit activity:

	Restricted Stock Units		Performance
(shares in thousands)	**Equity Awards**	**Liability Awards**	**Stock Units**
Outstanding January 1, 2017	13,308	—	17,919
Granted	4,216	25,168	6,564
Payments	(4,825)	(8,487)	(6,031)
Cancelled/Forfeited	(66)	(2,690)	(217)
Outstanding December 31, 2017	12,633	13,991	18,235
Granted	4,134	15,157	5,779
Payments	(5,977)	(6,860)	(4,526)
Cancelled/Forfeited	(213)	(2,362)	(2,583)
Outstanding December 31, 2018	10,577	19,926	16,905
Granted	**3,169**	**5,814**	**4,593**
Payments	**(6,397)**	**(9,429)**	**(3,255)**
Cancelled/Forfeited	**(90)**	**(1,598)**	**(2,692)**
Outstanding December 31, 2019	**7,259**	**14,713**	**15,551**

As of December 31, 2019, unrecognized compensation expense related to the unvested portion of Verizon's RSUs and PSUs was approximately $765 million and is expected to be recognized over approximately two years.

The equity RSUs granted in 2019 and 2018 have weighted-average grant date fair values of $56.66 and $49.19 per unit, respectively. During 2019, 2018 and 2017, we paid $737 million, $773 million and $750 million, respectively, to settle RSUs and PSUs classified as liability awards.

Stock-Based Compensation Expense

After-tax compensation expense for stock-based compensation related to RSUs and PSUs described above included in Net income attributable to Verizon was $872 million, $720 million and $384 million for 2019, 2018 and 2017, respectively.

Note 11. Employee Benefits

We maintain non-contributory defined benefit pension plans for certain employees. In addition, we maintain postretirement health care and life insurance plans for certain retirees and their dependents, which are both contributory and non-contributory, and include a limit on our share of the cost for certain current and future retirees. In accordance with our accounting policy for pension and other postretirement benefits, operating expenses include service costs associated with pension and other postretirement benefits while other credits and/or charges based on actuarial assumptions, including projected discount rates, an estimated return on plan assets, and impact from health care trend rates are reported in Other income (expense), net. These estimates are updated in the fourth quarter to reflect actual return on plan assets and updated actuarial assumptions or upon a remeasurement. The adjustment is recognized in the income statement during the fourth quarter or upon a remeasurement event pursuant to our accounting policy for the recognition of actuarial gains and losses.

Pension and Other Postretirement Benefits

Pension and other postretirement benefits for certain employees are subject to collective bargaining agreements. Modifications in benefits have been bargained from time to time, and we may also periodically amend the benefits in the management plans. The following tables summarize benefit costs, as well as the benefit obligations, plan assets, funded status and rate assumptions associated with pension and postretirement health care and life insurance benefit plans.

Obligations and Funded Status

(dollars in millions)

	Pension		Health Care and Life	
At December 31,	**2019**	2018	**2019**	2018
Change in Benefit Obligations				
Beginning of year	**$ 19,567**	$ 21,531	**$ 16,364**	$ 19,460
Service cost	**247**	284	**96**	127
Interest cost	**695**	690	**629**	615
Plan amendments	**—**	230	**(22)**	(8)
Actuarial (gain) loss, net	**2,860**	(1,418)	**(414)**	(2,729)
Benefits paid	**(1,248)**	(1,475)	**(984)**	(1,101)
Curtailment and termination benefits	**—**	181	**—**	—
Settlements paid	**(873)**	(456)	**—**	—
End of year	**21,248**	19,567	**15,669**	16,364
Change in Plan Assets				
Beginning of year	**17,816**	19,175	**1,175**	1,119
Actual return on plan assets	**3,385**	(494)	**103**	(26)
Company contributions	**371**	1,066	**449**	1,183
Benefits paid	**(1,248)**	(1,475)	**(984)**	(1,101)
Settlements paid	**(873)**	(456)	**—**	—
End of year	**19,451**	17,816	**743**	1,175
Funded Status				
End of year	**$ (1,797)**	$ (1,751)	**$ (14,926)**	$ (15,189)

(dollars in millions)

At December 31,	Pension 2019	Pension 2018	Health Care and Life 2019	Health Care and Life 2018
Amounts recognized on the balance sheet				
Noncurrent assets	**$ 5**	$ 3	**$ —**	$ —
Current liabilities	**(67)**	(71)	**(603)**	(292)
Noncurrent liabilities	**(1,735)**	(1,683)	**(14,323)**	(14,897)
Total	**$ (1,797)**	$ (1,751)	**$ (14,926)**	$ (15,189)
Amounts recognized in Accumulated Other Comprehensive Income (Pre-tax)				
Prior service cost (benefit)	**$ 524**	$ 585	**$ (3,749)**	$ (4,698)
Total	**$ 524**	$ 585	**$ (3,749)**	$ (4,698)

The accumulated benefit obligation for all defined benefit pension plans was $21.2 billion and $19.5 billion at December 31, 2019 and 2018, respectively.

2018 Collective Bargaining Negotiations

The extension agreement ratified in August 2018 extended our collective bargaining agreements with the Communications Workers of America and the International Brotherhood of Electrical Workers that were due to expire on August 3, 2019 for four years until August 5, 2023. Amendments triggered by the collective bargaining negotiations were made to certain pension plans for certain union-represented employees and retirees. The impact of the plan amendments was an increase in our defined benefit pension plans plan obligations and a net decrease to Accumulated other comprehensive income of $230 million (net of taxes of $170 million). The annual impact of the amount recorded in Accumulated other comprehensive income that will be reclassified to net periodic benefit cost is insignificant.

2017 Postretirement Plan Amendments

During 2017, amendments were made to certain postretirement plans related to retiree medical benefits for management and certain union-represented employees and retirees. The impact of the plan amendments was a reduction in our postretirement benefit plan obligations of approximately $527 million, which has been recorded as a net increase to Accumulated other comprehensive income of $317 million (net of taxes of $210 million). The impact of the amount recorded in Accumulated other comprehensive income that will be reclassified to net periodic benefit cost is insignificant.

2016 Collective Bargaining Negotiations

During 2016, we adopted changes to our defined benefit pension plans and other postretirement benefit plans to reflect the agreed upon terms and conditions of the collective bargaining agreements ratified in June 2016. The impact includes a net increase to Accumulated other comprehensive income of $2.9 billion (net of taxes of $1.8 billion). The amount recorded in Accumulated other comprehensive income will be reclassified to net periodic benefit cost on a straight-line basis over the average remaining service period of the respective plans' participants, which, on a weighted-average basis, is 12.2 years for defined benefit pension plans and 7.8 years for other postretirement benefit plans. The above-noted reclassification resulted in a decrease to net periodic benefit cost and increase to pre-tax income of approximately $658 million during 2019, 2018 and 2017, respectively.

Information for pension plans with an accumulated benefit obligation in excess of plan assets follows:

(dollars in millions)

At December 31,	2019	2018
Projected benefit obligation	**$ 21,190**	$ 19,510
Accumulated benefit obligation	**21,134**	19,461
Fair value of plan assets	**19,388**	17,757

Net Periodic Benefit Cost (Income)

The following table summarizes the components of net periodic benefit cost (income) related to our pension and postretirement health care and life insurance plans:

(dollars in millions)

	Pension			Health Care and Life		
Years Ended December 31,	**2019**	2018	2017	**2019**	2018	2017
Service cost - Cost of services	**$ 202**	$ 230	$ 215	**$ 78**	$ 104	$ 116
Service cost - Selling, general and administrative expense	**45**	54	65	**18**	23	33
Service cost	**247**	284	280	**96**	127	149
Amortization of prior service cost (credit)	**61**	48	39	**(971)**	(976)	(949)
Expected return on plan assets	**(1,130)**	(1,293)	(1,262)	**(37)**	(44)	(53)
Interest cost	**695**	690	683	**629**	615	659
Remeasurement loss (gain), net	**606**	369	337	**(480)**	(2,658)	546
Curtailment and termination benefits	**—**	181	11	**—**	—	—
Other components	**232**	(5)	(192)	**(859)**	(3,063)	203
Total	**$ 479**	$ 279	$ 88	**$ (763)**	$ (2,936)	$ 352

The service cost component of net periodic benefit cost (income) is recorded in Cost of services and Selling, general and administrative expense in the consolidated statements of income while the other components, including mark-to-market adjustments, if any, are recorded in Other income (expense), net.

Other pre-tax changes in plan assets and benefit obligations recognized in other comprehensive (income) loss are as follows:

(dollars in millions)

	Pension			Health Care and Life		
At December 31,	**2019**	2018	2017	**2019**	2018	2017
Prior service cost (benefit)	**$ —**	$ 230	$ —	**$ (22)**	$ (8)	$ (544)
Reversal of amortization items						
Prior service cost (benefit)	**(61)**	(48)	(39)	**971**	976	949
Total recognized in other comprehensive loss (income) (pre-tax)	**$ (61)**	$ 182	$ (39)	**$ 949**	$ 968	$ 405

The estimated prior service cost for the defined benefit pension plans that will be amortized from Accumulated other comprehensive income into net periodic benefit cost over the next fiscal year is $61 million. The estimated prior service cost for the defined benefit postretirement plans that will be amortized from Accumulated other comprehensive income into net periodic benefit income over the next fiscal year is $1.0 billion.

Assumptions

The weighted-average assumptions used in determining benefit obligations follow:

	Pension		Health Care and Life	
At December 31,	**2019**	2018	**2019**	2018
Discount Rate	**3.30%**	4.40%	**3.20%**	4.30%
Rate of compensation increases	**3.00**	3.00	**N/A**	N/A

The weighted-average assumptions used in determining net periodic cost follow:

	Pension			Health Care and Life		
At December 31,	**2019**	2018	2017	**2019**	2018	2017
Discount rate in effect for determining service cost	**4.60%**	4.10%	4.70%	**4.60%**	3.90%	4.60%
Discount rate in effect for determining interest cost	**3.80**	3.40	3.40	**4.00**	3.20	3.50
Expected return on plan assets	**6.80**	7.00	7.70	**4.30**	4.80	4.50
Rate of compensation increases	**3.00**	3.00	3.00	**N/A**	N/A	N/A

In determining our pension and other postretirement benefit obligations, we used a weighted-average discount rate of 3.3% in 2019. The rates were selected to approximate the composite interest rates available on a selection of high-quality bonds available in the market at December 31, 2019. The bonds selected had maturities that coincided with the time periods during which benefits payments are expected to occur, were non-callable and available in sufficient quantities to ensure marketability (at least $300 million par outstanding).

In order to project the long-term target investment return for the total portfolio, estimates are prepared for the total return of each major asset class over the subsequent 10-year period. Those estimates are based on a combination of factors including the current market interest rates and valuation levels, consensus earnings expectations and historical long-term risk premiums. To determine the aggregate return for the pension trust, the projected return of each individual asset class is then weighted according to the allocation to that investment area in the trust's long-term asset allocation policy.

The assumed health care cost trend rates are as follows:

	Health Care and Life		
At December 31,	**2019**	2018	2017
Healthcare cost trend rate assumed for next year	**6.10%**	6.30%	7.00%
Rate to which cost trend rate gradually declines	**4.50**	4.50	4.50
Year the rate reaches the level it is assumed to remain thereafter	**2027**	2027	2026

A one-percentage point change in the assumed health care cost trend rate would have the following effects:

(dollars in millions)

One-Percentage Point	Increase	Decrease
Effect on 2019 service and interest cost	**$ 20**	**$ (21)**
Effect on postretirement benefit obligation as of December 31, 2019	**626**	**(696)**

Plan Assets

The Company's overall investment strategy is to achieve a mix of assets that allows us to meet projected benefit payments while taking into consideration risk and return. While target allocation percentages will vary over time, the current target allocation for plan assets is designed so that 48% to 68% of the assets have the objective of achieving a return in excess of the growth in liabilities (comprised of public equities, private equities, real estate, hedge funds and emerging debt) and 35% to 55% of the assets are invested as liability hedging assets (where cash flows from investments better match projected benefit payments, typically longer duration fixed income) and a maximum of 15% is in cash. This allocation will shift as funded status improves to a higher allocation of liability hedging assets. Target policies will be revisited periodically to ensure they are in line with fund objectives. Both active and passive management approaches are used depending on perceived market efficiencies and various other factors. Due to our diversification and risk control processes, there are no significant concentrations of risk, in terms of sector, industry, geography or company names.

Pension and healthcare and life plans assets do not include significant amounts of Verizon common stock.

Pension Plans

The fair values for the pension plans by asset category at December 31, 2019 are as follows:

(dollars in millions)

Asset Category	**Total**	**Level 1**	**Level 2**	**Level 3**
Cash and cash equivalents	**$ 1,529**	**$ 1,507**	**$ 22**	**$ —**
Equity securities	**2,988**	**2,850**	**135**	**3**
Fixed income securities				
U.S. Treasuries and agencies	**1,986**	**1,768**	**218**	**—**
Corporate bonds	**3,818**	**524**	**3,149**	**145**
International bonds	**1,355**	**25**	**1,304**	**26**
Other	**768**	**—**	**768**	**—**
Real estate	**810**	**—**	**—**	**810**
Other				
Private equity	**737**	**—**	**—**	**737**
Hedge funds	**293**	**—**	**164**	**129**
Total investments at fair value	**14,284**	**6,674**	**5,760**	**1,850**
Investments measured at NAV	**5,167**			
Total	**$ 19,451**	**$ 6,674**	**$ 5,760**	**$ 1,850**

The fair values for the pension plans by asset category at December 31, 2018 are as follows:

(dollars in millions)

Asset Category	Total	Level 1	Level 2	Level 3
Cash and cash equivalents	$ 1,701	$ 1,694	$ 7	$ —
Equity securities	2,253	2,220	20	13
Fixed income securities				
U.S. Treasuries and agencies	1,684	1,557	127	—
Corporate bonds	3,645	124	3,244	277
International bonds	1,113	19	1,076	18
Other	—	—	—	—
Real estate	727	—	—	727
Other				
Private equity	664	—	—	664
Hedge funds	459	—	373	86
Total investments at fair value	12,246	5,614	4,847	1,785
Investments measured at NAV	5,570			
Total	$ 17,816	$ 5,614	$ 4,847	$ 1,785

The following is a reconciliation of the beginning and ending balance of pension plan assets that are measured at fair value using significant unobservable inputs:

(dollars in millions)

	Equity Securities	Corporate Bonds	International Bonds	Real Estate	Private Equity	Hedge Funds	Total
Balance at January 1, 2018	$ 1	$ 104	$ 20	$ 627	$ 580	$ 185	$ 1,517
Actual gain (loss) on plan assets	1	(7)	3	134	25	—	156
Purchases (sales)	11	177	(5)	(34)	59	62	270
Transfers out	—	3	—	—	—	(161)	(158)
Balance at December 31, 2018	13	277	18	727	664	86	1,785
Actual gain (loss) on plan assets	**1**	**(1)**	**(1)**	**30**	**32**	**—**	**61**
Purchases (sales)	**(11)**	**18**	**9**	**53**	**41**	**116**	**226**
Transfers out	**—**	**(149)**	**—**	**—**	**—**	**(73)**	**(222)**
Balance at December 31, 2019	**$ 3**	**$ 145**	**$ 26**	**$ 810**	**$ 737**	**$ 129**	**$ 1,850**

Health Care and Life Plans

The fair values for the other postretirement benefit plans by asset category at December 31, 2019 are as follows:

(dollars in millions)

Asset Category	Total	Level 1	Level 2	Level 3
Cash and cash equivalents	**$ 220**	**$ 167**	**$ 53**	**$ —**
Equity securities	**225**	**225**	**—**	**—**
Fixed income securities				
U.S. Treasuries and agencies	**28**	**28**	**—**	**—**
Corporate bonds	**76**	**76**	**—**	**—**
International bonds	**18**	**18**	**—**	**—**
Total investments at fair value	**567**	**514**	**53**	**—**
Investments measured at NAV	**176**			
Total	**$ 743**	**$ 514**	**$ 53**	**$ —**

The fair values for the other postretirement benefit plans by asset category at December 31, 2018 are as follows:

(dollars in millions)

Asset Category	Total	Level 1	Level 2	Level 3
Cash and cash equivalents	$ 471	$ 431	$ 40	$ —
Equity securities	239	239	—	—
Fixed income securities				
U.S. Treasuries and agencies	24	24	—	—
Corporate bonds	96	96	—	—
International bonds	18	18	—	—
Total investments at fair value	848	808	40	—
Investments measured at NAV	327			
Total	$ 1,175	$ 808	$ 40	$ —

The following are general descriptions of asset categories, as well as the valuation methodologies and inputs used to determine the fair value of each major category of assets.

Cash and cash equivalents include short-term investment funds (less than 90 days to maturity), primarily in diversified portfolios of investment grade money market instruments and are valued using quoted market prices or other valuation methods. The carrying value of cash equivalents approximates fair value due to the short-term nature of these investments.

Investments in securities traded on national and foreign securities exchanges are valued by the trustee at the last reported sale prices on the last business day of the year or, if no sales were reported on that date, at the last reported bid prices. Government obligations, corporate bonds, international bonds and asset-backed debt are valued using matrix prices with input from independent third-party valuation sources. Over-the-counter securities are valued at the bid prices or the average of the bid and ask prices on the last business day of the year from published sources or, if not available, from other sources considered reliable such as multiple broker quotes.

Commingled funds not traded on national exchanges are priced by the custodian or fund's administrator at their net asset value (NAV). Commingled funds held by third-party custodians appointed by the fund managers provide the fund managers with a NAV. The fund managers have the responsibility for providing this information to the custodian of the respective plan.

The investment manager of the entity values venture capital, corporate finance and natural resource limited partnership investments. Real estate investments are valued at amounts based upon appraisal reports prepared by either independent real estate appraisers or the investment manager using discounted cash flows or market comparable data. Loans secured by mortgages are carried at the lesser of the unpaid balance or appraised value of the underlying properties. The values assigned to these investments are based upon available and current market information and do not necessarily represent amounts that might ultimately be realized. Because of the inherent uncertainty of valuation, estimated fair values might differ significantly from the values that would have been used had a ready market for the securities existed. These differences could be material.

Forward currency contracts, futures, and options are valued by the trustee at the exchange rates and market prices prevailing on the last business day of the year. Both exchange rates and market prices are readily available from published sources. These securities are classified by the asset class of the underlying holdings.

Hedge funds are valued by the custodian at NAV based on statements received from the investment manager. These funds are valued in accordance with the terms of their corresponding offering or private placement memoranda.

Commingled funds, hedge funds, venture capital, corporate finance, natural resource and real estate limited partnership investments for which fair value is measured using the NAV per share as a practical expedient are not leveled within the fair value hierarchy and are included as a reconciling item to total investments.

Employer Contributions

In 2019, we made a $300 million discretionary contribution to our qualified pension plans, $71 million of contributions to our nonqualified pension plans and $449 million of contributions to our other postretirement benefit plans. No qualified pension plans contributions are expected to be made in 2020. Nonqualified pension plans contributions are estimated to be approximately $70 million and contributions to our other postretirement benefit plans are estimated to be approximately $700 million in 2020.

Estimated Future Benefit Payments

The benefit payments to retirees are expected to be paid as follows:

(dollars in millions)

Year	Pension Benefits	Health Care and Life
2020	**$ 2,227**	**$ 961**
2021	**1,680**	**947**
2022	**1,620**	**930**
2023	**1,577**	**968**
2024	**1,072**	**951**
2025 to 2029	**5,248**	**4,569**

Savings Plan and Employee Stock Ownership Plans

We maintain four leveraged employee stock ownership plans (ESOP). We match a certain percentage of eligible employee contributions to certain savings plans with shares of our common stock from this ESOP. At December 31, 2019, the number of allocated shares of common stock in this ESOP was 49 million. There were no unallocated shares of common stock in this ESOP at December 31, 2019. All leveraged ESOP shares are included in earnings per share computations.

Total savings plan costs were $897 million in 2019, $1.1 billion in 2018 and $838 million in 2017.

Severance Benefits

The following table provides an analysis of our severance liability:

(dollars in millions)

Year	Beginning of Year	Charged to Expense	Payments	Other	End of Year
2017	$ 656	$ 581	$ (564)	$ (46)	$ 627
2018	627	2,093	(560)	(4)	2,156
2019	**2,156**	**260**	**(1,847)**	**(4)**	**565**

Severance, Pension and Benefits (Credits) Charges

During 2019, in accordance with our accounting policy to recognize actuarial gains and losses in the period in which they occur, we recorded net pre-tax pension and benefits charges of $126 million in our pension and postretirement benefit plans. The charges were recorded in Other income (expense), net in our consolidated statement of income and were primarily driven by a decrease in our discount rate assumption used to determine the current year liabilities of our pension plans and postretirement benefit plans from a weighted-average of 4.4% at December 31, 2018 to a weighted-average of 3.3% at December 31, 2019 ($4.3 billion), partially offset by the difference between our estimated return on assets and our actual return on assets ($2.3 billion) and other assumption adjustments of $1.9 billion, of which $1.6 billion related to healthcare claims experience. During 2019, we also recorded net pre-tax severance charges of $260 million in Selling, general and administrative expense in our consolidated statements of income.

During 2018, we recorded net pre-tax pension and benefits credits of $2.1 billion in accordance with our accounting policy to recognize actuarial gains and losses in the period in which they occur. The pension and benefits remeasurement credits of $2.3 billion, which were recorded in Other income (expense), net in our consolidated statements of income, were primarily driven by an increase in our discount rate assumption used to determine the current year liabilities of our pension plans and postretirement benefit plans from a weighted-average of 3.7% at December 31, 2017 to a weighted-average of 4.4% at December 31, 2018 ($2.6 billion), and mortality and other assumption adjustments of $1.7 billion, $1.6 billion of which related to healthcare claims and trend adjustments, offset by the difference between our estimated return on assets of 7.0% and our actual return on assets of (2.7)% ($1.9 billion). The credits were partially offset by $177 million due to the effect of participants retiring under the Voluntary Separation Program.

In September 2018, Verizon announced a Voluntary Separation Program for select U.S.-based management employees. Approximately 10,400 eligible employees separated from the Company under this program as of the end of June 2019. The severance benefit payments to these employees were substantially completed by the end of September 2019. Principally as a result of this program but also as a result of other headcount reduction initiatives, the Company recorded a severance charge of $1.8 billion ($1.4 billion after-tax) during the year ended December 31, 2018, which was recorded in Selling, general and administrative expense in our consolidated statement of income. During 2018, we also recorded $339 million in severance costs under our other existing separation plans.

During 2017, we recorded net pre-tax severance, pension and benefits charges of $1.4 billion, exclusive of acquisition related severance charges, in accordance with our accounting policy to recognize actuarial gains and losses in the period in which they occur. The pension and benefits remeasurement charges of approximately $911 million, which were recorded in Other income (expense), net in our consolidated statements of income, were primarily driven by a decrease in our discount rate assumption used to determine the current year liabilities of our pension and postretirement benefit plans from a weighted-average of 4.2% at December 31, 2016 to a weighted-average of 3.7% at December 31, 2017 ($2.6 billion). The charges were partially offset by the difference between our estimated return on assets of 7.0% and our actual return on assets

of 14.0% ($1.2 billion), a change in mortality assumptions primarily driven by the use of updated actuarial tables (MP-2017) issued by the Society of Actuaries ($227 million) and other assumption adjustments ($320 million). As part of these charges, we also recorded severance costs of $497 million under our existing separation plans, which were recorded in Selling, general and administrative expense in our consolidated statement of income.

Note 12. Taxes

The components of income before provision (benefit) for income taxes are as follows:

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Domestic	**$ 21,655**	$ 19,801	$ 19,645
Foreign	**1,078**	(178)	949
Total	**$ 22,733**	$ 19,623	$ 20,594

The components of the provision (benefit) for income taxes are as follows:

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Current			
Federal	**$ 518**	$ 2,187	$ 3,630
Foreign	**221**	267	200
State and Local	**974**	741	677
Total	**1,713**	3,195	4,507
Deferred			
Federal	**1,150**	175	(14,360)
Foreign	**(13)**	30	(66)
State and Local	**95**	184	(37)
Total	**1,232**	389	(14,463)
Total income tax provision (benefit)	**$ 2,945**	$ 3,584	$ (9,956)

The following table shows the principal reasons for the difference between the effective income tax rate and the statutory federal income tax rate:

Years Ended December 31,	2019	2018	2017
Statutory federal income tax rate	**21.0 %**	21.0 %	35.0 %
State and local income tax rate, net of federal tax benefits	**3.7**	3.7	1.6
Preferred stock disposition	**(9.9)**	—	—
Affordable housing credit	**(0.4)**	(0.6)	(0.6)
Employee benefits including ESOP dividend	**(0.3)**	(0.3)	(0.5)
Impact of tax reform re-measurement	**—**	—	(81.6)
Internal restructure	**—**	(9.1)	(0.6)
Noncontrolling interests	**(0.5)**	(0.5)	(0.6)
Non-deductible goodwill	**0.1**	4.7	1.0
Other, net	**(0.7)**	(0.6)	(2.0)
Effective income tax rate	**13.0 %**	18.3 %	(48.3)%

The effective income tax rate for 2019 was 13.0% compared to 18.3% for 2018. The decrease in the effective income tax rate and the provision for income taxes was primarily due to the recognition of approximately $2.2 billion of a non-recurring tax benefit in connection with the disposition of preferred stock, representing a minority interest in a foreign affiliate in 2019 compared to the non-recurring deferred tax benefit of approximately $2.1 billion, as a result of an internal reorganization of legal entities within the historical Wireless business, which was offset by a goodwill charge that is not deductible for tax purposes in 2018.

The effective income tax rate for 2018 was 18.3% compared to (48.3)% for 2017. The increase in the effective income tax rate and the provision for income taxes was primarily due to the non-recurring, non-cash income tax benefit of $16.8 billion recorded in 2017 for the re-measurement of U.S. deferred tax liabilities at the lower 21% U.S. federal corporate income tax rate, as a result of the enactment of the TCJA on December 22, 2017. In addition, the provision for income taxes for 2018 includes the tax impact of the Media goodwill impairment charge not deductible for tax purposes, offset by the reduction in the statutory U.S federal corporate income tax rate from 35% to 21%, effective January 1, 2018 under the TCJA and a non-recurring deferred tax benefit of approximately $2.1 billion as a result of an internal reorganization of legal entities within the historical Wireless business.

In December 2017, the Securities and Exchange Commission staff issued Staff Accounting Bulletin (SAB) 118 to provide guidance for companies that had not completed their accounting for the income tax effects of the TCJA. Due to the complexities involved in accounting for the enactment of the TCJA, SAB 118 allowed for a provisional estimate of the impacts of the TCJA in our earnings for the year ended December 31, 2017, as well as up to a one year measurement period that ended on December 22, 2018, for any subsequent adjustments to such provisional estimate. In 2018, Verizon completed its analysis of the impacts of the TCJA, including analyzing the effects of any IRS and U.S. Treasury guidance issued, and state tax law changes enacted, within the one year measurement period resulting in no significant adjustments to the $16.8 billion provisional amount recorded in December 2017.

The amounts of cash taxes paid by Verizon are as follows:

(dollars in millions)

Years Ended December 31,	**2019**	2018	2017
Income taxes, net of amounts refunded	**$ 3,583**	$ 2,213	$ 4,432
Employment taxes	**1,044**	1,066	1,207
Property and other taxes	**1,551**	1,598	1,737
Total	**$ 6,178**	$ 4,877	$ 7,376

Deferred Tax Assets and Liabilities

Deferred taxes arise because of differences in the book and tax bases of certain assets and liabilities. Significant components of deferred tax assets and liabilities are as follows:

(dollars in millions)

At December 31,	**2019**	2018
Deferred Tax Assets		
Employee benefits	**$ 5,048**	$ 5,403
Tax loss and credit carry forwards	**3,012**	3,576
Other - assets	**5,595**	1,650
	13,655	10,629
Valuation allowances	**(2,260)**	(2,741)
Deferred tax assets	**11,395**	7,888
Deferred Tax Liabilities		
Spectrum and other intangible amortization	**22,388**	21,976
Depreciation	**16,884**	15,662
Other - liabilities	**6,742**	3,976
Deferred tax liabilities	**46,014**	41,614
Net deferred tax liability	**$ 34,619**	$ 33,726

At December 31, 2019, undistributed earnings of our foreign subsidiaries indefinitely invested outside the U.S. amounted to approximately $3.8 billion. The majority of Verizon's cash flow is generated from domestic operations and we are not dependent on foreign cash or earnings to meet our funding requirements, nor do we intend to repatriate these undistributed foreign earnings to fund U.S. operations. Furthermore, a portion of these undistributed earnings represents amounts that legally must be kept in reserve in accordance with certain foreign jurisdictional requirements and are unavailable for distribution or repatriation. As a result, we have not provided U.S. deferred taxes on these undistributed earnings because we intend that they will remain indefinitely reinvested outside of the U.S. and therefore unavailable for use in funding U.S. operations. Determination of the amount of unrecognized deferred taxes related to these undistributed earnings is not practicable.

At December 31, 2019, we had net after-tax loss and credit carry forwards for income tax purposes of approximately $3.0 billion that primarily relate to state and foreign taxes. Of these net after-tax loss and credit carry forwards, approximately $2.0 billion will expire between 2020 and 2039 and approximately $1.0 billion may be carried forward indefinitely.

During 2019, the valuation allowance decreased approximately $481 million. The balance of the valuation allowance at December 31, 2019 and the 2019 activity is primarily related to state and foreign taxes.

Unrecognized Tax Benefits

A reconciliation of the beginning and ending balance of unrecognized tax benefits is as follows:

(dollars in millions)

	2019	2018	2017
Balance at January 1,	**$ 2,871**	$ 2,355	$ 1,902
Additions based on tax positions related to the current year	**149**	160	219
Additions for tax positions of prior years	**297**	699	756
Reductions for tax positions of prior years	**(300)**	(248)	(419)
Settlements	**(58)**	(40)	(42)
Lapses of statutes of limitations	**(89)**	(55)	(61)
Balance at December 31,	**$ 2,870**	$ 2,871	$ 2,355

Included in the total unrecognized tax benefits at December 31, 2019, 2018 and 2017 is $2.4 billion, $2.3 billion and $1.9 billion, respectively, that if recognized, would favorably affect the effective income tax rate.

We recognized the following net after-tax expenses related to interest and penalties in the provision for income taxes:

Years Ended December 31,	(dollars in millions)
2019	**$ 35**
2018	75
2017	77

The after-tax accruals for the payment of interest and penalties in the consolidated balance sheets are as follows:

At December 31,	(dollars in millions)
2019	**$ 385**
2018	348

Verizon and/or its subsidiaries file income tax returns in the U.S. federal jurisdiction, and various state, local and foreign jurisdictions. As a large taxpayer, we are under audit by the IRS and multiple state and foreign jurisdictions for various open tax years. The IRS is currently examining the Company's U.S. income tax returns for tax years 2013-2014 and Cellco Partnership's U.S. income tax return for tax year 2013-2014. Tax controversies are ongoing for tax years as early as 2005. The amount of the liability for unrecognized tax benefits will change in the next twelve months due to the expiration of the statute of limitations in various jurisdictions and it is reasonably possible that various current tax examinations will conclude or require reevaluations of the Company's tax positions during this period. An estimate of the range of the possible change cannot be made until these tax matters are further developed or resolved.

Note 13. Segment Information

Reportable Segments

As discussed in Note 1, in November 2018, we announced a strategic reorganization of our business. Under the new structure, effective April 1, 2019, there are two reportable segments that we operate and manage as strategic business units - Consumer and Business. We measure and evaluate our reportable segments based on segment operating income, consistent with the chief operating decision maker's assessment of segment performance.

Our segments and their principal activities consist of the following:

Segment	Description
Verizon Consumer Group	Our Consumer segment provides consumer-focused wireless and wireline communications services and products. Our wireless services are provided across one of the most extensive wireless networks in the United States under the Verizon brand and through wholesale and other arrangements. Our wireline services are provided in nine states in the Mid-Atlantic and Northeastern U.S., as well as Washington D.C., over our 100% fiber-optic network under the Fios brand and over a traditional copper-based network to customers who are not served by Fios.
Verizon Business Group	Our Business segment provides wireless and wireline communications services and products, video and data services, corporate networking solutions, security and managed network services, local and long distance voice services and network access to deliver various IoT services and products. We provide these products and services to businesses, government customers and wireless and wireline carriers across the U.S. and select products and services to customers around the world.

Our Consumer segment's wireless and wireline products and services are available to our retail customers, as well as resellers that purchase wireless network access from us on a wholesale basis. Our Business segment's wireless and wireline products and services are organized by the primary customer groups targeted by these offerings: Global Enterprise, Small and Medium Business, Public Sector and Other, and Wholesale.

Corporate and other includes the results of our media business,Verizon Media, and other businesses, investments in unconsolidated businesses, unallocated corporate expenses, certain pension and other employee benefit related costs and interest and financing expenses. Corporate and

other also includes the historical results of divested businesses and other adjustments and gains and losses that are not allocated in assessing segment performance due to their nature. Although such transactions are excluded from the business segment results, they are included in reported consolidated earnings. Gains and losses from these transactions that are not individually significant are included in segment results as these items are included in the chief operating decision maker's assessment of segment performance.

We completed our acquisition of Yahoo's operating business on June 13, 2017 and as such results are included since the acquisition date.

In May 2017, we completed the Data Center Sale, where we sold 23 customer-facing data center sites in the U.S. and Latin America to Equinix. The results of operations for this divestiture and other insignificant transactions are included within Corporate and other for all periods presented to reflect comparable segment operating results consistent with the information regularly reviewed by our chief operating decision maker.

The reconciliation of segment operating revenues and expenses to consolidated operating revenues and expenses below includes the effects of special items that the chief operating decision maker does not consider in assessing segment performance, primarily because of their nature.

The following tables provides operating financial information for our two reportable segments:

(dollars in millions)

2019	Consumer	Business	Total Reportable Segments
External Operating Revenues			
Service	**$ 65,384**	**$ —**	**$ 65,384**
Wireless equipment	**18,048**	**—**	**18,048**
Other	**7,384**	**—**	**7,384**
Global Enterprise	**—**	**10,815**	**10,815**
Small and Medium Business	**—**	**11,447**	**11,447**
Public Sector and Other	**—**	**5,922**	**5,922**
Wholesale	**—**	**3,198**	**3,198**
Intersegment revenues	**240**	**61**	**301**
Total Operating Revenues[1]	**91,056**	**31,443**	**122,499**
Cost of services	**15,884**	**10,655**	**26,539**
Cost of wireless equipment	**18,219**	**4,733**	**22,952**
Selling, general and administrative expense	**16,639**	**8,188**	**24,827**
Depreciation and amortization expense	**11,353**	**4,105**	**15,458**
Total Operating Expenses	**62,095**	**27,681**	**89,776**
Operating Income	**$ 28,961**	**$ 3,762**	**$ 32,723**

[1] Service and other revenues and Wireless equipment revenues included in our Business segment amounted to approximately $27.9 billion and $3.5 billion, respectively, for the year ended December 31, 2019.

(dollars in millions)

2018	Consumer	Business	Total Reportable Segments
External Operating Revenues			
Service	$ 64,207	$ —	$ 64,207
Wireless equipment	18,874	—	18,874
Other	6,447	—	6,447
Global Enterprise	—	11,197	11,197
Small and Medium Business	—	10,732	10,732
Public Sector and Other	—	5,830	5,830
Wholesale	—	3,713	3,713
Intersegment revenues	234	62	296
Total Operating Revenues[1]	89,762	31,534	121,296
Cost of services	15,335	10,859	26,194
Cost of wireless equipment	18,763	4,560	23,323
Selling, general and administrative expense	15,701	7,689	23,390
Depreciation and amortization expense	11,952	4,258	16,210
Total Operating Expenses	61,751	27,366	89,117
Operating Income	$ 28,011	$ 4,168	$ 32,179

(1) Service and other revenues and Wireless equipment revenues included in our Business segment amounted to approximately $28.1 billion and $3.4 billion, respectively, for the year ended December 31, 2018.

(dollars in millions)

2017	Consumer	Business	Total Reportable Segments
External Operating Revenues			
Service	$ 63,769	$ —	$ 63,769
Wireless equipment	17,292	—	17,292
Other	5,735	—	5,735
Global Enterprise	—	11,444	11,444
Small and Medium Business	—	9,793	9,793
Public Sector and Other	—	5,652	5,652
Wholesale	—	3,978	3,978
Intersegment revenues	258	46	304
Total Operating Revenues[1]	87,054	30,913	117,967
Cost of services	14,981	11,094	26,075
Cost of wireless equipment	17,713	4,434	22,147
Selling, general and administrative expense	17,292	7,448	24,740
Depreciation and amortization expense	11,308	4,483	15,791
Total Operating Expenses	61,294	27,459	88,753
Operating Income	$ 25,760	$ 3,454	$ 29,214

(1) Service and other revenues and Wireless equipment revenues included in our Business segment amounted to approximately $29.3 billion and $1.6 billion, respectively, for the year ended December 31, 2017.

The following table provides Fios revenues for our two reportable segments:

(dollars in millions)

Years Ended December 31,	**2019**	2018	2017
Consumer	**$ 11,175**	$ 11,056	$ 10,903
Business	**967**	883	788
Total Fios revenue	**$ 12,142**	$ 11,939	$ 11,691

The following table provides Wireless service revenue for our reportable segments and includes intersegment activity:

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Consumer	**$ 53,791**	$ 52,459	$ 51,954
Business	**11,188**	10,484	11,093
Total Wireless service revenue	**$ 64,979**	$ 62,943	$ 63,047

Reconciliation to Consolidated Financial Information

A reconciliation of the reportable segment operating revenues to consolidated operating revenues is as follows:

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Operating Revenues			
Total reportable segments	**$ 122,499**	$ 121,296	$ 117,967
Corporate and other	**9,812**	9,936	8,098
Reconciling items:			
Operating results from divested businesses (Note 3)	**—**	—	368
Eliminations	**(443)**	(369)	(399)
Consolidated Operating Revenues	**$ 131,868**	$ 130,863	$ 126,034

A reconciliation of the total reportable segments' operating income to consolidated income before provision for income taxes is as follows:

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Operating Income			
Total reportable segments	**$ 32,723**	$ 32,179	$ 29,214
Corporate and other	**(1,403)**	(1,326)	(1,119)
Reconciling items:			
Severance charges	**(204)**	(2,157)	(497)
Other components of net periodic pension and benefit (charges) credits (Note 11)	**(813)**	(823)	(800)
Net gain on sale of divested businesses (Note 3)	**—**	—	1,774
Acquisition and integration related charges (Note 3)	**—**	(553)	(884)
Gain on spectrum license transactions (Note 3)	**—**	—	270
Operating results from divested businesses	**—**	—	149
Impairment charges	**(186)**	(4,591)	—
Product realignment charges	**—**	(451)	(682)
Net gain from dispositions of assets and businesses	**261**	—	—
Consolidated operating income	**30,378**	22,278	27,425
Equity in losses of unconsolidated businesses	**(15)**	(186)	(77)
Other income (expense), net	**(2,900)**	2,364	(2,021)
Interest expense	**(4,730)**	(4,833)	(4,733)
Income Before (Provision) Benefit For Income Taxes	**$ 22,733**	$ 19,623	$ 20,594

No single customer accounted for more than 10% of our total operating revenues during the years ended December 31, 2019, 2018 and 2017. International operating revenues are not significant.

The chief operating decision maker does not review disaggregated assets on a segment basis; therefore, such information is not presented. Depreciation included in the measure of segment profitability is primarily allocated based on proportional usage.

Note 14. Equity and Comprehensive Income

Equity

In December 2019, 46,100 preferred shares of a foreign affiliate of Verizon was sold for cash consideration of $51 million and is reflected in non-controlling interests. The preferred shares pay cumulative dividends of 8.25% per annum.

Common Stock

In February 2020, the Verizon Board of Directors authorized a share buyback program to repurchase up to 100 million shares of the Company's common stock. The program will terminate when the aggregate number of shares purchased reaches 100 million, or a new share repurchase plan superseding the current plan is authorized, whichever is sooner. During the years ended December 31, 2019, 2018, and 2017, Verizon did not repurchase any shares of Verizon's common stock under our previously authorized share buyback programs. At December 31, 2019, the maximum number of shares that could be purchased by or on behalf of Verizon under our share buyback program was 100 million.

Common stock has been used from time to time to satisfy some of the funding requirements of employee and shareholder plans. During the years ended December 31, 2019, 2018, and 2017, we issued 3.8 million, 3.5 million and 2.8 million common shares from Treasury stock, respectively, which had an insignificant aggregate value.

In connection with our acquisition of Straight Path in February 2018, we issued approximately 49 million shares of Verizon common stock, valued at approximately $2.4 billion.

Accumulated Other Comprehensive Income

Comprehensive income consists of net income and other gains and losses affecting equity that, under U.S. GAAP, are excluded from net income. Significant changes in the components of Other comprehensive income, net of provision for income taxes are described below.

The changes in the balances of Accumulated other comprehensive income by component are as follows:

(dollars in millions)	Foreign currency translation adjustments	Unrealized gains (losses) on cash flow hedges	Unrealized gains (losses) on marketable securities	Defined benefit pension and postretirement plans	Total
Balance at January 1, 2017	$ (713)	$ (80)	$ 46	$ 3,420	$ 2,673
Other comprehensive income	245	818	10	327	1,400
Amounts reclassified to net income	—	(849)	(24)	(541)	(1,414)
Net other comprehensive income (loss)	245	(31)	(14)	(214)	(14)
Balance at December 31, 2017	(468)	(111)	32	3,206	2,659
Opening balance sheet adjustment (Note 1)	(15)	(24)	(13)	682	630
Adjusted opening balance	(483)	(135)	19	3,888	3,289
Other comprehensive income (loss)	(117)	(574)	—	(164)	(855)
Amounts reclassified to net income	—	629	1	(694)	(64)
Net other comprehensive income (loss)	(117)	55	1	(858)	(919)
Balance at December 31, 2018	**(600)**	**(80)**	**20**	**3,030**	**2,370**
Other comprehensive income (loss)	**16**	**(699)**	**8**	**—**	**(675)**
Amounts reclassified to net income	**—**	**(37)**	**(1)**	**(659)**	**(697)**
Net other comprehensive income (loss)	**16**	**(736)**	**7**	**(659)**	**(1,372)**
Balance at December 31, 2019	**$ (584)**	**$ (816)**	**$ 27**	**$ 2,371**	**$ 998**

The amounts presented above in net other comprehensive income (loss) are net of taxes. The amounts reclassified to net income related to unrealized gains (losses) on cash flow hedges in the table above are included in Other income (expense), net and Interest expense in our consolidated statements of income. See Note 9 for additional information. The amounts reclassified to net income related to unrealized gains (losses) on marketable securities in the table above are included in Other income (expense), net in our consolidated statements of income. The amounts reclassified to net income related to defined benefit pension and postretirement plans in the table above are included in Cost of services and Selling, general and administrative expense in our consolidated statements of income. See Note 11 for additional information.

Note 15. Additional Financial Information

The following tables provide additional financial information related to our consolidated financial statements:

Income Statement Information

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Depreciation expense	**$ 14,371**	$ 15,186	$ 14,741
Interest costs on debt balances	**5,221**	5,399	5,256
Net amortization of debt discount	**165**	174	155
Capitalized interest costs	**(656)**	(740)	(678)
Advertising expense	**3,071**	2,682	2,643
Other income (expense), net			
Interest income	**$ 121**	$ 94	$ 82
Other components of net periodic benefit (cost) income	**627**	3,068	(11)
Early debt extinguishment costs	**(3,604)**	(725)	(1,983)
Other, net	**(44)**	(73)	(109)
	$ (2,900)	$ 2,364	$ (2,021)

Balance Sheet Information

(dollars in millions)

At December 31,	2019	2018
Prepaid expenses and other		
Prepaid taxes	**$ 2,438**	$ 348
Deferred contract costs	**2,578**	2,083
Restricted cash	**1,221**	1,047
Other prepaid expense and other	**1,791**	1,975
	$ 8,028	$ 5,453
Accounts payable and accrued liabilities		
Accounts payable	**$ 7,725**	$ 7,232
Accrued expenses	**5,984**	5,948
Accrued vacation, salaries and wages	**4,885**	6,268
Interest payable	**1,441**	1,570
Taxes payable	**1,771**	1,483
	$ 21,806	$ 22,501
Other current liabilities		
Dividends payable	**$ 2,566**	$ 2,512
Contract liability	**4,651**	4,207
Other	**1,807**	1,520
	$ 9,024	$ 8,239

Cash Flow Information

(dollars in millions)

Years Ended December 31,	2019	2018	2017
Cash Paid			
Interest, net of amounts capitalized	**$ 4,714**	$ 4,408	$ 4,369
Income taxes, net of amounts refunded	**3,583**	2,213	4,432
Other, net Cash Flows from Operating Activities			
Changes in device payment plan agreement non-current receivables	**$ 23**	$ (509)	$ (579)
Early debt extinguishment costs	**3,604**	725	1,983
Other, net	**(228)**	3	(728)
	$ 3,399	$ 219	$ 676
Other, net Cash Flows from Financing Activities			
Net debt related costs	**$ (1,797)**	$ (141)	$ (3,599)
Change in short-term obligations, excluding current maturities	**—**	(790)	(170)
Other, net	**(1,120)**	(893)	(670)
	$ (2,917)	$ (1,824)	$ (4,439)

Note 16. Commitments and Contingencies

In the ordinary course of business, Verizon is involved in various commercial litigation and regulatory proceedings at the state and federal level. Where it is determined, in consultation with counsel based on litigation and settlement risks, that a loss is probable and estimable in a given matter, the Company establishes an accrual. In none of the currently pending matters is the amount of accrual material. An estimate of the reasonably possible loss or range of loss in excess of the amounts already accrued cannot be made at this time due to various factors typical in contested proceedings, including: (1) uncertain damage theories and demands; (2) a less than complete factual record; (3) uncertainty concerning legal theories and their resolution by courts or regulators; and (4) the unpredictable nature of the opposing party and its demands. We continuously monitor these proceedings as they develop and adjust any accrual or disclosure as needed. We do not expect that the ultimate resolution of any pending regulatory or legal matter in future periods will have a material effect on our financial condition, but it could have a material effect on our results of operations for a given reporting period.

Verizon is currently involved in approximately 25 federal district court actions alleging that Verizon is infringing various patents. Most of these cases are brought by non-practicing entities and effectively seek only monetary damages; a small number are brought by companies that have sold products and could seek injunctive relief as well. These cases have progressed to various stages and a small number may go to trial in the coming 12 months if they are not otherwise resolved.

In connection with the execution of agreements for the sales of businesses and investments, Verizon ordinarily provides representations and warranties to the purchasers pertaining to a variety of nonfinancial matters, such as ownership of the securities being sold, as well as indemnity from certain financial losses. From time to time, counterparties may make claims under these provisions, and Verizon will seek to defend against those claims and resolve them in the ordinary course of business.

Subsequent to the sale of Verizon Information Services Canada in 2004, we continue to provide a guarantee to publish directories, which was issued when the directory business was purchased in 2001 and had a 30-year term (before extensions). The preexisting guarantee continues, without modification, despite the subsequent sale of Verizon Information Services Canada and the spin-off of our domestic print and Internet yellow pages directories business. The possible financial impact of the guarantee, which is not expected to be adverse, cannot be reasonably estimated as a variety of the potential outcomes available under the guarantee result in costs and revenues or benefits that may offset each other. We do not believe performance under the guarantee is likely.

As of December 31, 2019, letters of credit totaling approximately $632 million, which were executed in the normal course of business and support several financing arrangements and payment obligations to third parties, were outstanding.

During 2019, Verizon entered into a renewable energy purchase agreement (REPA) with a third party. The REPA is based on the expected operation of a renewable energy-generating facility and has a fixed price term of 12 years from the commencement of the facility's entry into commercial operation, which is expected to begin by the end of 2020. The REPA generally is expected to be financially settled based on the prevailing market price as energy is generated by the facility.

We have various commitments, totaling $18.8 billion, primarily to purchase programming and network services, equipment, software and marketing services, which will be used or sold in the ordinary course of business, from a variety of suppliers. Of this total amount, $8.4 billion is attributable to 2020, $7.5 billion is attributable to 2021 through 2022, $1.4 billion is attributable to 2023 through 2024 and $1.5 billion is attributable to years thereafter. These amounts do not represent our entire anticipated purchases in the future, but represent only those items that are the subject of contractual obligations. Our commitments are generally determined based on the noncancelable quantities or termination

amounts. Purchases against our commitments totaled approximately $10.9 billion for 2019, $9.0 billion for 2018 and $8.2 billion for 2017. Since the commitments to purchase programming services from television networks and broadcast stations have no minimum volume requirement, we estimated our obligation based on number of subscribers at December 31, 2019, and applicable rates stipulated in the contracts in effect at that time. We also purchase products and services as needed with no firm commitment.

Note 17. Quarterly Financial Information (Unaudited)

(dollars in millions, except per share amounts)

Quarter Ended	**First Quarter**	**Second Quarter**	**Third Quarter**	**Fourth Quarter**	**Full Year**
2019					
Operating Revenues	**$ 32,128**	**$ 32,071**	**$ 32,894**	**$ 34,775**	**$ 131,868**
Operating Income	**7,709**	**7,850**	**8,180**	**6,639**	**30,378**
Net Income	**5,160**	**4,074**	**5,337**	**5,217**	**19,788**
Net Income Attributable to Verizon	**5,032**	**3,944**	**5,194**	**5,095**	**19,265**
Basic Earnings Per Share Attributable to Verizon[1]	**$ 1.22**	**$ 0.95**	**$ 1.26**	**$ 1.23**	**$ 4.66**
Diluted Earnings Per Share Attributable to Verizon[1]	**$ 1.22**	**$ 0.95**	**$ 1.25**	**$ 1.23**	**$ 4.65**
2018					
Operating Revenues	$ 31,772	$ 32,203	$ 32,607	$ 34,281	$ 130,863
Operating Income	7,349	6,617	7,675	637	22,278
Net Income	4,666	4,246	5,062	2,065	16,039
Net Income Attributable to Verizon	4,545	4,120	4,924	1,939	15,528
Basic Earnings Per Share Attributable to Verizon[1]	$ 1.11	$ 1.00	$ 1.19	$ 0.47	$ 3.76
Diluted Earnings Per Share Attributable to Verizon[1]	$ 1.11	$ 1.00	$ 1.19	$ 0.47	$ 3.76

[1] Net income attributable to Verizon per common share is computed independently for each quarter and the sum of the quarters may not equal the annual amount.

Results of operations for 2019 and 2018 include the following after-tax charges (credits) attributable to Verizon:

(dollars in millions)

	2019				2018			
	First Quarter	**Second Quarter**	**Third Quarter**	**Fourth Quarter**	First Quarter	Second Quarter	Third Quarter	Fourth Quarter
Severance, pension and benefits charges (credits)	**$ (71)**	**$ —**	**$ 215**	**$ 108**	$ —	$ 250	$ (335)	$ 108
Early debt redemption costs	**—**	**1,140**	**—**	**1,520**	184	—	352	—
Acquisition and integration related charges	**—**	**—**	**—**	**—**	82	92	103	142
Product realignment charges	**—**	**—**	**—**	**—**	—	509	—	—
Net gain from dispositions of assets and businesses	**—**	**—**	**(224)**	**—**	—	—	—	—
Disposition of preferred stock	**—**	**—**	**—**	**(2,247)**	—	—	—	—
Impairment charges	**—**	**—**	**—**	**214**	—	—	—	4,527
Historical Wireless legal entity restructuring	**—**	**—**	**—**	**—**	—	—	—	(2,065)

Disposition of Preferred Stock

During the fourth quarter of 2019, we completed the disposition of preferred stock, representing a minority interest in a foreign affiliate, which resulted in a non-recurring income tax benefit of approximately $2.2 billion in our consolidated statement of income for the year ended December 31, 2019.

Historical Wireless Legal Entity Restructuring

During the fourth quarter of 2018, we completed an internal reorganization of legal entities within the historical Wireless business which resulted in a non-recurring income tax benefit of approximately $2.1 billion in our consolidated statement of income for the year ended December 31, 2018, which reduced our deferred tax liability by the same amount.

EXHIBIT 21

Verizon Communications Inc. and Subsidiaries
Principal Subsidiaries of Registrant at December 31, 2019

Name	State of Incorporation / Organization
Verizon Delaware LLC	Delaware
Verizon Maryland LLC	Delaware
Verizon New England Inc.	New York
Verizon New Jersey Inc.	New Jersey
Verizon New York Inc.	New York
Verizon Pennsylvania LLC	Delaware
Verizon Virginia LLC	Virginia
Bell Atlantic Mobile Systems LLC	Delaware
Cellco Partnership (d/b/a Verizon Wireless)	Delaware
GTE LLC	Delaware
GTE Wireless LLC	Delaware
MCI Communications Corporation	Delaware
Verizon Americas Inc.	Delaware
Verizon Business Global LLC	Delaware

EXHIBIT 23

Consent of Independent Registered Public Accounting Firm and Report on Schedule

Consent

We consent to the incorporation by reference in the following Registration Statements:

Form S-4, No. 333-11573; Form S-8, No. 333-41593; Form S-8, No. 333-50146; Form S-4, No. 333-76171; Form S-8, No. 333-76171; Form S-8, No. 333-53830; Form S-8, No. 333-82690; Form S-4, No. 333-124008; Form S-8, No. 333-124008; Form S-4, No. 333-132651; Form S-8, No. 333-172501; Form S-8, No. 333-172999; Form S-8, No. 333-200398; Form S-8, No. 333-217717; Form S-8, No. 333-223523; and Form S-3, No. 333-233608;

of our reports dated February 21, 2020, with respect to the consolidated financial statements and the effectiveness of internal control over financial reporting of Verizon Communications Inc. ("Verizon"), incorporated by reference in this Annual Report (Form 10-K) of Verizon for the year ended December 31, 2019, and the financial statement schedule of Verizon, included herein.

Report on Schedule

To the Shareholders and the Board of Directors of Verizon Communications Inc.:

We have audited the consolidated financial statements of Verizon as of December 31, 2019 and 2018, and for each of the three years in the period ended December 31, 2019, and have issued our report thereon dated February 21, 2020 incorporated by reference in this Annual Report (Form 10-K) of Verizon from the 2019 Annual Report to Shareholders of Verizon. Our audits of the consolidated financial statements included the financial statement schedule listed in Item 15(a) of this Annual Report (Form 10-K) (the “schedule”). This schedule is the responsibility of Verizon's management. Our responsibility is to express an opinion on Verizon’s schedule based on our audits.

In our opinion, the schedule presents fairly, in all material respects, the information set forth therein when considered in conjunction with the consolidated financial statements.

/s/ Ernst & Young LLP

Ernst & Young LLP

New York, New York

February 21, 2020

EXHIBIT 24

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg, Matthew D. Ellis and Anthony T. Skiadas and each of them, her true and lawful attorneys-in-fact and agents with full power of substitution, for her and in her name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as she might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Shellye L. Archambeau

Shellye L. Archambeau

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Mark T. Bertolini

Mark T. Bertolini

POWER OF ATTORNEY

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Vittorio Colao

Vittorio Colao

POWER OF ATTORNEY

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Melanie L. Healey

Melanie L. Healey

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Clarence Otis, Jr.

Clarence Otis, Jr.

POWER OF ATTORNEY

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Daniel H. Schulman

Daniel H. Schulman

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg, Matthew D. Ellis and Anthony T. Skiadas and each of them, his true and lawful attorneys-in-fact and agents with full power of substitution, for him and in his name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as he might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Rodney E. Slater

Rodney E. Slater

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg, Matthew D. Ellis and Anthony T. Skiadas and each of them, her true and lawful attorneys-in-fact and agents with full power of substitution, for her and in her name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as she might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Kathryn A. Tesija

Kathryn A. Tesija

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg, Matthew D. Ellis and Anthony T. Skiadas and each of them, her true and lawful attorneys-in-fact and agents with full power of substitution, for her and in her name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as she might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Carol B. Tomé

Carol B. Tomé

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Matthew D. Ellis and Anthony T. Skiadas and each of them, his true and lawful attorneys-in-fact and agents with full power of substitution, for him and in his name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as he might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Hans E. Vestberg

Hans E. Vestberg

POWER OF ATTORNEY

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IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Gregory G. Weaver

Gregory G. Weaver

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg and Anthony T. Skiadas and each of them, his true and lawful attorneys-in-fact and agents with full power of substitution, for him and in his name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as he might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Matthew D. Ellis

Matthew D. Ellis

POWER OF ATTORNEY

WHEREAS, VERIZON COMMUNICATIONS INC., a Delaware corporation (hereinafter referred to as the "Company"), proposes to file with the Securities and Exchange Commission under the provisions of the Securities Exchange Act of 1934, as amended, an annual report on Form 10-K (the "Form 10-K") for the fiscal year ended December 31, 2019.

NOW, THEREFORE, the undersigned hereby appoints Hans E. Vestberg and Matthew D. Ellis and each of them, his true and lawful attorneys-in-fact and agents with full power of substitution, for him and in his name, place and stead, in any and all capacities, to sign the Form 10-K and any and all amendments to the Form 10-K, and to file the same, with all exhibits thereto and all documents in connection therewith, making such changes in the Form 10-K as such person or persons so acting deems appropriate, with the Securities and Exchange Commission, granting unto said attorneys-in-fact and agents, and each of them, full power and authority to do and perform each and every act and thing requisite and necessary to be done in and about the premises, as fully to all intents and purposes as he might or could do in person, hereby ratifying and confirming all that said attorneys-in-fact and agents or any of them, or his, her or their substitute or substitutes, may lawfully do or cause to be done by virtue hereof.

IN WITNESS WHEREOF, the undersigned has executed this Power of Attorney this 20th day of February, 2020.

/s/ Anthony T. Skiadas

Anthony T. Skiadas

EXHIBIT 31.1

I, Hans E. Vestberg, certify that:

1. I have reviewed this annual report on Form 10-K of Verizon Communications Inc.;

2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;

3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;

4. The registrant's other certifying officer and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e)) and internal control over financial reporting (as defined in Exchange Act Rules 13a-15(f) and 15d-15(f)) for the registrant and have:

   (a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;

   (b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;

   (c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

   (d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

5. The registrant's other certifying officer and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):

   (a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

   (b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: February 21, 2020

/s/ Hans E. Vestberg

Hans E. Vestberg

Chairman and Chief Executive Officer

EXHIBIT 31.2

I, Matthew D. Ellis, certify that:

1. I have reviewed this annual report on Form 10-K of Verizon Communications Inc.;

2. Based on my knowledge, this report does not contain any untrue statement of a material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this report;

3. Based on my knowledge, the financial statements, and other financial information included in this report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this report;

4. The registrant's other certifying officer and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-15(e) and 15d-15(e)) and internal control over financial reporting (as defined in Exchange Act Rules 13a-15(f) and 15d-15(f)) for the registrant and have:

   (a) Designed such disclosure controls and procedures, or caused such disclosure controls and procedures to be designed under our supervision, to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this report is being prepared;

   (b) Designed such internal control over financial reporting, or caused such internal control over financial reporting to be designed under our supervision, to provide reasonable assurance regarding the reliability of financial reporting and the preparation of financial statements for external purposes in accordance with generally accepted accounting principles;

   (c) Evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

   (d) Disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

5. The registrant's other certifying officer and I have disclosed, based on our most recent evaluation of internal control over financial reporting, to the registrant's auditors and the audit committee of the registrant's board of directors (or persons performing the equivalent functions):

   (a) All significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

   (b) Any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: February 21, 2020

/s/ Matthew D. Ellis

Matthew D. Ellis

Executive Vice President and Chief Financial Officer

EXHIBIT 32.1

CERTIFICATION OF CHIEF EXECUTIVE OFFICER PURSUANT TO SECTION 906 OF THE SARBANES-OXLEY ACT OF 2002, PURSUANT TO SECTION 1350 OF CHAPTER 63 OF TITLE 18 OF THE UNITED STATES CODE

I, Hans E. Vestberg, Chairman and Chief Executive Officer of Verizon Communications Inc. (the Company), certify that:

(1) the report of the Company on Form 10-K for the annual period ending December 31, 2019 (the Report) fully complies with the requirements of section 13(a) of the Securities Exchange Act of 1934 (the Exchange Act); and

(2) the information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company as of the dates and for the periods referred to in the Report.

Date: February 21, 2020

/s/ Hans E. Vestberg

Hans E. Vestberg

Chairman and Chief Executive Officer

A signed original of this written statement required by Section 906, or other document authenticating, acknowledging, or otherwise adopting the signature that appears in typed form within the electronic version of this written statement required by Section 906, has been provided to Verizon Communications Inc. and will be retained by Verizon Communications Inc. and furnished to the Securities and Exchange Commission or its staff upon request.

EXHIBIT 32.2

CERTIFICATION OF CHIEF FINANCIAL OFFICER PURSUANT TO SECTION 906 OF THE SARBANES-OXLEY ACT OF 2002, PURSUANT TO SECTION 1350 OF CHAPTER 63 OF TITLE 18 OF THE UNITED STATES CODE

I, Matthew D. Ellis, Executive Vice President and Chief Financial Officer of Verizon Communications Inc. (the Company), certify that:

(1) the report of the Company on Form 10-K for the annual period ending December 31, 2019 (the Report) fully complies with the requirements of section 13(a) of the Securities Exchange Act of 1934 (the Exchange Act); and

(2) the information contained in the Report fairly presents, in all material respects, the financial condition and results of operations of the Company as of the dates and for the periods referred to in the Report.

Date: February 21, 2020

/s/ Matthew D. Ellis

Matthew D. Ellis

Executive Vice President and Chief Financial Officer

A signed original of this written statement required by Section 906, or other document authenticating, acknowledging, or otherwise adopting the signature that appears in typed form within the electronic version of this written statement required by Section 906, has been provided to Verizon Communications Inc. and will be retained by Verizon Communications Inc. and furnished to the Securities and Exchange Commission or its staff upon request.

Hvilken proces man normalt vil benytte ved godkendelse af miljøtoksin?

(MST, reference 1):

side 5: Hovedparten af befolkningen skal fortsat beskyttes (dvs.

hensyntagen til særligt udsatte skal også inddrages).
- Der skal tages specifikt hensyn til børn.
- Det fastholdes, at der højst anvendes en samlet usikkerhedsfaktor på 10.000, når resultater fra dyreforsøg overføres til mennesker.[1]
- Der accepteres fortsat en livstidsrisiko på 10^{-6}.

Denne vejledning indarbejder disse principper og gennemgår de metoder, som skal anvendes ved fastsættelse kvalitetskriterier for jord, luft og drikkevand med henblik på at beskytte sundheden[2].

1.1.2 Generelle principper for fastsættelse af kvalitetskriterier for kemikalier

Kvalitetskriterierne fastsættes på et niveau, hvor udsættelse gennem et helt liv ikke fører til skadevirkninger i befolkningen. De fastsættes på baggrund af den eksisterende viden og under hensyntagen til de mangler, der ligger i datagrundlaget. For at minimere risici for skadelig påvirkning af befolkningen indgår beskyttelse af særligt følsomme grupper fx børn, gravide, syge, ældre og svækkede ved fastsættelse af kvalitetskriterier. Viden om et kemisk stofs sundhedsskadelige egenskaber og om bestemte befolkningsgruppers særlige følsomhed er sjældent så eksakt, at der kan fastsættes et kvalitetskriterium, der præcist definerer skillelinien (hvis en sådan overhovedet findes) mellem et ufarligt og farligt niveau. Kvalitetskriterierne kan således ikke opfattes som en streg i sandet, hvor enhver overskridelse er farlig. Ved fastsættelse af kvalitetskriterier for kemikalier skal anvendes en forsigtighedstilgang, da målet er at sikre et højt beskyttelsesniveau for alle ved udsættelse over et helt liv.

---

1 : Her er NTP studiet og Ramazinistudiet særdeles relevant. NTP studiet bruger eksponeringer omkring (over og under) nuværende grænseværdier. Dermed bør grænseværdierne sænkes med en faktor 10.000. Rammazinistudiet viser effekter ved langt lavere værdier, altså burde eksponeringen være endnu lavere

Hvilke krav vil der normalt blive stillet til et miljøtoksin, førend det godkendes?

Hvilke tests vil man normalt udføre, forud for godkendelse?

Ref 1 (MST):
Det videnskabelige grundlag for fastsættelse af sundhedsmæssigt *baserede kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand består af en* ***farlighedsvurdering, en dosis- respons (effekt) vurdering (farlighedskarakterisering), samt en eksponeringsvurdering***[2]***.*** *Farlighedsvurderingen og farlighedskarakteriseringen tager udgangspunkt i undersøgelser af* ***det pågældende stofs toksikologiske effekter i mennesker og i dyr.***

På næste side gives en oversigt over det væsentligste indhold i de enkelte kapitler i miljøstyrelsens vejledning

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2 Der er ikke foretaget en sådan vurdering for RF-EMF i DK. Fra EU foreligger: ***1999/519/EC: Council Recommendation of 12 July 1999 on the limitation of exposure of the general public to electromagnetic fields (0 Hz to 300 GHz).***
https://op.europa.eu/en/publication-detail/-/publication/9509b04f-1df0-4221-bfa2-c7af77975556/language-en (ICNIRPs standard stort set pakket ind i EU anbefaling til medlemslandende, Ikke bindende - Ifølge denne recommendation er ICNIRPs grænseværdier et minimumskrav (for beskyttelse af mennesker) og der er ingen recommendation for beskyttelse af miljøet. Ifølge recomendationen er det op til det enkelte medlemdsland selv at følge med I forskningen på feltet og vurdere om der skal skrappere grænseværdier for beskyttelse af menneskers sundhed (punkt 19). Dette er IKKE sket i DK, jvf implementationsrapport og DK har ikke fastalgt gennemførselsforanstaltninger i forbindelse med basisrestriktionerne. Jeg vil mene, at Dk ikke lever op til recommendation III, V, VI og VII.

På side 17 i "First Implementation report 1999-2001":
"Denmark follows the ICNIRP recomendations and **has not implementedany legally binding measures** to protect the **public** against exposure to electromagnetic fields. Its Labour Inspectorate follows ICNIRP recomendations when evaluating exposure.
"https://ec.europa.eu/health/ph_determinants/environment/EMF/implement_rep_en.pdf

Second Implementation report 2002-2007: (2st version) https://op.europa.eu/en/publication-detail/-/publication/5dbcd92f-7849-4017-8cee-742f40ff8143
s 4-5 (og Skema 2) : *De fleste medlemsstater har gennemført henstillingen, og nogle har indført bindende foran-staltninger for at begrænse offentlighedens eksponering for elektromagnetiske felter..... Imidlertid er der i Cypern, Danmark, Tyskland, Irland, Litauen, Slovenien og Slovakiet ikke fastlagt gennemførselsforanstaltninger i forbindelse med basisrestriktioner.*

Ifølge 1999/519/EC anbefales det at evaluere hvert 3. år (se side 3 stk VII.). Så der burde være en rapport hvert 3. år fra 1999-2020 – men vi har kun kunnet finde de to.

Se desuden ref 4: Hollandsk regeringsrapport om EMF Grænseværdier i Europa:
Comparison of international policies on electromagnetic fields 2018.pdf

- I kapitel 2 omtales *Datagrundlaget* som anvendes som udgangspunkt for arbejdet. Data hentes primært fra internationale og nationale dokumenter, via litteratursøgning i internationale databaser, samt fra originalartikler. [3]
- Kapitel 3 behandler de faglige metoder, der anvendes i forbindelse med farlighedsvurderingen og farlighedskarakteriseringen. Dosis-effekt og dosis-responssammenhænge og udpegning af NOAEL (No Observed Adverse Effect Level) og LOAEL (Lowest Observed Adverse Effect Level) beskrives.
- I kapitel 4 beskrives hvordan farlighedskarakteriseringen udmunder i udpegning af en kritisk effekt,[4] som danner udgangspunkt for fastsættelse af en *tolerabel daglig indtagelse,TDI*. Her omtales, hvordan anvendelsen af usikkerhedsfaktorer indgår i beregningerne.
- I kapitel 5 omtales, hvordan TDI beregnes for kræftfremkaldende stoffer uden tærskelværdi.[5] Risikoniveauet for TDI-værdien defineres, og der gives retningslinier medhensyn til valg af metode til, hvordan beregningen af dette risikoniveau foretages.

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3 Kapitel 2: "*kvalitetetskriterierne vil som oftest være baseret på viden opnået fra dyreforsøg med mere veldefineret udsættelse eller in vitro data*". - For EMF igenoreres dyrestudierne, påstås irrelevante for mennesker!

"*Ved fastsættelsen af kvalitetskriterier anvendes der internationalt anerkendte principper. I denne forbindelse skal fremhæves de principper og metoder, der er beskrevet i to publikationer af WHO/ IPCS[6,7] om udarbejdelsen af vejledende grænseværdier og risikovurdering af kemisk udsættelse "*

"Det anbefales generelt, at data, der skal danne udgangspunkt for beregning af kvalitetskriteriet, altid hjemskaffes som originallitteratur til vurdering af den konkrete undersøgelses kvalitet og relevans."

4 Der er ikke fastsat nogen form for dosis-effekt sammenhænge for hverken mennesker eller miljø. Der vil altid være huller I viden, men der er virkelig mange eksperimentelle dyrestudier (insekter og en lang række hvirveldyr) af forskellig art som man kunne anvende til at undersøge dette aspekt for RF-EMR. Ref 1, s20: En længerevarende dyreeksperimentel undersøgelse vil således kunne give viden om forskellige typer effekter ved forskellige eksponeringsniveauer (dosis-effekt), og om hvor lang tid det tager, før de optræder i forhold til eksponeringen. Bemærk side 24 vedr. Artsspecifikke effekter. For FR-EMR er der observeret effekter I så mange dyrearter, at man kan udelukke at effekterne af EMR kun ses i én dyreart. Derfor må dyreforsøgene vurderes som repræsentative for mennesker.

5 Dette er relevant ift EMF, idet det også gælder agens i kategori 2B, jvf side 36 i dokumentet. Bemærk iøvrigt hvad der skrives om genotoksiske agens. Da der er mindst 40 studier som viser DNA skader er dette ikke uvæsentligt.

Supplerende bemærkninger:

- Det største problem er at de definerede normer går på kemiske miljøgifte. Men det er relevant til sammenligning af fremdrage procedurerne for kemiske stoffer i både DK (MST) og EU. De er meget velbeskrevne.

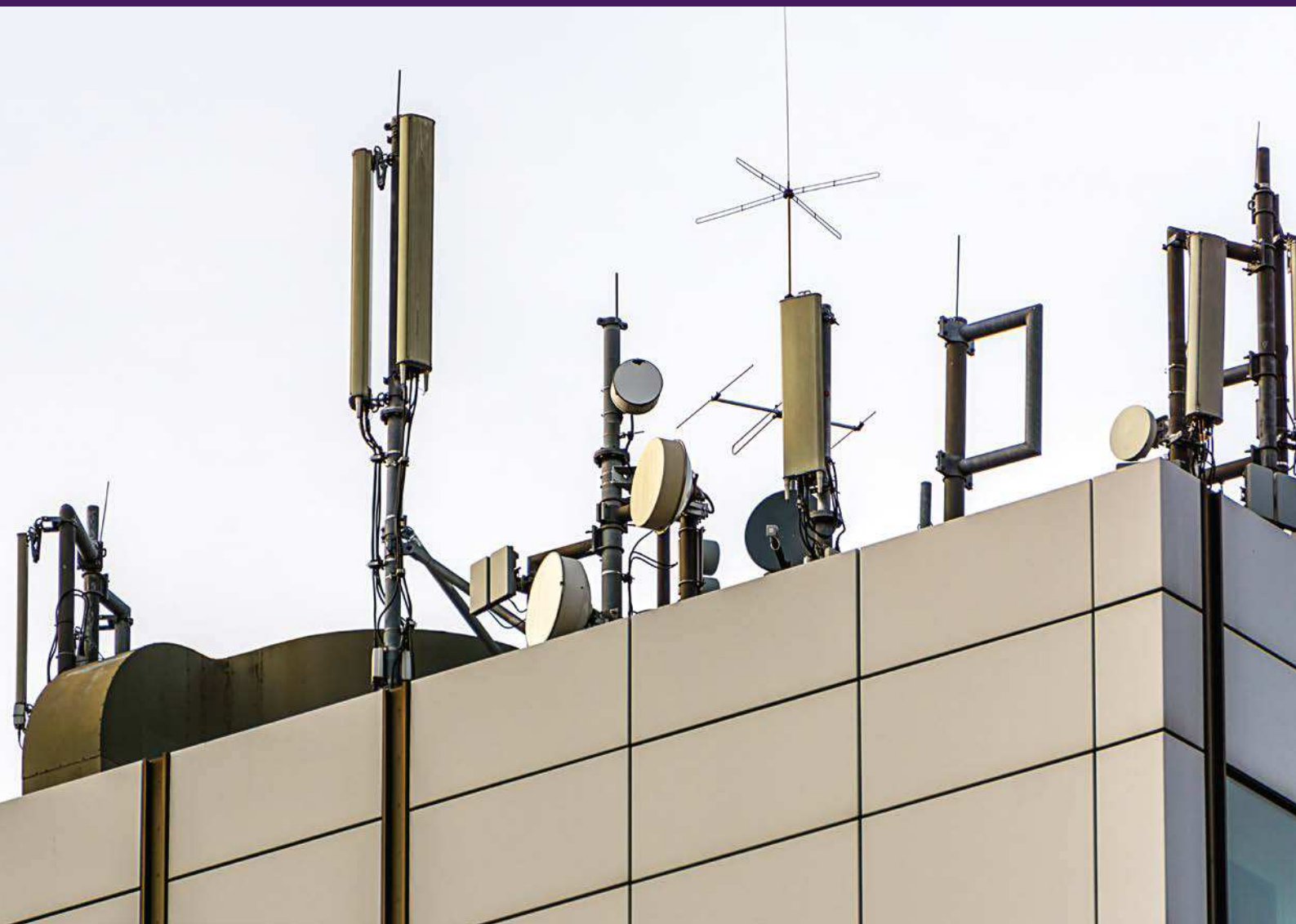
- Desuden har EU kommissionens " Scientific Committee on Health, Environmental and Emerging Risks " =SCHEER defineret EMR som muligt miljøtoksin (fysisk hazard), som foreløbigt vurderes af være af størst mulig betydning for ikke bare menneskers sundhed men også for økosystemer og andre arter (Ref 2, punkt 4.4): Højeste ranking for "urgency", højeste ranking for "scale" (omfang), og højeste rank for interactions (interaktion med ikke bare mennsker men også økosystemer og andre arter). Desuden tilføjes: This concern is more related to the change to 5G rather than a completely new concern. Preliminary Estimation of importance: RF-EMR gives højest mulig rank

- Endelig har EEA vurderet at forsigtighedsprincippet bør tages i anvendelse overfor RF EMR (Ref 3, nederst side 38 til side 39): It is therefore very important that large scale emerging technologies, such as biotechnologies, nanotechnologies and information **and communication technologies**, apply the precautionary principle based on the experiences and lessons learned from these and other case studies.
- Det afviger fra EUs normale procedurer at udpege en privat organisation til at fastsætte grænseværdier. Da der er tale om et "large scale ermerging" miljøtoksin burde EU burde rådgivning fra EEA højere.
- Samtidig siger EU også at det er op til de enkelte lande at vurdere om det er nødvendigt med strammere grænseværdier end dem ICNIRP fastsætter (- se fodnote 2 ovenfor) – men dette vedrører kun eksponering af mennsker. Der er INGEN miljøvurdering overhovedet, af effekter på selve miljøet.

National Institute for Public Health
and the Environment
Ministry of Health, Welfare and Sport

Comparison of *international* policies on *electromagnetic fields*

(power frequency and radiofrequency fields)



Comparison of international policies on electromagnetic fields *(power frequency and radiofrequency fields)*

This document is an update of an earlier overview from May 2011 (RIVM 118/2011). It was prepared as part of a research project commissioned by the Ministry of Infrastructure and Water Management and the Ministry of Social Affairs and Employment of the Netherlands. The information that forms the basis for this summary was obtained from searches of governmental and scientific websites, scientific publications, policy summaries by other organisations and personal contacts with experts in the countries in question. The information was last updated in the period from January to July 2017 [1].

Introduction

Time-varying electric, magnetic and electromagnetic fields (EMF) are generated by moving electric charges and by variable electric fields such as those generated near a conductor for alternating current. Power frequency EMF are generated in the production, transport, distribution and use of electricity. The frequency of alternating current and the resulting EMF is 50 hertz in Africa, most of Asia, Australia, Europe and part of South America and 60 hertz in the remainder of America, the Philippines, Korea, Saudi-Arabia and part of Japan. Radiofrequency EMF are generated, among others, by mobile telecommunication systems, broadcasting transmitters, radar installations, microwave ovens and dryers, plastic welders, certain medical applications and equipment for electronic article surveillance and identification.

In 1999, the Council of the European Union (EU) published a Recommendation (1999/519/EC, further called 'EU recommendation') on the limitation of exposure of the general public to EMF (0 hertz to 300 gigahertz). It contains basic restrictions for the induced electric fields and currents and the absorbed power in the body and reference levels for the strength of EMF outside the body (for values at selected frequencies, see **Table 1**). The limits in the EU recommendation are derived from the 1998 guidelines for limiting exposure to EMF by the International Commission on Non-Ionizing Radiation Protection (ICNIRP). ICNIRP has issued new guidelines for EMF with frequencies between 1 hertz and 100 kilohertz in 2010, and for frequencies between 0 and 1 hertz in 2014, but these have not yet led to changes in the EU recommendation.

In 2013, the European Parliament and the Council of the EU issued a directive (2013/35/EU, further called 'EU directive') on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (EMF). It contains exposure limit values for the induced electric fields and the absorbed power in the body and action levels for the strength of EMF outside the body (for values at selected frequencies, see **Table 2**). The limits for static and low frequency fields in the EU directive are derived from the 2009 and 2010 ICNIRP guidelines for limiting exposure to static and low frequency time-varying EMF. The limits for radiofrequency fields are derived from the 1998 ICNIRP guidelines. ICNIRP has reconfirmed the validity of its 1998 guidelines for EMF with frequencies between 100 kilohertz and 300 gigahertz in a 2009 statement. For the sake of consistency, the terminology of the EU recommendation and EU directive is also used for equivalent public and occupational exposure limits in national legislation in the present summary, regardless of whether these are derived directly from ICNIRP or from other sources.

The European Parliament and Council of the EU have also issued directives on the marketing of low voltage electrical equipment (2014/35/EU) and radio equipment (2014/53/EU), which require that such equipment does not endanger the health or safety of persons. The European Committee for Electrotechnical Standardisation (CENELEC), in liaison with the European Telecommunications Standards Institute (ETSI), has developed harmonised standards for measurement and calculation of EMF exposure which can be used to demonstrate that this requirement is met.

[1] **Disclaimer:** The author has taken care to obtain correct and up-to-date information from relevant websites, policy documents and experts in the countries in question. However, no rights can be deduced from any of the information in this document. For further information and corrections, please contact Dr. R. Stam, National Institute for Public Health and the Environment, the Netherlands. E-mail: *rianne.stam@rivm.nl*

Apart from ICNIRP, influential guidelines on the protection against risks of EMF have also been published by the Institute of Electrical and Electronics Engineers (IEEE), for both exposure of the general public and controlled environments (occupational exposure). For power frequency fields, the IEEE basic restrictions for induced electric fields are similar to those of ICNIRP and EU for exposure of the head (brain) but less strict than ICNIRP for exposure of the rest of the body. For radiofrequency fields, IEEE basic restrictions are the same as those of ICNIRP and EU. The reference levels of IEEE are less strict than those of ICNIRP and EU (for radiofrequency fields only at some frequencies). Differences in the limits between different guidelines are mainly caused by differences in the dosimetric models of the human body and in the use of safety factors. The limits advised by IEEE are used in national EMF legislation of some countries outside the EU and referred to in a safety standard of the North Atlantic Treaty Organization (NATO).

Exposure of the general public, power frequency fields

European Union

Because the EU recommendation is not legally binding, EMF policy in member states can be divided into three different approaches. Details on limits at selected frequencies per member state can be found in **Table 1** and a visual overview in **Figure 1**. In the **first group** of member states the EU recommendation has been transposed in binding national legislation or national policy. This means that the basic restrictions and reference levels must be applied. EU member states in this group are the *Czech Republic, Estonia, France, Greece, Hungary, Ireland, Luxemburg, Portugal* and *Romania*. In the *Czech Republic*, the reference levels differ from the EU recommendation, but the basic restrictions are the same. In *France* the limits only apply to new or modified installations. In *Germany* and *Slovakia* the reference levels in the EU recommendation are applied as *de facto* exposure limits, without reference to basic restrictions.

In the **second group** of member states, the national limits based on the EU recommendation or ICNIRP are not binding, there are more lenient limits or there is no regulation. However, it may be that the authorities or grid companies apply the limits in the EU recommendation in practice. EU member states in this group are *Austria, Cyprus, Denmark, Finland, Latvia, Malta, the Netherlands, Spain, Sweden* and *the United Kingdom*.

Whether or not they have legally binding limits on the strength of power frequency fields, in some of the EU member states in the first and second group a precautionary policy has been advised by the government or voluntarily agreed to by the electricity supply sector to limit the exposure of members of the general population to power frequency magnetic fields. Alternatively, the legislation contains an obligation to minimise fields as far as this can be done with reasonable cost and with reasonable consequences. The motivation is either the epidemiological evidence for a possibly increased risk of childhood leukaemia in children who live near overhead power lines, or a more general argument to keep fields as low as reasonably possible in the light of scientific uncertainty. These precautionary policies in addition to formal legislation are as follows:

First group

France: A ministerial recommendation advises the Prefectures to avoid as far as possible the creation of new hospitals, maternity wards and childcare facilities near power lines, cables, transformers and bus bars where children are exposed to a magnetic field stronger than 1 microtesla. For new or modified electricity infrastructure, the grid operator usually tries to avoid as much as possible the creation of new electricity infrastructure near such locations when planning a new grid development. The grid operator has the legal obligation to monitor the strength of EMF near power lines in urbanised areas. Citizens can also request information about the strength of EMF from local power lines via their mayor.

Germany: National legislation requires that all possibilities to minimise EMF should be exhausted in accordance with the technical state of the art when creating or substantially modifying direct current and alternating current facilities with voltages greater than 1 kilovolt. High-voltage power lines for alternating current on a newly planned route may not pass over buildings meant for the long-term stay of people. The obligation to minimise EMF only applies to locations with homes, hospitals, schools, childcare facilities, playgrounds or any other location not exclusively meant for the temporary stay of people. Minimisation measures need to be proportional with regard to cost, functionality, or negative effects on the environment, well-being and occupational safety.

Luxemburg: There is a ministerial recommendation not to create any new living spaces in the immediate vicinity of overhead power lines (within 20 metres for 65 kilovolt lines and 30 metres for 100 to 220 kilovolt lines).

Second group

Austria: Although precautionary limits are not formally advised, the panel of experts appointed by the relevant authority for new electricity lines requiring environmental impact assessment usually require compliance with a maximum magnetic flux density of 1 microtesla (1% of the reference level in the EU recommendation), derived from Swiss legislation.

Denmark: The Danish Health Authority (Sundhetsstyrelsen) recommended in 1993 not to build new homes or children's institutions close to power lines or new power lines close to homes or children's institutions. The exact distance was left to pragmatic considerations. The Danish electricity sector has published guidelines for situations where measures at reasonable cost to reduce the magnetic field must be investigated. Like the Danish Health Authority's advice, the guidelines apply only to new developments.

Finland: The Radiation safety authority (STUK) recommends avoiding the construction of permanent residences in areas where the magnetic flux density continuously exceeds the level of approximately 0.4 microtesla.

Netherlands: A ministerial recommendation advises local authorities and grid companies to avoid as far as reasonably possible creating new situations with long-term stay of children in areas around overhead high-voltage power lines with an annually averaged magnetic flux density greater than 0.4 microtesla. The advice applies when making spatial plans and determining the trajectory of overhead high-voltage power lines, or when changing existing plans or existing overhead high-voltage power lines. For existing situations, the reference level in the EU recommendation should apply.

United Kingdom: In response to the conclusions of a national stakeholders' dialogue, the government noted that ICNIRP exposure guidelines in place in the United Kingdom remain appropriate. It also supports the implementation of low-cost options such as optimal phasing to reduce the magnetic field of overhead power lines, but considers additional exposure reduction by creating exclusion zones between homes and power lines to be disproportionate in the light of the evidence on the potential health risks. The government also supported exploring the reinforcement of best practice for wiring of distribution circuits and providing consistent, helpful and proportionate public health messages to raise awareness.

In the **third group** of member states, there are stricter basic restrictions and/or reference levels, based on the precautionary principle or due to public pressure. These stricter reference levels are often applied as a *de facto* exposure limit that may not be exceeded. Since there is a great diversity in particular rules and limits, a summary is given per member state:

Belgium: In Belgium, the limitation of EMF exposure of the general population is a matter for the three devolved regions. In Flanders, a ministerial recommendation for the planning of new power lines states that passing over schools and childcare centres should be avoided and passing over homes kept to a minimum. New schools and childcare centres should not be placed in the magnetic field zone with year-averaged exposure greater than 0.4 microtesla (0.4% of the reference level in de EU recommendation). In addition, an indoor environment decree requires those responsible for building or managing homes and public buildings to keep exposure to power frequency magnetic fields below 10 microtesla (10% of the reference level in de EU recommendation) and advises them to strive for a 'quality aim' of 0.2 microtesla (0.2% of the reference level in de EU recommendation). In the Brussels region, a ministerial instruction for environmental permits requires that the magnetic field in places near newly installed transformers where children under 15 may stay is kept below a 24-hour average of 0.4 microtesla. Wallonia does not have a precautionary policy for power frequency magnetic fields, but applies the limits in the EU recommendation to transformers.

Bulgaria: Minimal distances between homes and power lines or substations are in force depending on voltage. There are no other limits for exposure of the general public to power frequency EMF except for limits on emission by video screens. At a distance of 50 centimetres from video screens, the limit is 0.5% of the reference level in the EU recommendation for electric field strength and 0.25% for magnetic flux density.

Croatia: For public spaces in general, limits for the electric and magnetic field identical to the reference levels in the EU recommendation may not be exceeded. For 'sensitive areas' (homes, offices, schools, playgrounds, kindergartens, maternity wards, hospitals, homes for the elderly and disabled and tourist accommodations), the limits for the electric and magnetic field are 40% of the reference levels in the EU recommendation.

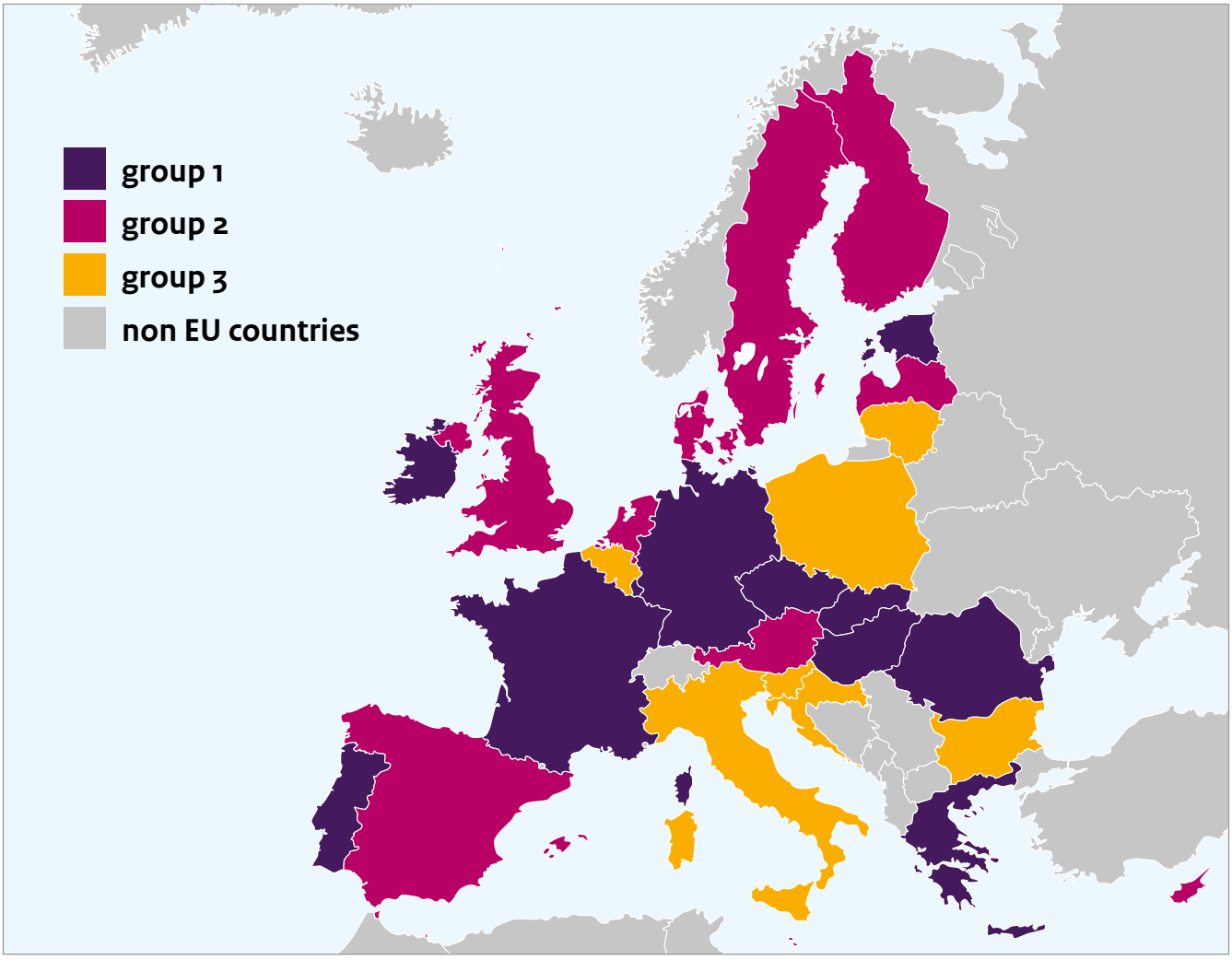
Italy: For all low frequency sources other than power lines, the reference levels and basic restrictions in the EU recommendation apply. For 50-hertz electric and magnetic fields from power lines and associated installations, the reference level in the EU recommendation may not be exceeded. In addition, a precautionary 'attention value' and 'quality goal' apply to 24-hour median exposure in homes, playgrounds, schools and places where people can stay for more than four hours. The 'attention value' of 10% of the EU reference level for magnetic flux density applies to existing situations. The 'quality goal' of 3% of the EU reference level for magnetic flux density applies to new situations. An even stricter limit for magnetic flux density (0.2% of the reference level) was adopted in three regions before the federal law came into force. This too applies to power lines near homes, schools and other places where people may stay for more than 4 hours per day.

Lithuania: A limit of 10% (electric field) or 20% (magnetic field) of the reference level in the EU recommendation applies inside residential and public buildings. A limit of 20% (electric field) or 40% (magnetic field) of the reference level in the EU recommendation applies to the living environment outside buildings.

Poland: A limit of 20% (electric field) or 75% (magnetic field) of the reference level in the EU recommendation applies to residential areas.

Slovenia: A limit of 10% of the reference level in the EU recommendation applies to electric and magnetic fields from new or modified sources near homes, schools, kindergartens, hospitals, sanatoria, playgrounds, parks, recreational areas, public buildings and buildings with a tourist destination. For other locations, limits equal to the reference levels in the EU recommendation apply.

Figure 1 Overview of limits for exposure of the general population to power frequency EMF in the EU.
Group 1 (purple): legal limits derived from EU recommendation, precautionary policy in some countries;
Group 2 (pink): no legal limits or limits less strict than in EU recommendation, precautionary policy in some countries;
Group 3 (yellow): stricter limits than in EU recommendation.



Other countries

Different approaches to limiting exposure to power frequency EMF also exist in industrialised countries outside Europe. Seven examples are given below and further details on exposure limits can be found in **Table 1**.

Australia: No official government regulation or guidelines for exposure of the general population to EMF with frequencies lower than 3 kilohertz are currently in place. The Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) has stated that the ICNIRP low frequency guidelines are consistent with its interpretation of the scientific basis for the protection of the general public from exposure to low frequency EMF. The grid operators have a 'prudent avoidance' policy to take reasonable steps to limit field exposures from new facilities (overhead power lines, underground cables and substations) at no cost or very low cost while not unduly compromising other issues such as worker safety, site availability, reliability and environmental impact.

China: A national standard for protection of the general population under the Environmental Protection Law sets limits for environmental exposure to EMF, but does not apply to household appliances. The limits for power frequency magnetic fields equal the reference levels in the EU recommendation up to 800 hertz, but are lower for frequencies greater than 800 hertz. For electric fields the limits are lower than the reference levels in the EU recommendation for all frequencies. The standard also cites the precautionary principle and encourages facility and equipment owners to take effective measures to reduce public exposure.

India: There is no national regulation of the strength of power frequency EMF. Technical standards for the electricity supply sector give minimal distances to buildings, but these measures are related to electrical safety.

Japan: Ministerial regulations for technical standards of electrical equipment and railways limit power frequency magnetic fields to the reference level in the 2010 ICNIRP guidelines (200 microtesla at 50 hertz). The limit for power frequency electric fields (3000 volt per metre at 50 hertz) is lower than that in the ICNIRP guidelines and EU recommendation and meant to prevent electric shocks.

Russia: General rules for the protection are set in a 1999 framework law. Exposure limits for specific frequency ranges are set in so-called 'Hygienic-epidemiological standards'. The public exposure limit for power frequency magnetic fields is 5% of the reference level in the EU recommendation for living quarters, preschool, children's, general and medical institutions; 10% of the reference level in the EU recommendation for non-residential parts of residential buildings and in public and administrative buildings; 20% of the reference level in the EU recommendation in inhabited areas outside residential built-up areas; equal to the reference level in the EU recommendation in non-populated areas with occasional stay of people.

Switzerland: An Ordinance relating to Protection from Non-Ionising Radiation has been in force since 2000. Exposure limits identical to the reference levels in the EU recommendation apply to all areas accessible to the public. A stricter, precautionary limit on magnetic flux density of 1% of the reference level in the EU recommendation applies at so called places of sensitive use (for example apartments, schools, children's playgrounds) to the following classes of installations, unless the owner can prove that all technically possible and economically acceptable measures to reduce exposure have been taken: new high voltage power lines (overhead and cables); significant modification of existing high voltage power lines; existing and new transformers and substations. For existing high voltage power lines, the phase order has to be optimised when the precautionary limit on magnetic flux density is exceeded.

United States: No federal legislation is in force. In some states (Colorado, Connecticut, Hawaii, Maryland, Ohio), variations on the 'prudent avoidance' principle have been adopted. This means that exposure of the public to EMF of 60 hertz must be limited at reasonable cost. In other states, fixed limits for the electric or magnetic field of power lines are set, varying from 20% to 240% of the reference level in the EU recommendation (Florida, Minnesota, Montana, New Jersey, New York, Oregon).

Exposure of the general public, radiofrequency fields

European Union

Because the EU recommendation is not legally binding, EMF policy in member states can be divided into three different approaches. Details on exposure limits per member state can be found in **Table 1** and a visual overview in **Figure 2**. In the **first group** of member states the EU recommendation has been transposed in binding national legislation or national policy. This means that the basic restrictions and reference levels must be applied. Member states in this group are *Cyprus, Czech Republic, Estonia, Finland, France, Hungary, Ireland, Malta, Portugal, Romania* and *Spain.* In *Germany* and *Slovakia* the reference levels have become *de facto* exposure limits. In *France* there is an additional legal obligation to provide information on options for exposure reduction when selling or promoting a mobile phone and to provide citizens with measurement results for the strength of radiofrequency EMF in their homes or in public buildings.

In the **second group** of member states, the national limits based on the EU recommendation or ICNIRP are not binding, there are more lenient limits or there is no regulation. Member states in this group are *Austria, Denmark, Latvia, the Netherlands, Sweden* and *the United Kingdom*. In some countries, for example the Netherlands and the United Kingdom, telecommunication companies have signed up to a voluntary code to respect the limits in the EU recommendation in places accessible to the public. In the United Kingdom the national planning policy framework for local government also requires that applications for expansion of base stations certify that these limits will not be exceeded.

In the **third group** of member states, there are stricter reference levels and/or basic restrictions based on the precautionary principle and/or due to public pressure. The limits chosen are sometimes based on the principle 'as low as reasonably achievable without endangering service'. One practical choice for stricter limits can be to adopt the lower limit for interference in the European standards for electromagnetic compatibility (for example in Belgium). In other countries the reasons for particular limits are unclear or arbitrary (for example in Greece and Italy). In some member states the stricter reference levels are applied as exposure limits that may not be exceeded. Since there is a great diversity in particular rules and limits, a summary is given per member state:

Belgium: The advertising and sale of mobile phones specially designed for children younger than 7 years is prohibited. For all other phones, information must be provided on specific absorption rate and possibilities to lower exposure. Regulation of exposure limits in Belgium is a matter for the three devolved regions. In Flanders, the limit for electrical field strength per antenna for telecommunication is 7% of the reference level in the EU recommendation in places of stay like homes, schools, rest homes and nurseries. The maximum exposure in all publicly accessible places is 50% of the reference level for frequencies between 10 megahertz and 10 gigahertz. The Brussels Region limits total exposure in residences for frequencies between 100 kilohertz and 300 gigahertz to a power density of 2% of the reference level in the EU recommendation (corresponding with 15% for the electric field strength). For the same frequency range, Wallonia sets a fixed limit for the electrical field strength per antenna in residences which is 7% of the reference level at 900 megahertz.

Bulgaria: Fixed limits for electrical field strength and power density are set. Their percentage of the reference levels in the EU recommendation decreases with frequency. For power density it is 2% at 900 megahertz and less than 2% for higher frequencies.

Croatia: For public spaces in general, fixed limits for the electric and magnetic fields are applied which are 95% of the reference levels in the EU recommendation (90% for power density). For 'sensitive areas' (homes, offices, schools, playgrounds, kindergartens, maternity wards, hospitals, homes for the elderly and disabled and tourist accommodations), the limits for the electric and magnetic field are 40% of the reference levels in the EU recommendation (16% for power density).

Greece: The law on electronic communications sets basic restrictions of 70% of those in the EU recommendation and 60% when antenna stations are located closer than 300 metres from the property boundaries of schools, kindergartens, hospitals or eldercare facilities. Installation of mobile phone antenna stations is not allowed within the property boundaries of aforementioned facilities. Reference levels calculated from these two basic restrictions are 84% and 77% of the reference levels in the EU recommendation (70% and 60% for power density).

Italy: For EMF from high frequency sources other than fixed systems for telecommunication and radio or TV broadcasting, the reference levels and basic restrictions in the EU recommendation apply. For EMF from fixed systems for telecommunication and radio or TV broadcasting, there are exposure limits in terms of the strength of environmental EMF that may not be exceeded. In contrast with the limits in the EU recommendation, these are constant (not frequency dependent) between 3 megahertz and 3 gigahertz.

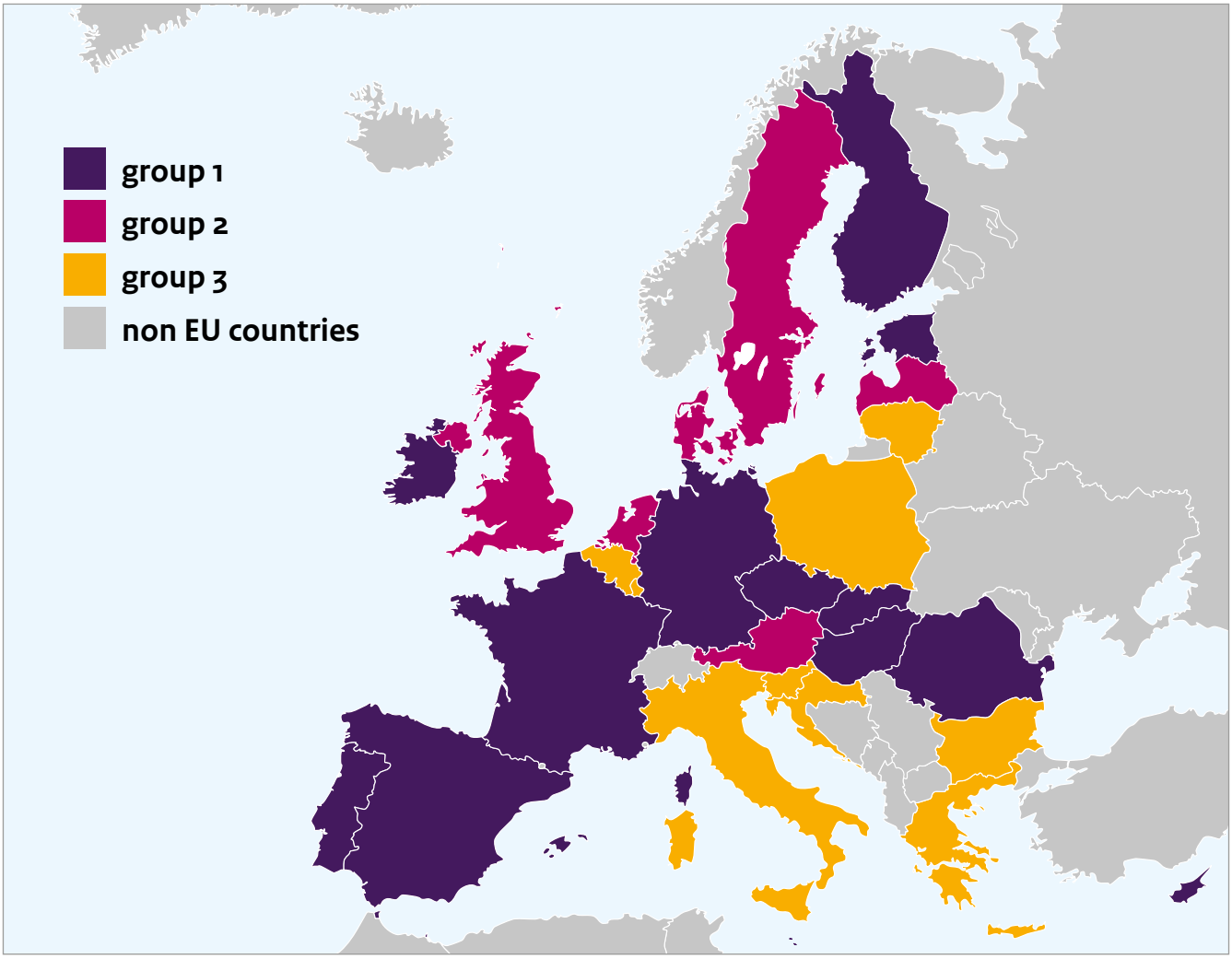
The exposure limit for electric field strength at 900 megahertz is 49% of the reference level in the EU recommendation (22% for power density). In homes, schools, playgrounds and places where people may stay for longer than four hours, the 'attention value' for electric field strength is 15% of the reference level in the EU recommendation at 900 megahertz (2% for power density). The 'quality goal' for highly frequented outdoor areas is identical to the attention value.

Lithuania: There are limits for EMF with frequencies between 10 megahertz and 300 gigahertz inside and surrounding residential and public buildings which may not be exceeded and are lower than the reference levels in the EU recommendation. The percentage varies with frequency, but for power density the limit is 10% of the EU reference level at 900 megahertz.

Luxemburg: Precautionary policy is applied to mobile telephony through a law on classified locations and technical standards. These set a fixed exposure limit for the electrical field strength per radiating element for antennas with a power of 100 watt and higher which is 7% of the reference level in the EU recommendation at 900 megahertz. The limit for other antennas and for the total number of antenna elements in one location equals the reference level in the EU recommendation.

Poland: In locations that are accessible to the public, frequency-dependent exposure limits lower than the reference levels in the EU recommendation are set for electrical field strength and power density. At 900 megahertz the limit for electrical field strength is 17% of the reference level in the EU recommendation (2% for power density).

Figure 2 Overview of limits for exposure of the general population to radiofrequency EMF in the EU. Group 1 (purple): legal limits derived from EU recommendation; Group 2 (pink): no legal limits or limits less strict than in EU recommendation; Group 3 (yellow): stricter limits than in EU recommendation.



Slovenia: For frequencies higher than 10 kilohertz, exposure limits for electric and magnetic field strength of 31% of the reference levels in the EU recommendation (10% for power density) apply in 'sensitive areas' such as homes, schools and hospitals. In all other locations the reference levels in the EU recommendation are applied as *de facto* exposure limits that may not be exceeded.

Other countries

Industrialised countries outside the EU also have different ways of limiting exposure of the public to radiofrequency EMF. Seven examples are given below and further details on exposure limits can be found in **Table 1**.

Australia: The mandatory basic restrictions and reference levels in the national radiation protection and radiocommunication standards are identical to those in the EU recommendation.

China: A national standard for protection of the general population under the Environmental Protection Law sets limits for environmental exposure to EMF, but does not apply to wireless communication terminal equipment. The limits are lower than the reference levels in the EU recommendation, but the percentage varies with frequency. At 900 megahertz the limit for electric field strength is 29% of the reference level in the EU recommendation (9% for power density). The standard also cites the precautionary principle and encourages facility and equipment owners to take effective measures to reduce public exposure. The basic restrictions for mobile phones in a separate standard are identical to those in the EU recommendation.

India: A ministerial memorandum amending the Unified Access Service License sets limits on exposure of the general public to EMF from telecommunication base stations. The limit is 33% of the reference levels in the EU recommendation for electric and magnetic field strength and 10 % for power density. Government-approved interministerial committee recommendations set a limit on the specific absorption rate for mobile handsets which is 80% of the basic restriction for local exposure of the head in the EU recommendation.

Japan: The ministerial radiofrequency radiation protection guidelines for human exposure to EMF contain a mandatory basic restriction for mobile phones which is identical to that in the EU recommendation. The guidelines also contain mandatory basic restrictions with reference levels for the strength of EMF from mobile phone base stations, which are almost identical to the reference levels in the EU recommendation.

Russia: General conditions for protection of the population are set in a 1999 framework law. Limits for specific frequency ranges are set in subsequent 'Hygienic-epidemiological requirements'. The exposure limit for power density for EMF with frequencies between 300 megahertz and 300 gigahertz in and around residential buildings and inside public and industrial premises is 2% of the reference level in the EU recommendation. The reason is to prevent biological effects that are not generally seen as a health risk in Western countries. There is no basic restriction in terms of specific absorption rate, but there is a limit on the plain wave power density of mobile phones which is 22% of the reference level in the EU recommendation.

Switzerland: An Ordinance relating to Non-Ionising Radiation is in force since 2000. Mandatory exposure limits identical to the reference levels in the EU recommendation apply in all areas accessible to the public. A stricter, precautionary limit for the electric field strength of approximately 10 % of the reference level in the EU Recommendation applies at so called places of sensitive use (for example apartments, schools, children's playgrounds) near mobile phone antennae, broadcasting and radar installations.

United States: The basic restriction for whole body exposure in federal legislation for radio transmitters is identical to that in the EU recommendation. However, the reference levels are higher because a different model is used to calculate them. At 900 megahertz the difference is 15% and 14% for the electric and magnetic field strength respectively (33% for power density). The reference levels are applied as *de facto* exposure limits for non-portable devices. For portable devices close to the body, the mandatory basic restriction for local exposure of all parts of the body except the extremities is 80% of the basic restriction for head and trunk in the EU recommendation. The basic restriction for the extremities (hands, wrists, ankles, feet, outer ears) is identical to the basic restriction for limbs in the EU recommendation.

In addition to the above legal obligations, in Australia, Austria, Belgium, Cyprus, Denmark, Finland, France, Germany, Greece, Ireland, India, Italy, Luxemburg, the Netherlands, Spain, Sweden, Russia, Switzerland, the United Kingdom and the United States the government or national scientific organisations have published advice on how to reduce exposure to radiofrequency EMF from mobile phones, such as limiting calling time, using earpieces or speakers, not holding the phone close to the body, avoiding calls in areas with poor reception and texting instead of calling.

Occupational exposure, Power frequency fields

European Union

In all member states of the EU, protection or workers against the risks of EMF is regulated by national legislation based on directive 2013/35/EU. The directive contains general rules and appendices with exposure limits. The directive distinguishes three layers of action levels for low frequency magnetic fields: low action levels related to sensory effects exposure limit values (equivalent to ICNIRP's 2010 basic restrictions for the central nervous system) and high action levels and limb action levels related to health effects exposure limit values (equivalent to ICNIRP's 2010 basic restrictions for the peripheral nervous system). When the action levels are exceeded, this is an indication that the related exposure limit values could be exceeded.

The directive sets minimum requirements, but allows member states to set stricter rules or limits, which are detailed below and in **Table 2**. The directive also gives member states the possibility to apply a conditional exemption from the exposure limits (but not from the general rules) for worker exposure related to magnetic resonance imaging (MRI) for patients in the health sector, to apply a different but equivalent or more specific protection system for military personnel and to allow the exposure limits to be temporarily exceeded under certain conditions for specific sectors or activities in duly justified circumstances. Details of whether and how individual member states have applied these possibilities for exemptions can be found in **Table 2**. Two member states have action levels and/or exposure limit values that differ from those in the EU directive:

Czech Republic: For EMF with frequencies from 1 hertz to 10 megahertz, there is only one action level, which is equivalent to the low action level in the EU directive. Nevertheless, there are still two levels of exposure limit values for the internal electric field, the higher for exposure of the head and the lower for exposure of the rest of the body.

Poland: For EMF with frequencies between 0 and 300 gigahertz, there are six sets of action levels delimiting a 'danger zone', 'threat zone' and 'intermediate zone', action levels for local exposure of extremities and ancillary action levels for peak levels for modulated fields. The two levels of exposure limit values for the internal electric field are identical to the sensory effects and health effects exposure limit values in the EU directive, but have been extended to frequencies between 0 and 1 hertz based on the 2014 ICNIRP guidelines for magnetic fields below 1 hertz.

Other countries

Australia: There are radiation protection regulations which are only applicable to Commonwealth employees and set limits on occupational exposure to EMF from 'controlled apparatus', that is, specified categories of devices that could cause EMF which exceed these limits (for example induction heaters). Its reference levels and basic restrictions are identical to those in the 2009 and 2010 ICNIRP guidelines on static and low frequency fields. They therefore have the same basis as the limits in the EU directive, but apply to a narrower range of devices. The reference levels equal the low action levels in the EU directive. For non-Commonwealth employees there is no official government regulation but the ARPANSA advice on the ICNIRP guidelines also applies to workers.

China: The national standard with occupational exposure limits for physical agents in the workplace has a limit of 5 kilovolt per metre for exposure to power frequency electric fields. There are no occupational limits for power frequency magnetic fields.

India: There are no legally binding limits on occupational exposure to power frequency EMF. Protection of workers would therefore fall under general health and safety legislation such as the Factories Act.

Japan: There are no legally binding limits on occupational exposure to power frequency EMF. The Japan Society for Occupational Health has recommended occupational exposure limits for EMF in terms of the strength of external electric and magnetic field, which are identical to the low action levels in the EU directive.

Russia: A national standard sets limits for power frequency magnetic fields, which depend on the exposure duration. For exposures shorter than 1 hour, the limit for whole body exposure is 33% of the high action level, but for 8 hours it is 2% of the high action level in the EU directive. The limit for 'limbs only' exposure is four to ten times higher than the limit for whole body exposure.

Switzerland: The federal law on accident insurance gives general rules to prevent illness caused by physical agents. The national accident insurer has specified that exposure limits identical to the occupational reference levels in the 1998 ICNIRP guidelines may not be exceeded. For power frequency, the limit is 50% of the low action level in the EU directive for the magnetic field and 100% for the electric field.

United States: There are no legal limits for occupational exposure to power frequency EMF. The American Conference of Governmental Industrial Hygienists has recommended 'threshold limit values' which are 20% of the high action level in EU directive for magnetic fields but 125% of the high action level for electric fields. These are to be used by trained industrial hygienists as a supplement to their occupational safety and health program.

NATO: The standardisation treaty for protection of military personnel of the North Atlantic Treaty Organization (NATO) refers to a standard of the Institute of Electrical and Electronics Engineers (IEEE). The level of the IEEE equivalent of exposure limit values for the induced electric field in the brain is similar to that of the sensory effects exposure limit values in the EU directive. The IEEE equivalent of exposure limit values for the rest of the body are less strict than the health effects exposure limit values in the EU directive (263% at 50 hertz for restricted working environments). The corresponding IEEE equivalent of action levels are less strict than the EU high action levels, due to different dosimetric considerations and safety factors. The IEEE limits for contact currents are also less strict than those in the EU directive.

Occupational exposure, radiofrequency fields

European Union

In all member states of the EU, protection or workers against the risks of EMF is regulated by national legislation based on directive 2013/35/EU. The directive sets minimum requirements, but allows member states to set stricter rules or limits and conditional exemptions, which are detailed below and in **Table 2**. For radiofrequency fields, the EU directive has action levels in terms of the electric field strength, magnetic flux density and power density outside the body, which are related to the health effects exposure limit values (equivalent to ICNIRP occupational basic restrictions for specific absorption rate and power density). One member state has action levels that differ from those in the EU directive:

Poland: For EMF with frequencies between 0 and 300 gigahertz, there are six sets of action levels delimiting a 'danger zone', 'threat zone' and 'intermediate zone', action levels for local exposure of extremities and ancillary action levels for peak levels for modulated fields. The exposure limit values for specific absorption rate are identical to the health effects exposure limit values in the EU directive.

Other countries

Australia: The national radiation protection regulations, which are only applicable to Commonwealth employees, set limits on occupational exposure to EMF from 'controlled apparatus', that is specified categories of devices that could cause EMF exceeding these limits (for example diathermy equipment). Its reference levels and basic restrictions are set by the national radiation protection standard and are identical to those in the 1998 ICNIRP guidelines. They therefore have the same basis as the limits in the EU directive, but apply to a narrower range of devices. In addition, a national radiocommunications standard limits the exposure of 'aware users' of mobile radiofrequency devices to basic restrictions identical to those in the EU directive.

China: The national standard with occupational exposure limits for physical agents in the workplace has limits for radiofrequency EMF with frequencies from 100 kilohertz to 300 gigahertz. For frequencies from 100 kilohertz to 300 megahertz, exposure limits are 8% to 41% of the action levels in the EU directive. For frequencies from 300 megahertz to 300 gigahertz, exposure limits do not vary with frequency but depend on the duration of exposure. At 900 megahertz the limit for whole body exposure varies from 222% of the EU action level for short exposure to 1% of the EU action level for 8-hour average exposure. Limits for partial body exposure are ten times higher than those for whole body exposure.

India: There are no legally binding limits on occupational exposure to radiofrequency EMF. Protection of workers would therefore fall under general health and safety legislation such as the Factories Act.

Japan: There are no legally binding limits on occupational exposure to radiofrequency EMF. The Japan Society for Occupational Health has recommended occupational exposure limits for EMF in terms of the strength of external electric and magnetic field and power density. These are identical to the thermal effects action levels in the EU directive.

Russia: The relevant 'Hygienic-epidemiological requirements' set a fixed limit per frequency band for maximum exposure to radiofrequency EMF with frequencies between 3 kilohertz and 300 gigahertz which is 44% of the action value for power density in the EU directive at 900 megahertz for whole body exposure and 222% for peak exposure of limbs. There are also lower time-dependent limits.

Switzerland: The federal law on accident insurance gives general rules to prevent illness caused by physical agents. The national accident insurer has specified that exposure limits identical to the action levels in the EU directive may not be exceeded.

United States: The equivalent of exposure limit values for whole body and for local exposure of the extremities (hands, wrists, ankles, feet, outer ears) in the federal legislation for transmitters are identical to those for whole body and for local exposure of limbs in the EU directive. The equivalent of exposure limit values for local exposure of all parts of the body except the extremities is 80% of that in the EU directive. The equivalent of action levels for electric and magnetic field strength are 18% higher than those in the EU directive (33% for power density), because a different model is used to calculate them. The equivalent of exposure limit values in the United States must be used for portable devices close to the body. The action levels are applied as *de facto* exposure limits for non-portable devices.

NATO: The standardisation treaty for protection of military personnel of the North Atlantic Treaty Organization (NATO) refers to a standard of the Institute of Electrical and Electronics Engineers (IEEE) with the same level of the equivalent of exposure limit values and action levels as those in the federal legislation of the United States, with the exception of the IEEE equivalent of exposure limit values for local exposure of the head which is identical to that in the EU directive. The limits for contact currents are higher than those in the EU directive.

Table 1 Reference levels or exposure limits for the general public for electromagnetic fields in inhabited areas in member states of the European Union and selected industrial nations outside the European Union (situation July 2017)

	50 Hz		900 MHz			1800 MHz			2100 MHz		
Country:	**electric field strength (V/m)**	**magnetic flux density (µT)**	**electric field strength (V/m)**	**magnetic flux density (µT)**	**equivalent plain wave power density (W/m^2)**	**electric field strength (V/m)**	**magnetic flux density (µT)**	**equivalent plain wave power density (W/m^2)**	**electric field strength (V/m)**	**magnetic flux density (µT)**	**equivalent plain wave power density (W/m^2)**
1999/519/EC	**5000**	**100**	**41**	**0.14**	**4.5**	**58**	**0.20**	**9**	**61**	**0.20**	**10**
Austria	[5000]	[100] [1]	[41]	[0.14]	[4.5]	[58]	[0.20]	[9]	[61]	[0.20]	[10]
Belgium	—	10 [2]	21 [3]	—	—	29 [3]	—	—	31 [3]	—	—
Bulgaria	— [4]	— [4]	—	—	0.1	—	—	0.1	—	—	0.1
Croatia	2000 [5]	40 [5]	17 [5]	0.055 [5]	0.72 [5]	23 [5]	0.078 [5]	1.4 [5]	25 [5]	0.084 [5]	1.7 [5]
Cyprus	[5000]	[100]	41	0.14	4.5	58	0.20	9	61	0.20	10
Czech Republic	2000	200	41	0.14	4.5	58	0.20	9	61	0.20	10
Denmark	—	— [6]	—	—	—	—	—	—	—	—	—
Estonia	5000	100	41	0.14	4.5	58	0.20	9	61	0.20	10
Finland	[5000]	[100] [7]	41	0.14	4.5	58	0.20	9	61	0.20	10
France	5000 [8]	100 [8]	41	0.14	4.5	58	0.20	9	61	0.20	10
Germany	5000 [9]	100 [9]	41	0.14	4.5	58	0.20	9	61	0.20	10
Greece	5000	100	32 [10]	0.11 [10]	2.7 [10]	45 [10]	0.15 [10]	5.4 [10]	47 [10]	0.16 [10]	6 [10]
Hungary	5000	100	41	0.14	4.5	58	0.20	9	61	0.20	10
Ireland	5000 [11]	100 [11]	41	0.14	4.5	58	0.20	9	61	0.20	10
Italy	—	3 [12]	6 [13]	0.02 [13]	0.1 [13]	6 [13]	0.02 [13]	0.1 [13]	6 [13]	0.02 [13]	0.1 [13]
Latvia	—	—	—	—	—	—	—	—	—	—	—
Lithuania	500 [14]	20 [14]	—	—	0.45	—	—	0.9	—	—	1
Luxemburg	5000 [15]	100 [15]	41 [16]	0.14	4.5	58 [16]	0.20	9	61 [16]	0.20	10
Malta	[5000]	[100]	41	0.14	4.5	58	0.20	9	61	0.20	10
Netherlands	[5000] [17]	[100] [17]	—	—	—	—	—	—	—	—	—
Poland	1000	75	7	—	0.1	7	—	0.1	7	—	0.1
Portugal	5000	100	41	0.14	4.5	58	0.20	9	61	0.20	10
Romania	5000	100	41	0.14	4.5	58	0.20	9	61	0.20	10
Slovakia	5000	100	41	0.14	4.5	58	0.20	9	61	0.20	10
Slovenia	500 [18]	10 [18]	13 [18]	0.04 [18]	0.45 [18]	18 [18]	0.06 [18]	0.9 [18]	19 [18]	0.06 [18]	1 [18]
Spain	[5000] [19]	[100] [19]	41	0.14	4.5	58	0.20	9	61	0.20	10
Sweden	[5000]	[100]	[41]	[0.14]	[4.5]	[58]	[0.20]	[9]	[61]	[0.20]	[10]
United Kingdom	[9000]	[360]	[41]	[0.14]	[4.5]	[58]	[0.20]	[9]	[61]	[0.20]	[10]
Australia	—	—	41	0.14	4.5	58	0.20	9	61	0.20	10
China	4000	100	12	0.04	0.4	12	0.04	0.4	12	0.04	0.4
India	—	—	13	0.041	0.45	18	0.058	0.9	20	0.063	1.1
Japan	3000 [20]	200 [20]	48	0.16	6	61	0.20	10	61	0.20	10
Russia	500	5 [21]	—	—	0.1	—	—	0.1	—	—	0.1
Switzerland	—	1 [22]	4 [23]	—	—	6 [23]	—	—	6 [23]	—	—
U.S.A.	— [24]	— [24]	—	—	6	—	—	10	—	—	10

Legend to Table 1: All limits are given as root mean square (rms) value. Where necessary magnetic flux density was calculated from magnetic field strength using a magnetic permeability of $4\pi \times 10^{-7}$ H/m. Normal typeface: reference level for the external field in the meaning of Recommendation 1999/519/EC, derived from basic restriction. Application is mandatory unless value is in square brackets. Italic typeface: mandatory exposure limit in terms of the external field outside the body. Radiofrequency limits are standardised to approximate mobile telecommunication frequency bands in Europe, but actual network frequencies may vary.

Notes:

1) *For new power lines requiring environmental impact assessment, authorities usually require compliance with Swiss limit of 1 µT*
2) *Flanders: indoor environment limit 10 µT, quality aim 0.2 µT, government recommendation for new situations near power line 0.4 µT; Brussels: 0.4 µT near new transformers and 100 µT near existing transformers; Wallonia: 5000 V/m and 100 µT near transformers*
3) *Limit in table is for publicly accessible places in Flanders, limit per antenna in places of stay 3.0 V/m at 900 MHz, 4.2 V/m at 1800 MHz, 4.5 V/m at 2100 MHz; Wallonia: limit per antenna 3 V/m; Brussels: limit per location 0.096 W/m² at 900 MHz, 0.19 W/m² at 1800 MHz, 0.22 W/m² at 2100 MHz*
4) *Minimal distances to power lines and to electrical distribution systems, differentiated by voltage; separate regulation for video display units*
5) *In homes, offices, schools, kindergartens, playgrounds, hospitals, care homes, tourist facilities; for other public spaces reference levels in 1999/519/EC apply*
6) *Danish Health Authority recommends that new homes and new institutions where children stay should not be built close to existing power lines and new power lines should not be built close to existing homes and institutions where children stay*
7) *Radiation safety authority recommends avoiding construction of permanent residences and premises meant for children in areas where magnetic flux density exceeds 0.4 µT*
8) *For new or modified installations; there is also government advice to local authorities not to create new establishments with children in zones with magnetic flux density above 1 µT*
9) *For new or modified installations exhaust all possibilities to minimise EMF; new power lines ≥ 220 kV may not span buildings for long-term stay of people*
10) *For antenna stations closer than 300 m to sensitive locations (schools, kindergartens, hospitals, care homes); elsewhere 35 V/m, 0.11 µT, 3.1 W/m² at 900 MHz; 49 V/m, 0.16 µT, 6.3 W/m² at 1800 MHz; 51 V/m, 0.17 µT, 7 W/m² at 2100 MHz*
11) *For new energy infrastructure, State Companies and energy developers must comply with ICNIRP limits and associated EU Recommendations as an intrinsic part of the planning process*
12) *For new situations with power lines near homes, schools, playgrounds, places with stay > 4 hours; 10 µT for existing situations near homes, schools, playgrounds, places with stay > 4 hours; 100 µT and 5000 kV/m for all other exposures from power lines*
13) *EMF from fixed systems for telecommunication and radio or TV broadcasting near homes and their outdoor annexes, in schools and playgrounds, in places with stay greater than 4 hours; elsewhere 20 V/m, 0.06 µT, 1 W/m²*
14) *Inside residential and public buildings; limits for living environment outside residential and public buildings 1000 V/m, 40 µT*
15) *Security conditions for electricity lines, there are also voluntary minimal distances to power lines for new developments*
16) *Limit per antenna at places where people can stay 3.0 V/m, applies to antennas with power of 100 W and higher*
17) *Ministerial recommendation: create no new situations of long-term stay of children in magnetic flux density greater than 0.4 µT around overhead power lines, otherwise reference level in 1999/519/EC applies*
18) *Applies to homes, hospitals, health resorts, public buildings, tourism buildings, schools, nurseries, playgrounds, parks, recreational areas; otherwise limit for external electric and magnetic field strength equal to reference level in 1999/519/EC; for power frequency limits apply to new or reconstructed sources only*
19) *No binding national limits for 50 Hz fields, but in practice electricity companies and the authorities apply the limits in 1999/519/EC*
20) *Limit listed is for 50 Hz fields, power frequency is 50 Hz in East Japan and 60 Hz in West Japan*
21) *Limit for living quarters, children's, preschool, general and medical institutions; non-residential premises 10 µT, inhabited areas outdoors 20 µT, uninhabited areas 100 µT*
22) *Limit at places of sensitive use (buildings in which persons regularly stay for longer periods, playgrounds) for all high voltage installations except existing powerlines; otherwise reference level in 1999/519/EC applies at all places accessible for the public*
23) *Limit at places of sensitive use (buildings in which persons regularly stay for longer periods, playgrounds) for individual antenna installations; otherwise reference level in 1999/519/EC applies at all places accessible for the public*
24) *Power frequency is 60 Hz; no federal regulation, limits in some states, prudent avoidance policy in others (measures to reduce exposure at reasonable cost)*

Table 2 Occupational reference levels or exposure limits for electromagnetic fields in member states of the European Union and selected industrial nations outside the European Union (situation July 2017)

	50 Hz		900 MHz					
Country:	**electric field strength (high AL) (V/m)**	**magnetic flux density (high AL) (µT)**	**electric field strength (V/m)**	**magnetic flux density (µT)**	**equivalent plain wave power density (W/m^2)**	**conditional exemption from ELV for MRI**	**alternative protection system for armed forces**	**temporary exemption from ELV for specific sectors or activities**
2013/35/EU	**20000**	**6000**	**90**	**0.30**	**—**	**yes**	**yes**	**yes**
Austria	20000 [1)]	6000 [1)]	90 [1)]	0.30 [1)]	—	yes	no	yes [2)]
Belgium	20000	6000	90	0.30	—	yes	no	yes
Bulgaria	20000	6000	90	0.30	—	yes	yes (NATO)	no
Croatia	20000	6000	90	0.30	—	yes	yes	yes
Cyprus	20000	6000	90	0.30	—	yes	yes	yes
Czech Republic	10000	1000	90	0.30	22.5	no	no	no
Denmark	20000	6000	90	0.30	—	yes	no	no
Estonia	20000	6000	90	0.30	—	yes	yes (NATO)	no
Finland	20000	6000	90	0.30	—	yes	yes	yes
France	20000 [3)]	6000 [3)]	90 [3)]	0.30 [3)]	—	yes [4)]	no	no
Germany	20000	6000	90	0.30	—	yes [4)]	no	yes [4)]
Greece	20000	6000	90	0.30	—	yes	Yes (NATO)	Yes [5)]
Hungary	20000	6000	90	0.30	—	no [6)]	yes (NATO)	yes [6)]
Ireland	20000	6000	90	0.30	—	yes	no	no
Italy	20000	6000	90	0.30	—	no [7)]	yes	yes [7)]
Latvia	20000	6000	90	0.30	—	yes	yes	no
Lithuania	20000	6000	90	0.30	—	yes	yes [8)]	no
Luxemburg	20000	6000	90	0.30	—	yes [9)]	yes (NATO) [9)]	yes [9)]
Malta	20000	6000	90	0.30	—	yes	yes	yes
Netherlands	20000	6000	90	0.30	—	yes	yes	no
Poland	10000 [10)]	2000 [10)]	60 [10)]	0.20 [10)]	—	no	yes	no
Portugal	20000 [11)]	6000 [11)]	90 [11)]	0.30 [11)]	—	yes	yes	no
Romania	20000	6000	90	0.30	—	yes	yes	yes
Slovakia	20000	6000	90	0.30	—	yes	yes	yes
Slovenia	20000	6000	90	0.30	—	yes	yes	yes [12)]
Spain	20000	6000	90	0.30	—	yes	yes (NATO)	yes
Sweden	20000	6000	90	0.30	—	yes	yes	no
United Kingdom	20000	6000	90	0.30	—	yes	yes	yes [13)]
Australia	*10000*	*1000*	*92*	*0.31*	*22.5*			
China	*5000*	—	—	—	50 [14)]			
India	—	—	—	—	—			
Japan	— [15)]	— [15)]	— [15)]	— [15)]	— [15)]			
Russia	—	*2000* [16)]	—	—	*10* [16)]			
Switzerland	*10000* [17)]	*500* [17)]	*90* [17)]	*0.30* [17)]	*22.5* [17)]			
U.S.A.	— [18)]	— [18)]	—	—	30			

Legend to Table 2: All limits are given as root mean square (rms) value. Where necessary magnetic flux density was calculated from magnetic field strength using a magnetic permeability of $4\pi \times 10^{7}$ H/m. Normal typeface: action level (AL)/reference level for the external field in the meaning of Directive 2013/35/EU or ICNIRP guidelines, derived from exposure limit value (ELV)/basic restriction. Application is mandatory unless value is in square brackets. Italic typeface: mandatory exposure limit in terms of the external field outside the body.

Notes:

1) *Limits in EU recommendation 1999/519/EC apply to pregnant workers; AL may not be exceeded for workers younger than 18 years; sensory effects ELV may only be exceeded for resistance welding and electricity supply sector*
2) *Sensory and health effects ELV may be temporarily exceeded for workers in delimited areas in establishments for generation, transport and distribution of electrical energy*
3) *Limits in EU recommendation 1999/519/EC apply to pregnant workers; sensory effects ELV may not be exceeded for workers younger than 18 years*
4) *Exemption with additional obligations to those in Directive 2013/35/EU*
5) *For any temporary exemption from ELV for a specific sector or activity, the National Occupational Health & Safety Council shall give its expert opinion beforehand*
6) *Regional radiation safety officer may allow exposure of workers to exceed health effects ELV in specific circumstances where state-of-the-art technical and organisational protection measures have been implemented; annexes to national legislation contain list of equipment requiring risk assessment approval, including MRI*
7) *Ministers of Labour and Social Policy and of Health may grant a conditional and temporary derogation at the request of the employer, with additional requirements for MRI*
8) *Scope extended: military personnel or national security, public security and customs officials as determined by Lithuanian intelligence regulations*
9) *Employer is obliged to check the appropriateness of the measures taken with an approved expert acting within the competences and authority of the labour inspectorate*
10) *Values listed are for basic 'threat' AL, there are also higher 'danger' AL, lower 'intermediate' AL for indirect effects and ancillary AL for modulated fields*
11) *Employer shall ensure that the exposure of workers to electromagnetic fields is reduced to the lowest possible level, but in any case it should not exceed ELV*
12) *Sensory and health effects ELV may be temporarily exceeded for workers in police, other units and services for protection, rescue and relief in specific circumstances*
13) *Temporary conditional exemption from ELV for electrolysis, dielectric heating, induction heating, manual resistance welding, MRI equipment other than that for patients*
14) *Limit for short exposures, for longer exposures limits decrease down to 0.5 W/m^2 (continuous wave) or 0.25 W/m^2 (pulsed) for 8 hours with whole body exposure*
15) *No legal limits for workers, Japan Society for Occupational Health has recommended occupational exposure limits in terms of the strength of external electric and magnetic field and power density identical to the low action levels and thermal effects action levels in the EU directive*
16) *Limit for exposures shorter than 1 hour, for longer exposures limits decrease down to 100 µT for 8 hours; for radiofrequency fields there are also limits on exposure x time*
17) *For pregnant workers, exposure limits identical to the reference levels in EU recommendation 1999/519/EC apply*
18) *No legal limits for workers, American Conference of Governmental Industrial Hygienists has recommended 'threshold limit values' of 25000 V/m and 1000 µT at 60 Hz as guidelines to assist in the control of potential workplace health hazards*

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Colophon

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MILJØMINISTERIET

Miljøstyrelsen

Metoder til fastsættelse af kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand med henblik på at beskytte sundheden

Vejledning fra Miljøstyrelsen **Nr. 5** 2006

Indhold

Forord

Principperne for vurdering og fastsættelse af kvalitetskriterier for kemikalier med henblik på at beskytte sundheden er beskrevet tidligere[1]. Men i forbindelse med Miljøministerens redegørelse om jordforureningsloven i 2003, besluttede regeringen at nedsætte en arbejdsgruppe, som fik til opgave at "*vurdere, hvorvidt de sundhedsmæssige kvalitetskriterier ligger på det rigtige niveau i relation til international praksis, samt give forslag til ændringer*.

Arbejdsgruppen udpegede en række politiske valg, der indgår i principperne for fastsættelse af kvalitetskriterier. På den baggrund blev der i december 2005 indgået en aftale mellem Miljøministeren og et bredt udsnit af Folketingets partier, som bl.a. fastlægger følgende generelle principper for fastsættelsen af forebyggende kriterier :

- Hovedparten af befolkningen skal fortsat beskyttes (dvs. hensyntagen til særligt udsatte skal også inddrages).
- Der skal tages specifikt hensyn til børn.
- Det fastholdes, at der højst anvendes en samlet usikkerhedsfaktor på 10.000, når resultater fra dyreforsøg overføres til mennesker.
- Der accepteres fortsat en livstidsrisiko på 10^{-6}.

Denne vejledning indarbejder disse principper og gennemgår de metoder, som skal anvendes ved fastsættelse kvalitetskriterier for jord, luft og drikkevand med henblik på at beskytte sundheden[2].

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[1] Miljøstyrelsens Vejledning nr. 1 "Sundhedsmæssig vurdering af kemiske stoffer i drikkevand ", 1992, og i bilag til Miljøstyrelsens Vejledning nr. 6, 1990 "Begrænsning af luftforurening fra virksomheder" samt i Miljøprojekt nr. 12, 1995 "Toksikologiske kvalitetskriterier for jord og drikkevand".

[2] Principper for fastsættelse af kvalitetskriterier med henblik på at beskytte miljøet er tilsvarende beskrevet i Miljøstyrelsens vejled-

Vejledningen henvender sig til centrale og lokale myndigheder, herunder embedslægeinstitutionerne, konsulenter, rådgivere og virksomheder.

Miljøstyrelsen fastsætter løbende kvalitetskriterier for konkrete kemiske stoffer i jord, luft og drikkevand med henblik på at beskytte sundheden, jf. Miljøbeskyttelsesloven § 14, stk. 1,. Kvalitetskriterierne bliver fastsat på baggrund af videnskabelige rapporter og efter drøftelse i en styregruppe med deltagelse af bl.a. Fødevarestyrelsen, Arbejdstilsynet og Sundhedsstyrelsen.

Denne vejledning og detaljeringsgraden, ikke mindst i den tilhørende faglige rapport[3], skal give grundlag for større forståelse for, hvordan kvalitetskriterier skal udarbejdes.

Kvalitetskriterierne anvendes dels til at vurdere alvoren af en allerede given forurening, og dels når myndighederne stiller krav i forbindelse med udledning af konkrete stoffer til omgivelserne.

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ning nr. 4 (2004): Principper for fastsættelse af vandkvalitetskriterier for stoffer i overfladevand.

[3] Miljøprojekt Nr. 974, Miljøstyrelsen (2005). Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand. Rapporten er udarbejdet af Institut for Fødevaresikkerhed og Ernæring, Fødevaredirektoratet (nu Danmarks Fødevareforskning) i samarbejde med Miljøstyrelsen.

1 Indledning

1.1 Hvad er kvalitetskriterier

1.1.1 Formål med fastsættelse af kvalitetskriterier for kemikalier

Denne vejledning beskriver den administrative praksis ved fastsættelse af kvalitetskriterier for kemikalier med henblik på at beskytte sundheden.

Kvalitetskriterier danner basis for en række vurderinger af administrativ karakter, som vedrører både jord, luft og drikkevand.

Målsætningen ved fastsættelsen af kvalitetskriterier for kemikalier er, at de skal medvirke til *at forebygge forurening og skader på sundheden*. Kvalitetskriterierne ses som et element i lovens overordnede politiske målsætning om at oppebære et højt beskyttelsesniveau for befolkningens sundhed og sikre en bæredygtig udvikling, herunder fremme af renere teknologi.

Kvalitetskriterierne angiver et højt beskyttelsesniveau, hvor ingen effekt kan forventes, selv ved udsættelse gennem et helt liv, eller hvis der er tale om et stof uden tærskelværdi, en teoretisk forøget risiko for kræft hos én ud af en million mennesker, som er udsat for stoffer gennem et helt liv på 70 år.

Kvalitetskriterierne bruges af myndighederne i forbindelse med vurdering af alvoren af et givet forureningsniveau. Det kan være relevant, hvis der er tale om en eksisterende forurening (fortidens synder), eller hvis der skal fastsættes kravværdier for udledning af konkrete stoffer i miljøet (forebyggende). For begge situationer kan andre forhold end de sundhedsmæssige spille ind, såsom baggrundsniveauer og tekniske/økonomiske overvejelser. Ved indsatsen i forhold til eksisterende forureninger kan der også være behov for at

vurdere, hvad der er sundhedsmæssigt forsvarligt, når der tages hensyn til overordnede samfundsmæssige prioriteringer. Med udgangspunkt i de sundhedsmæssigt fastsatte kvalitetskriterier udarbejdes således en række administrativt fastsatte kriterier.

Eksempler på administrative kriterier som er baseret på kvalitetskriterier er:

1. Luftkvalitetskriteriet anvendes som udgangspunkt til at fastsætte bidrags værdier (B-værdier), der anvendes i forbindelse med regulering af virksomheders udslip af kemiske stoffer til udeluften.
2. Luftkvalitetskriteriet anvendes til at vurdere afdampning af kemiske stoffer fra jordforureninger. Det anvendes også ved kortlægning og offentlig oprydning af forurenede grunde.
3. Jordkvalitetskriterier anvendes som udgangspunkt ved fastsættelse af administrative kriterier, der anvendes ved de lokale myndigheders kortlægning af forurenede grunde og i forbindelse med vurderinger af arealanvendelse, samt ved offentlig oprydning af forurenede grunde[4].
4. Jordkvalitetskriteriet anvendes endvidere som udgangspunktet for fastsættelse af afskæringskriteriet for visse immobile stoffer. Afskæringskriteriet anvendes ved lettere forurenede områder som skillelinie mellem det niveau, hvor det er nødvendigt at fjerne jorden og det niveau, hvor det er tilstrækkeligt at iværksætte særlige forholdsregler i forbindelse med følsom anvendelse af områderne, som fx private haver eller børneinstitutioner.
5. Drikkevandskvalitetskriteriet anvendes af de centrale og decentrale myndigheder i forbindelse med håndtering af konkrete sager med kemisk forurening af drikkevand, som et supplement til de kravværdier, der er fastsat i bekendtgørelsen om vandkvalitet.
6. Drikkevandskvalitetskriterierne anvendes som udgangspunkt ved fastsættelse af grundvandskvalitetskriterier i tilknytning til nedsivning fra jordforureninger,

[4] Kvalitetskriterierne omfatter normalt ikke en vurdering af om arealer kan anvendes til dyrkning af nytteplanter eller til afgræsning for husdyr.

idet grundvandet skal være af en kvalitet, så de fastsatte drikkevandskrav/ -kvalitetskriterier kan overholdes.

Der eksisterer en række vejledninger og lister, som nærmere beskriver baggrunden for, hvordan et kvalitetskriterium kan anvendes.[5]

1.1.2 Generelle principper for fastsættelse af kvalitetskriterier for kemikalier

Kvalitetskriterierne fastsættes på et niveau, hvor udsættelse gennem et helt liv ikke fører til skadevirkninger i befolkningen. De fastsættes på baggrund af den eksisterende viden og under hensyntagen til de mangler, der ligger i datagrundlaget.

For at minimere risici for skadelig påvirkning af befolkningen indgår beskyttelse af særligt følsomme grupper fx børn, gravide, syge, ældre og svækkede ved fastsættelse af kvalitetskriterier.

Viden om et kemisk stofs sundhedsskadelige egenskaber og om bestemte befolkningsgruppers særlige følsomhed er sjældent så eksakt, at der kan fastsættes et kvalitetskriterium, der præcist definerer skillelinien (hvis en sådan overhovedet findes) mellem et ufarligt og farligt niveau. Kvalitetskriterierne kan således ikke opfattes som en streg i sandet, hvor enhver overskridelse er farlig. Ved fastsættelse af kvalitetskriterier for kemikalier skal anvendes en forsigtighedstilgang, da målet er at sikre et højt beskyttelsesniveau for alle ved udsættelse over et helt liv.

Et kvalitetskriterium skal således opfattes som en sikkerhedsgrænse og ikke en faregrænse. En overskridelse er "det gule lys", som advarer om, at her er noget, som måske kan blive et reelt problem.

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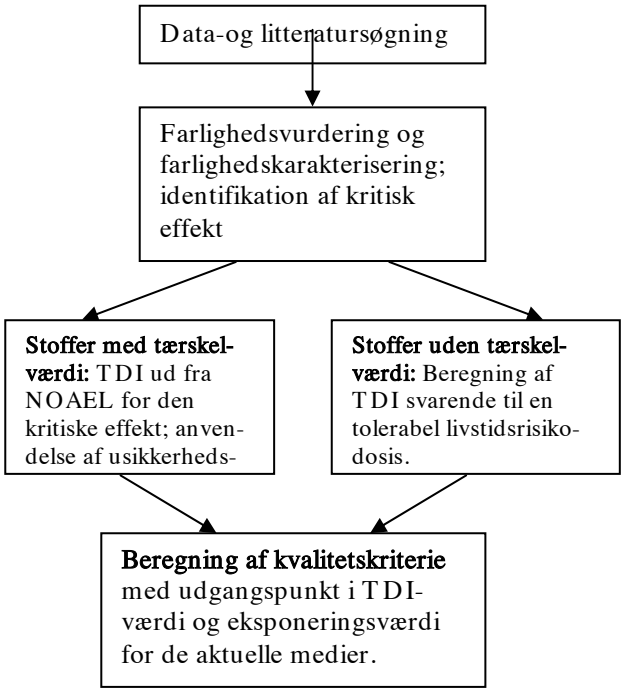
[5] Luftvejledningen, Miljøstyrelsens Vejledning nr. 2, 2001; Oprydning på forurenede lokaliteter

1.2 Vejledningens opbygning og indhold

Vejledningen udstikker retningslinier for den faglige risikovurdering og for de metoder/ principper der anvendes. Dernæst beskrives proceduren for beregning af kvalitetskriterierne (se figur 1).



Figur 1.
Fremgangsmåden ved beregning af kvalitetskriterium (TDI=Tolerabel Dagligt Indtag)

Det videnskabelige grundlag for fastsættelse af sundhedsmæssigt baserede kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand består af en farlighedsvurdering, en dosis-respons (effekt) vurdering (farlighedskarakterisering), samt en eksponeringsvurdering. Farlighedsvurderingen og farlighedskarakteriseringen tager udgangspunkt i undersøgelser af det pågældende stofs toksikologiske effekter i mennesker og i dyr.

I kapitel 2 omtales *Datagrundlaget* som anvendes som udgangspunkt for arbejdet. Data hentes primært fra internationale og nationale dokumenter, via litteratursøgning i internationale databaser, samt fra originalartikler.

Kapitel 3 behandler de faglige metoder, der anvendes i forbindelse med farlighedsvurderingen og farlighedskarakteriseringen. Dosis-effekt og dosis-responssammenhænge og udpegning af NOAEL (No Observed Adverse Effect Level) og LOAEL (Lowest Observed Adverse Effect Level) beskrives.

I kapitel 4 beskrives hvordan farlighedskarakteriseringen udmunder i udpegning af en kritisk effekt, som danner udgangspunkt for fastsættelse af en *tolerabel daglig indtagelse, TDI*. Her omtales, hvordan anvendelsen af usikkerhedsfaktorer indgår i beregningerne.

I kapitel 5 omtales, hvordan TDI beregnes for kræftfremkaldende stoffer uden tærskelværdi. Risikoniveauet for TDI-værdien defineres, og der gives retningslinier med hensyn til valg af metode til, hvordan beregningen af dette risikoniveau foretages.

Kapitel 6 beskriver, hvordan den videre beregning af kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand foretages ud fra de fastsatte TDI-værdier. I bilag 2 til vejledningen omtales rationalet for at anvende konkrete standardbetragtninger m.h.t. personers udsættelse for forureninger gennem jord, luft og drikkevand. Endvidere omtales hvilke andre faktorer (fx hensyntagen til lugt eller smag) end de rent sundhedsmæssige, der i visse tilfælde kan have indflydelse på kvalitetskriteriet.

1.3 Ændringer i forhold til tidligere praksis

Ved fastsættelsen af kvalitetskriterier anvendes der internationalt anerkendte principper. I denne forbindelse skal fremhæves de principper og metoder, der er beskrevet i to publikationer af WHO/ IPCS[6,7] om udarbejdelsen af vejledende grænseværdier og risikovurdering af kemisk udsættelse. WHO´s publikationer[8,9] omhandlende risikovurdering og fastsættelse af vejledende grænseværdier for en række kemiske stoffer i drikkevand og luft har også indgået i arbejdet. På tilsvarende vis er der en tæt sammenhæng med de principper og metoder, der anvendes i forbindelse med EU´s risikovurderingsprogram for kemiske stoffer[10].

På visse områder indebærer denne vejledning, at den hidtidige praksis for fastsættelse af kvalitetskriterier justeres. Dette gælder fx for beregning af kvalitetskriterier for kemikalier i luft og drikkevand, idet der nu som udgangspunkt anvendes standardværdier for børns udsættelse (se kapitel 6 og bilag 2).

I forbindelse med ekstrapolering af tolerabelt risikoniveau for genotoksiske kræftfremkaldende stoffer (dvs. kræftfremkaldende stoffer hvor der ikke anses at være en tærskelværdi for effekt), anvendes lineær ekstrapolation ud fra den så-

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[6] WHO/IPCS (1994). Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. Environmental Health Criteria no. 170. International Programme on Chemical Safety.

[7] WHO/IPCS (1999). Principals for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria no. 210. International Programme on Chemical Safety.

[8] WHO (1996). Guidelines for drinking-water quality 2nd edition, vol 2. Health Criteria and other supporting information. International Programme on Chemical Safety.

[9] WHO (2000). Air Quality Guidelines for Europe, 2nd edition. WHO Regional Publications, European Series, no. 91.

[10] EEC (2003). Technical Guidance Document in support of Commission Directive 93/67/EEC on risk assessment for new notified substances and Commission Regulation (EC) No. 1488/94 on risk assessment for existing substances and Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market.

kaldte T25- metode udviklet i forbindelse med EU´s risikovurderingsprogram (kapitel 5).

Hvis datagrundlaget er til stede, anbefales det at anvende benchmark-metoden ved fastsættelse af TDI (kapitel 3).

Det skal bemærkes, at denne vejledning anvender begrebet usikkerhedsfaktorer, hvor man tidligere anvendte begrebet sikkerhedsfaktorer. Dels beskriver ordet *usikkerhed* i højere grad faktorernes anvendelse, idet de netop tager højde for usikkerheder. Dels imødegås den misforståelse, at en stor sikkerhedsfaktor for et stof medfører større sikkerhed for dette stof i forhold til en lille anvendt sikkerhedsfaktor for et andet stof. Faktorerne anvendes derimod for at opnå et ensartet beskyttelsesniveau, og størrelsen af faktorerne afspejler usikkerheder som følge af usikker viden og manglende datagrundlag.

De angivne metoder i vejledningen anvendes fremover ved fastsættelse af kvalitetskriterier.

De ændringer der er sket i forhold til tidligere praksis betyder ikke, at de eksisterende kvalitetskriterier skal "laves om", idet anvendelse af de nye metoder ikke vurderes at påvirke beskyttesesniveauet væsentligt. Når et kvalitetskriterium tages op til revurdering bør dette ske både m.h.t. til revurdering af de sundhedsmæssige data og af de eksponeringsmæssige forhold. En revurdering foretages derfor fuldt ud med alle de vurderingsmæssige og beregningsmæssige faser, der er beskrevet i vejledningen.

Referencer

Bekendtgørelse af Lov om miljøbeskyttelse (Miljøbeskyttelsesloven). Lovbekendtgørelse nr. 753 af 25. august 2001.

Miljøstyrelsen (1991). Orientering om ny miljøbeskyttelseslov. Orientering nr. 6, 1991.

2 Datagrundlag for vurdering af farlighed

Fastsættelse af kvalitetskriterier for et kemisk stof sker på baggrund af eksisterende viden om stoffets sundhedsskadelige egenskaber.

Kvalitetskriterierne fastsættes med henblik på beskyttelse af menneskers sundhed, og det ideelle datagrundlag for fastsættelse af et kvalitetskriterium er derfor viden, hvor mennesker har været udsat for det konkrete stof. For langt de fleste kemiske stoffer er der kun begrænset viden om veldefineret udsættelse og effekter hos mennesker, og kvalitetetskriterierne vil som oftest være baseret på viden opnået fra dyreforsøg med mere veldefineret udsættelse eller *in vitro* data[11].

For en mere detaljeret beskrivelse af nedenstående afsnit henvises til kapitel 2, Miljøprojekt Nr. 974 (2005).

2.1 Data fra mennesker

Fordelen ved at anvende data, hvor mennesker har været udsat for et kemisk stof, er, at man undgår at skulle overføre data fra dyreforsøg, og estimere hvad en tilsvarende udsættelse betyder hos mennesker. Erfaringer med menneskers udsættelse kan stamme fra en række forskellige typer undersøgelser og afrapporteringer, der groft kan deles op på følgende måde:

- *case reports og kliniske undersøgelser*
- *befolkningsundersøgelser (fx arbejdsmiljø eller udvalgte dele af befolkningen)*
- *undersøgelser af frivillige forsøgspersoner*

[11] Data fra reagensglasforsøg.

Ved *case reports og kliniske undersøgelser* opnås data fra forgiftningstilfælde eller erfaringer fra undersøgelser i klinikken, hvor personer fx i behandlingsøjemed udsættes for stoffer for at vurdere eventuelle allergiske reaktioner. Fordelen ved disse typer data er, at man her umiddelbart kan se en sammenhæng mellem udsættelse og (akutte) effekter.

I *befolkningsundersøgelser* vurderes en større gruppe af mennesker mere systematisk med hensyn til sammenhængen mellem udsættelse og sundhedsskader. Disse undersøgelser kan fx omfatte sundhedsovervågning af særlige grupper i arbejdsmiljøet, hvor personerne er karakteriseret ved en særlig udsættelse, eller man kan undersøge, om der hos personer, der har udviklet nogle konkrete sygdomme, er nogle fælles karakteristika med hensyn til kemiske påvirkninger. Fordelen ved at anvende data fra denne type undersøgelser er, at man er meget tæt på den målgruppe, man ønsker at beskytte med kvalitetskriterierne. Samtidig ses effekterne i sammenhæng med den dagligdag, som mennesker nu engang fungerer i, hvor man udsættes for en kompleks blanding af livsstilsfaktorer og miljøfaktorer. Ulempen er, at det ofte er vanskeligt at vurdere omfanget af udsættelsen af en given komponent, og at det pga. mange andre samvirkende faktorer kan være svært at påvise sammenhænge, som kan være sløret af al "støjen" fra andre faktorer. Ligesom undersøgelserne kun sjældent kan dokumentere en årsagsvirkningssammenhæng, vil de også kun uhyre sjældent kunne anvendes til at dokumentere manglende sammenhæng, dvs. frikende stoffer.

I *undersøgelser med frivillige forsøgspersoner* udsætter man i reglen forsøgspersonerne i en kortere varighed for et konkret stof for at vurdere effekterne. Disse undersøgelser er meget sammenlignelige med dyreforsøg, hvor man tilsvarende har en meget veldefineret udsættelse. De naturlige og etiske begrænsninger ved disse undersøgelser betyder, at der hos mennesker kun kan undersøges for lettere grader af akutte effekter og i sammenhæng med kortere tids forsøgsudsættelse. Endvidere er forsøgspersonerne sjældent særligt følsomme, som visse undergrupper i befolkningen kan være.

Man skal være meget opmærksom på det etiske aspekt m.h.t. anvendelse af frivillige forsøgspersoner. I forbindelse

med vurdering af konkrete humane undersøgelser i risikovurderingssammenhæng, bør anvendelsen af disse data vurderes nøje såvel ud fra etiske som kvalitetsmæssige hensyn. Især ældre undersøgelser kan være udført under stærkt kritisable forhold og med store undersøgelsesmæssige mangler.

Kvalitetskriterier for kemikalier fastsættes ud fra eksisterende data, og det frarådes generelt at igangsætte humanforsøg for at opnå øget viden om konkrete stoffers skadelige effekter.

2.2 Dyreeksperimentelle undersøgelser

For de fleste kemiske stoffer foreligger der ikke data fra menneskers udsættelse, hvorfor kvalitetskriterier hyppigst baseres på data fra dyreeksperimentelle undersøgelser. Resultaterne fra dyreforsøgene anvendes således som model og anvendes til at forudsige hvilke effekter, der kan forventes hos mennesker. Dyreeksperimentelle data kan også benyttes som supplement til humane data, der ikke er entydige, eller til at udpege de aktive stoffer, når mennesker har været udsat og reageret over for blandinger af stoffer.

Fordelene ved dyreforsøg er, at der er tale om standardiserede forsøgsbetingelser, og at der er mulighed for at afsløre væsentlig flere effekter hos forsøgsdyr end hos mennesker, da organer og væv kan undersøges efter forsøgets afslutning. Der er også mulighed for at undersøge virkningsmekanismer og detaljerede dosis-effekt og dosis-responssammenhænge for enkeltstoffer.

Ideelt set ønskes der ved fastsættelsen af kvalitetskriterier for et kemikalie et fuldt datasæt bestående af dyreeksperimentelle undersøgelser til vurdering af en række toksikologiske egenskaber: Toksikokinetik (optagelse og udskillelse), akut toksicitet, irritation, sensibilisering (allergi), toksicitet ved gentagen administration af stoffet, mutagenicitet og genotoksicitet (påvirkning af arvematerialet), kræftfremkaldende effekter, samt effekter på reproduktion og fosterudvikling.

Dyreforsøg har imidlertid også begrænsninger, da nogle effekter kan være vanskelige at afsløre fx lettere grader af slimhindeirritation i øjne og luftveje, lettere grader af påvirkning af centralnervesystemet og visse typer nerveskader.

Endelig kan nogle dyrearter udvikle artspecifikke effekter over for visse stoffer, hvor relevansen i forhold til udsættelse af mennesker er meget omdiskuteret (se afsnit 3).

Det er endvidere vigtigt at vurdere kvaliteten af de dyreeksperimentelle undersøgelser, der anvendes som udgangspunkt for fastsættelse af kvalitetskriterier. Undersøgelser af høj kvalitet, som er udført efter eller på niveau med OECD´s eller EU´s retningslinier for forsøgsdyrstestning, bør foretrækkes. Ved risikovurderingen i forbindelse med fastsættelse af kvalitetskriterier er der dog ikke nogle formelle kvalitetskrav til undersøgelserne, idet en lang række undersøgelser i den videnskabelige litteratur ofte vil være udført i forskningsøjemed uden at undersøgelserne er udført efter en officiel forsøgsguideline eller i overensstemmelse med GLP-reglerne.[12] Sådanne undersøgelser, der ofte er kvalitetssikret i peer-reviewede tidsskrifter, kan indeholde væsentlig information. Forsøgets kvalitet og validitet må i de aktuelle tilfælde vurderes, og der tages stilling til om forsøget kan være af betydning ved fastsættelse af et kvalitetskriterium.

På baggrund af de etiske aspekter ved dyreforsøg arbejdes der internationalt med at udvikle alternative *in vitro* metoder.

2.3 Andre typer data

Ud over forsøg på levende dyr foreligger der ofte undersøgelser der er udført på udtagne organer, væv eller isolerede celler. Disse *in vitro* metoder finder især anvendelse til vurdering af stoffers toksiske effekt på organ-/celleniveau særligt m.h.t. mutagene og genotoksiske effekter.

[12] GLP står for Good Laboratory Practice, og er et regelsæt udviklet af OECD for at sikre kvaliteteten af undersøgelserne.

Normalt kan *in vitro* forsøg ikke anvendes til at fastlægge tærskel for effekt (NOAEL e.l.), men de vil indgå i bedømmelsen af stoffets farlighed. In vitro-data kan således styrke mistanken om skadeeffekter og derved påvirke valget af usikkerhedsfaktorer (se afsnit 4).

Ved mangel på data for et konkret stof kan data fra nært beslægtede stoffer indgå i vurderingen ud fra betragtninger om kemisk strukturlighed og sammenfaldende effekter. Sådanne data kan have indflydelse på den rent kvalitative, men også i visse tilfælde i den kvantitative vurdering, hvis det konkret vurderes, at der er grundlag for meget snævert koblede analogislutninger.

Endvidere kan der udføres en mere systematisk analyse vedrørende kvantitative struktur-aktivitets relationer (engelsk: Quantitative Structure Activity Relationships – QSARs). Anvendelse af denne form for computerbaserede modeller har i en række tilfælde vist sig som et alternativ til dyreforsøg med henblik på forudsigelse af toksikologiske egenskaber (MST 2001[13]). Vurderingerne kan sjældent anvendes som udgangspunkt for beregning af kvalitetskriteriet, men vil i visse tilfælde kunne anvendes til styrkelse af mistanken om konkrete effekter. QSAR vil således kunne påvirke valget af usikkerhedsfaktorer ved beregning af kvalitetskriteriet.

2.4 Indhentning af data

Som udgangspunkt for udarbejdelse af kvalitetskriterier anvendes i udstrakt grad internationalt anerkendte stofmonografier og dokumenter, hvor det aktuelle stof er vurderet og beskrevet m.h.t. dets sundhedsskadelige effekter. En række af disse værker (kilder) er nævnt i afsnit 2.5.1 i Miljøprojekt Nr. 974 (2005) . Sådanne dokumenter, der ofte er baseret på en grundig faglig vurdering ved en særlig nedsat ekspertgruppe, vil typisk kunne anvendes til at udpege de relevante undersøgelser og data, der skal anvendes til den videre beregning af kvalitetetskriteriet. Det anbefales gene-

[13] Miljøstyrelsen (2001). Report on the advisory list for selfclassification of dangerous substances. Environmental Project No. 636 2001. http://www.mst.dk/udgiv/publications/2001/87-7944-694-9/html/

relt, at data, der skal danne udgangspunkt for beregning af kvalitetskriteriet, altid hjemskaffes som originallitteratur til vurdering af den konkrete undersøgelses kvalitet og relevans.

Endvidere søges i en række relevante databaser (se afsnit 2.5.2 i den faglige rapport). Sådanne søgninger er især relevante, hvis en international stofmonografi for det konkrete stof ikke foreligger, eller hvis vurderingen ligger nogle år tilbage.
For visse stoffer kan man komme ud for at datasøgning giver så ringe resultat, at der ikke er tilstrækkeligt datagrundlag til at foretage en vurdering af stoffet. For eksempel hvis der kun er akutte studier til rådighed, eller ingen studier fastlægger NO(A)EL/LO(A)EL.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier i jord, luft og drikkevand.

3 Farlighedsvurdering og farlighedskarakterisering

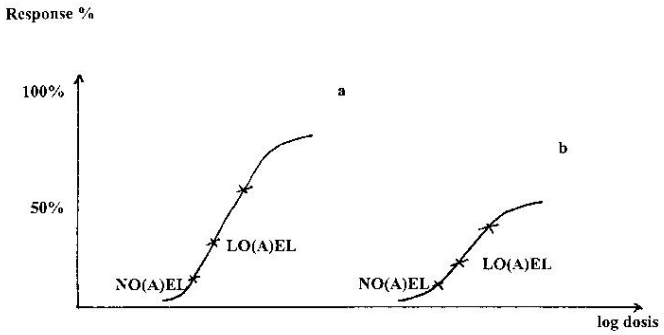
Farlighedsvurderingen og farlighedskarakteriseringen har til formål at beskrive stoffets farlige egenskaber. Der opstilles i videst muligt omfang dosis-effekt og dosis-respons sammenhænge, der danner baggrund for identifikation af den kritiske effekt. Den kritiske effekt er den effekt, der anses for at være den afgørende ved den sundhedsmæssige vurdering. Nuleffekt niveauet (evt. laveste effektniveau) for denne effekt anvendes til beregning af TDI (tolerabelt dagligt indtag/ tolerabelt daglig eksponering), som i den videre proces benyttes til beregning af kvalitetskriteriet.

3.1 Dosis-effekt og dosis-respons sammenhænge

Udsættelse for et kemisk stof kan medføre forskellige typer effekter afhængig af eksponeringsvej, eksponeringens størrelse og varighed. Lettere grader af effekter kan være forbigående genevirkninger i form af fx slimhindeirritation, mens alvorlige effekter kan være dødeligt forløbende akutte forgiftninger eller udvikling af kroniske sygdomme som kræft.

En længerevarende dyreeksperimentel undersøgelse vil således kunne give viden om forskellige typer effekter ved forskellige eksponeringsniveauer (dosis-effekt), og om hvor lang tid det tager, før de optræder i forhold til eksponeringen.

Yderligere vil man fra undersøgelsen kunne se, hvor stor en andel af de doserede dyr der er ramt af effekten (dosis-respons). Visse effekter kan være opdelt i forskellige sværhedsgrader eller stadier, hvor der så for hver af disse kan foreligge dosis-responssammenhænge.



Figur 1 Eksempler på dosisrespons-sammenhænge

Ovenstående figur viser, hvordan dosisrepons-kurver for forskellige effekter kan optegnes ud fra konkrete forsøgsdata med forskellige eksponeringsniveauer og observerede effekter ved disse (markeret med krydser). Kurve a angiver fx dosis-respons forløbet for luftvejsirritation hos forsøgsdyrene, og er her karakteriseret ved at være meget stejl (alle dyr påvirkes inden for et forholdsvist lille dosisinterval, dvs. lille spredning i følsomhed). Kurve b angiver forekomsten af kræftsvulster og viser en dosisrespons sammenhæng ved højere eksponeringsniveauer, og beskriver en noget fladere dosis-respons sammenhæng (dvs. dyrenes følsomhed over for udvikling af kræft er mere spredt). NOAEL-markeringerne på figuren repræsenterer eksponeringsniveauer, hvor der ikke blev fundet statistisk signifikant flere dyr i eksponeringsgruppen end i kontrolgruppen, der udvikler den pågældende effekt, mens LOAEL-værdierne repræsenterer det laveste eksponeringsniveau, der har medført en signifikant forøget forekomst af effekten.

Den faglige baggrundsrapport (Miljøprojekt Nr. 974 (2005)) angiver i afsnit 3.1 mere detaljerede beskrivelser og tolkningen af forskellige dosis-respons forløb.

3.2 Fastsættelse af nul-effektniveau og laveste effektniveau

For langt de fleste typer effekter vurderes der at være en tærskelværdi, der adskiller effektniveauer fra ikke-

effektniveauer, dvs. eksponeringen skal overskride en vis tærskelværdi før dosis er tilstrækkelig til at udløse effekt. Niveauet umiddelbart under denne teoretiske tærskelværdi betegnes nuleffektniveauet, der er den højeste dosis, der ikke medfører effekt. Dosisniveauet umiddelbart over tærskelværdien betegnes laveste effektniveau, da dette er det laveste dosisniveau, der lige netop udløser effekten.

I praksis anvendes for nuleffektniveauet det såkaldte *no observed adverse effect level*, *NOAEL*, der er den højeste af de i forsøget anvendte doser, hvor der i et konkret forsøg ikke er *observeret* den givne effekt. For laveste effekt-niveau anvendes det såkaldte *lowest observed adverse effect level, LOAEL*, der er den laveste dosis i forsøget hvor der er observeret den givne effekt.

I litteraturen anvendes betegnelserne NOAEL og NOEL (*no observed effect level)* samt LOAEL og LOEL (*lowest observed effect level).* Når betegnelserne anvendes korrekt, er det for at sondre mellem om de effektniveauer eller nuleffektniveauer der beskrives, er i forhold til *skadelige (adverse)* effekter eller effekter generelt, hvor også lettere grader af effekter som påvirkning af enzymniveauer og andre effekter af ikke direkte skadelig karakter er omfattet.

Man må dog være opmærksom på, at denne skelnen mellem skadelige og ikke-skadelige effekter i mange tilfælde ikke er gjort konsekvent i litteraturen, og at betegnelserne ofte benyttes i flæng. Det kan således i konkrete tilfælde være vanskeligt at afgøre, hvor grænsen går for, om en effekt skal tolkes som skadelig eller ej, og dermed om et LOEL snarere skal tolkes som et LOAEL.

Se endvidere afsnit 3.2 i den faglige rapport (1).

3.3 Benchmark-metoden

NO(A)EL/ LO(A)EL metoden til udpegning af et eksponeringsniveau er afhængig af, hvilke eksponeringsniveauer man har valgt ved udførelse af et forsøg, og i nogle tilfælde har man måske ikke engang fundet et NO(A)EL. Dette har medført, at en nyere metode, benchmark-metoden, visse

steder har vundet indpas i forbindelse med risikovurderinger, idet der ikke anvendes en NO(A)EL eller LO(A)EL men en benchmark-dosis for den videre beregning af TDI. Ved denne metode foretages en computerbaseret modellering af dosis-responskurven ud fra de tilgængelige data. På dosis-respons kurven findes ED_5 – eller ED_{10}-niveauet (dvs. det eksponeringsniveau, der medfører respons hos 5% eller 10% af de eksponerede). Denne dosis vælges derpå som udgangspunkt (benchmark-dosis, BMD_5 eller BMD_{10}) for den videre beregning på tilsvarende måde som man anvender en NO(A)EL- eller LO(A)EL-værdi.

Metoden anvendes af de canadiske miljømyndigheder og af US EPA, og også af WHO i forbindelse med "Air quality Guidelines for Europe" (2). WHO angiver i denne forbindelse, at en BMD_5 ud fra en gennemsnitsbetragtning kan sammenlignes med et NO(A)EL, mens en BMD_{10} kan sammenlignes med et LO(A)EL.

WHO omtaler også anvendelsen af benchmark-metoden som en alternativ metode i forbindelse med publikation om risikovurdering fra 1999 (3) og i forbindelse med publikation om fastsættelse af vejledende grænseværdier fra 1994 (4).

På nuværende tidspunkt haves der kun sparsom erfaring med anvendelsen af metoden herhjemme. Som det faglige miljøprojekt anfører, kan benchmark-metoden ikke umiddelbart anvendes for alle typer data, og metoden kræver for de relevante effektområder, hvor den kan anvendes, ofte flere dosis-niveauer end der sædvanligvis haves.

Benchmark-metoden er, når der foreligger tilstrækkelige data, et supplement til den traditionelle NO(A)EL/ LO(A)EL metode i forbindelse med fastsættelse af kvalitetskriterier. Uanset hvilken metode man vælger, bør man vurdere og begrunde den fra gang til gang, dvs. man bør referere til og begrunde de anvendte beregningsmetoder eller henvise til de originalreferencer, hvor den benyttede benchmark-dosis er beregnet.

3.4 Udpegning af kritisk effekt

Ved den samlede vurdering af dosis-effekt og dosis-respons sammenhænge foretages udpegning af den kritiske effekt samt udpegning af den NO(A)EL/ LO(A)EL værdi (evt. benchmarkdosis), der vurderes som mest relevant ved fastsættelse af kvalitetskriteriet. Ofte, men ikke nødvendigvis, vil dette være den laveste NO(A)EL/ LO(A)EL værdi, der er rapporteret vedrørende en skadelig effekt eller potentielt skadelig effekt.

Ved udpegning af den kritiske effekt vurderes relevansen af eksponeringsmåden (eksponeringsvejen i en given undersøgelse) i forhold til eksponeringsvejen for det medie (jord, luft eller vand) kvalitetskriteriet skal udarbejdes for. Alvorligheden af forskellige effekter sammenholdes i forhold til, hvor langt de ligger fra hinanden i eksponeringsniveau. Det vurderes fx om en alvorlig effekt kan være en følge af mindre alvorlige effekter, som således kan ses som en forløber og indikator for udvikling af en egentlig skadeeffekt. Endvidere vurderes i det konkrete tilfælde, om der optræder visse artsspecifikke effekter hos forsøgsdyrene, og om dokumentationen er tilstrækkelig til, at disse fund kan vurderes at være irrelevante i forhold til mennesker (se afsnit 3.5).

Ved tvivlstilfælde om udpegning af kritisk effekt og NO(A)EL/LO(A)EL værdi kan det være nødvendigt at udpege flere NO(A)ELs/LO(A)ELs for de potentielt kritiske effekter og anvende disse sideløbende ved den efterfølgende beregning af TDI/ kvalitetskriterier. Dette er for at vurdere, hvordan anvendelse af usikkerhedsfaktorer og andre beregningsmæssige forhold får indflydelse på kvalitetskriteriet, når der anvendes forskellige udgangspunkter m.h.t. kritisk effekt og NO(A)EL/LO(A)EL.

3.5 Relevans af visse effekter i forsøgsdyr

Som udgangspunkt anvendes dyremodeller som troværdige modeller ved forudsigelse af stoffets egenskaber hos mennesker. Visse effekter, der optræder i forsøgsdyr, har vist sig at være tæt knyttet til bestemte dyrearter. I tilfælde, hvor der foreligger dokumentation for at effekterne alene er forårsa-

get af en sådan artsspecifik virkningsmåde, anses disse for at være af meget begrænset relevans ved den videre vurdering af stoffet.

Især inden for kræftområdet har man fundet flere typer svulster (tumorer), hvor relevansen i forhold til mennesker i de enkelte tilfælde bør vurderes nøjere. I Miljøprojekt Nr. 974 (2005) er en række af disse beskrevet i afsnit 3.7.

Referencer

(1) Miljøprojekt Nr. 974 (2005). Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

(2) WHO (2000). Air Quality Guidelines for Europe. WHO regional Publications, European Series, No. 91.

(3) WHO (1999). Principles for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria 210. International Programme on Chemical Safety.

(4) WHO (1994). Assessing human health risks of chemicals: derivation of guidance values for health-based exposure limits. Environmental Health Criteria 170. International Programme on Chemical Safety.

4 Beregning af TDI for stoffer med tærskelværdi

4.1 TDI-begrebet

Det sidste trin i farlighedskarakteriseringen er beregning af tolerabelt dagligt indtag, *TDI (*eller tolerabel koncentration, *TK)*. Ved beregning for stoffer, hvor der anses at være en nedre tærskel for effekt anvendes usikkerhedsfaktorer:

$$TDI\,(TK) = \frac{NO(A)EL^{a}}{UF_{I} \times UF_{II} \times UF_{III}}$$

[a] *Alternativt kan et LO(A)EL eller en Benchmark-dosis* BMD_{x} *anvendes, se afsnit 3.3.*

Den tolerable daglige indtagelse (TDI) er et udtryk for den daglige gennemsnitsdosis (fra alle kilder), som befolkningen vurderes at kunne udsættes for (tolerere) gennem et helt livsforløb, uden at der forventes at opstå sundhedsskadelige effekter.
TDI angives sædvanligvis i enheden mg/kg legemsvægt per dag.

Analogt til TDI kan betegnelsen tolerabel koncentration (TK) defineres som den koncentration af et stof i jord, luft eller drikkevand som befolkningen vurderes at kunne udsættes for (tolerere) gennem et helt livsforløb, uden at der forventes at opstå sundhedsskadelige effekter. TK angives fx i enheden mg/m^3 (luft), mg/l (drikkevand), eller i mg/kg (jord).

For nogle stoffer er det nødvendigt at fastsætte en PTWI – værdi (Provisional Tolerable Weekly Intake – PTWI), der angiver det tolerable ugentlige indtag, i stedet for TDI. PTWI benyttes sædvanligvis over for stoffer, hvor det er

vigtigt at understrege, at det er den samlede eksponering over længere tid, der er af betydning for forekomst af effekter (fx bly og cadmium).

Begrundelse for anvendelse af usikkerhedsfaktorer omtales kort nedenfor, idet en mere detaljeret beskrivelse er givet i Miljøprojekt Nr. 974 (2005) afsnit 4.4

4.2 Anvendelse af usikkerhedsfaktorer

I kommentarerne til Miljøbeskyttelsesloven angives at "Miljøministeriet ved udstedelse af regler og vejledninger kan operere med fx sikkerhedsfaktorer ved fastsættelse af grænseværdier eller retningslinier for forureningsmæssige beregninger på de områder, hvor der ikke foreligger et tilstrækkeligt eksakt vidensgrundlag"

Tidligere har man anvendt begrebet sikkerhedsfaktorer. Det har imidlertid vist sig, at denne betegnelse kan misforstås, således at man forventer større sikkerhed, jo større sikkerhedsfaktor der anvendes. Dette er ikke i overensstemmelse med, hvordan faktorerne anvendes, idet de benyttes for at tage hensyn til usikkerheder og manglende viden i datagrundlaget, dvs. jo større usikkerhed jo større faktor. Ved brugen af disse faktorer tilstræbes det, at der opnås et ensartet beskyttelsesniveau udtrykt ved TDI-værdien. På denne baggrund er det derfor mere korrekt at anvende betegnelsen usikkerhedsfaktorer.

Også i udenlandsk litteratur anvendes forskellige termer for denne faktor fx: safety factor, uncertainty factor, Sicherheitsfaktor, assessment factor, bedömningsfaktor.

Nedenfor angives hvilke elementer og hensyn, der indgår i de tre usikkerhedsfaktorer.

4.2.1 Usikkerhedsfaktor I

Usikkerhedsfaktor I (UF_I) anvendes for at tage højde for, at mennesker kan være mere følsomme over for et givent stof end forsøgsdyr. Denne faktor har historisk været sat til 10.

Ekstrapolation af data fra dyr til mennesker kan opfattes som omhandlende to forskellige aspekter:

1) korrektion af dosis for forskelle i kropsstørrelse mellem forsøgsdyr og mennesker, såkaldt allometrisk skalering, og
2) andre former for forskelle mellem forsøgsdyr og mennesker, som ikke nødvendigvis afspejles i forskellene i kropsstørrelse.

Miljøprojekt Nr. 974 (2005) har gennemgået den eksisterende viden og understøtter, at der fortsat som udgangspunkt anvendes en standardværdi på 10 for UF_I, hvilket også er i overensstemmelse med international praksis, når dosis hos dyr omsættes til human dosis med samme enhed.

Hvis der foreligger veldokumenteret viden om toksikokinetiske og/eller toksikodynamiske forskelle mellem det givne forsøgsdyr og mennesker, anbefales det at tage udgangspunkt i denne viden med henblik på fastsættelse af en dataspecifik faktor i stedet for anvendelse af en standardværdi på 10. I den forbindelse kan 10-faktoren evt. opdeles i underfaktorer, hvor størrelsen af disse "delfaktorer" må vurderes konkret fra gang til gang.

Ved beregning af tolerabel koncentration (TK), hvor udgangspunktet er dyreforsøg, hvor eksponeringen foregår via inhalation, bør ekstrapolering til humaneksponering ved inhalation foretages direkte ud fra eksponeringsniveauet angivet i mg stof /m^3 (omregnet til kontinuerlig gennemsnitskoncentration per dag), frem for at foretage omregning fra indåndet dosis til mg/kg lgv/d for forsøgsdyrene. Ved at anvende indåndingskoncentrationen direkte svarer dette til at doseringen foretages i forhold til stofskiftet, hvilket anses for at være den mest relevante metode, når der skal korrigeres for forskelle i kropsstørrelse. Anvendelse af en UF_I skal ses i lyset heraf og bør derfor i denne situation ligge lavere end 10. En værdi på en halv tierpotens ($10^{0,5}$) kan som udgangspunkt anvendes til resterende forskelle m.h.t. artsforskelle i kinetik og dynamik, medmindre konkrete data tilsiger højere eller lavere værdi.

Der bør i videst muligt omfang tages hensyn til en række mætningsfænomener (fx mætning af absorption, metabolisme eller udskillelse), der kan forekomme i dyreforsøg ved høj dosering, idet disse mætningsfænomener ofte ikke vil være relevante ved lavere miljømæssigt relevante niveauer. I sådanne tilfælde bør der om muligt omregnes til "effektiv" dosis/ koncentration som dyrene har været udsat for.

4.2.2 Usikkerhedsfaktor II

Usikkerhedsfaktor II (UF_{II}) anvendes for at tage højde for, at nogle individer i befolkningen kan være mere følsomme over for et givent stof end den generelle befolkning (for eksempel børn, gravide, ældre, svækkede, kronisk syge). Denne faktor har oftest været sat til 10. Forskellene i følsomhed skyldes den biologiske variation, der findes mellem mennesker. Faktorer som alder, køn, graviditet, genotype, helbred, og livsstil kan være medvirkende til en øget biologisk følsomhed, som afspejler dels forskelle i toksikokinetik og dels i toksikodynamik.

Miljøprojekt Nr. 974 (2005) henviser til en række analyser, der har vurderet variationen mellem mennesker og dermed størrelsen af UF_{II}. Sammenfattende understøtter disse analyser anvendelse af en standardværdi på 10 for denne variation.

Denne usikkerhedsfaktor skal derfor som udgangpunkt sættes til 10. En alternativ værdi kan anvendes, hvis udgangspunktet for NO(A)EL/LO/A)EL værdien specifikt er relateret til data for særligt følsomme personer eller den kritiske effekt er en effekt, hvor man har særligt kendskab til variationsbredden i følsomhed.

4.2.3 Usikkerhedsfaktor III

Usikkerhedsfaktor III (UF_{III}) anvendes for at tage højde for manglende kvalitet og relevans af de tilgængelige data. I relation til fastsættelse af kvalitetskriterier i jord, luft og drikkevand har denne faktor typisk varieret fra 1 til 100 afhængigt af datagrundlaget for de pågældende stoffer.

I UF_{III} indgår bl.a. følgende elementer :

- kvaliteten af datasættet (fx ekstrapolation fra subkronisk nuleffektniveau til kronisk nuleffektniveau),,
- ekstrapolation fra en eksponeringsvej til en anden ("route to route" ekstrapolation fx omregning fra oral dosis til inhalationsdosis),
- ekstrapolation fra LO(A)EL til NO(A)EL,
- og alvorligheden af effekterne (fx kræftfremkaldende effekter).

Det er ikke muligt at pege på en specifik størrelsesorden for en standardværdi, hverken for de enkelte delelementer af UF_{III} eller for den samlede UF_{III}.

Der henvises til Miljøprojekt Nr. 974 (2005) for en nærmere beskrivelse af UF_{III} i rapportens afsnit 4.4.3.

Nedenfor anføres mere kortfattet retningslinierne for fastsættelse af UF_{III}.

Kvalitet og relevans af data

UF_{III} skal tage højde for, om der er kvalitetsmæssige eller datamæssige mangler. Vurdering af kvalitet, omfatter vurdering af om de enkelte undersøgelser (og især undersøgelserne der refererer til de kritiske effekter og effektniveauer) er udført og afrapporteret på en måde, så resultaterne anses for relevante og pålidelige. Selve omfanget af datasættet vurderes for data vedrørende alle relevante effektområder i relation til kortvarig og langvarig eksponering, og det vurderes om der i datasættet er væsentlige mangler i forhold til vurdering af kritisk effekt og estimering af TDI. En faktor på 1 anvendes ved datasæt, som vurderes at være fuldt tilstrækkeligt for det givne stof, mens der ved mindre eller større mangler har været anbefalet faktorer i størrelsesordenen 3-10. I visse tilfælde med store mangler kan en faktor på helt op til 100 komme på tale. Fastlæggelse af UF_{III} må ofte baseres på en ekspertvurdering og foretages under hensyntagen til det enkelte stofs toksikologiske profil.

Et særligt aspekt, er manglende viden om børn og ufødtes følsomhed for konkrete kemiske påvirkninger. For at beskyt-

te børn og ufødte er det vigtigt ved fastsættelse af UF_{III} at vurdere om der er tilstrækkelig viden om disse forhold ud fra reproduktionsforsøg og flergenerationsforsøg. Er disse forhold ikke tilstrækkeligt belyst bør mangel på data have indflydelse på valget af UF_{III}. Størrelsen af en sådan delfaktor må ses i sammenhæng med stoffets toksikologiske profil og hvilke øvrige data, der haves.

Ekstrapolation fra en eksponeringsvej til en anden

Ved mangel på data for den relevante eksponeringsvej kan det blive nødvendigt at foretage en "route-to-route" ekstrapolation. De analyser, der er foretaget med hensyn til ekstrapolation fra oralt NO(A)EL til et NO(A)EC ved inhalation, peger generelt på, at en ekstrapoleret værdi ofte vil være væsentligt højere end en observeret værdi (dvs toksiciteten undervurderes). Det modsatte gør sig generelt gældende når der ekstrapoleres fra oralt NO(A)EL til NO(A)EL ved hudkontakt, hvor den ekstrapolerede værdi ofte vil være lavere end en observeret værdi (dvs. toksiciteten overvurderes). Der kan imidlertid ikke peges på en konkret størrelse for usikkerhedsfaktor III ved disse ekstrapolationer, der hviler på et meget usikkert grundlag. Vurderingen af om en "route-to route" ekstrapolation skal foretages, og i hvilken udstrækning der skal anvendes en usikkerhedsfaktor i tilknytning hertil, må bero på en ekspertvurdering i det konkrete tilfælde.

For stoffer, hvor der kun findes subakutte eller subkroniske undersøgelser, er det ikke muligt at fastsætte et NO(A)EL for livstidseksponering, som generelt må forventes at ligge lavere. Der har været foretaget flere analyser af forholdet mellem NOAELs og LOAELs opnået i studier af forskellige eksponeringsvarigheder. Med baggrund i disse synes der at være belæg for en usikkerhedsfaktor af størrelsesorden minimum 10. En af de nyere analyser har således vist, at en faktor på 10 vil være tilstrækkelig i knap 90% af tilfældene, når der var tale om ekstrapolation fra subkroniske data, mens der ved ekstrapolation fra subakutte data skulle anvendes en væsentlig højere faktor (>20).

Ved ekstrapolation fra LO(A)EL til NO(A)EL kan der ikke peges på en generel faktorstørrelse, som afspejler en generel

usikkerhed. Praksis inden for området og de tilgængelige analyser af problemstillingen peger på, at en værdi omkring en faktor 10 i langt de fleste tilfælde er tilstrækkelig. Vurderingen må imidlertid bero på en ekspertvurdering i hvert enkelt tilfælde.

Med baggrund i vægtning af ovenstående aspekter anvendes en UF_{III} på 10 som udgangspunkt. Denne værdi kan så korrigeres, når der er grundlag for dette i konkrete tilfælde.

Denne problemstilling kan i visse situationer omgås, når det ud fra data er muligt at beregne en benchmark-dosis, således at denne anvendes i stedet for LO(A)EL-værdien.

Alvorligheden af effekter

Det har været praksis ved fastsættelse af kvalitetskriterier, at særligt alvorlige effekter (fx kræftfremkaldende effekt eller fosterbeskadigende effekter) afspejles i usikkerhedsfaktor III. Det gælder især i de tilfælde, hvor den alvorlige effekt optræder ved lave dosisniveauer. Her kan anvendes *en ekstra faktor* på op til 10 for at tage hensyn til dette. For at få den endelige værdi for UF_{III} ganger man de enkelte værdier sammen.

Sammenfattende kan det konkluderes, at det ikke er muligt at sætte en standardværdi for en samlet UF_{III}. Usikkerhederne inden for de forskellige områder må vægtes, når der tages stilling til en samlet faktor for UF_{III}, da en multiplikation af mange delfaktorer i UF_{III} kan give en meget høj værdi, og i visse tilfælde give et skævt billede af det samlede videngrundlag og kvaliteten heraf. Fastsættelse af UF_{III} bør således i høj grad bero på en ekspertvurdering, hvor valget af faktoren og elementerne heri tydeligt begrundes.

4.2.4 Samlet usikkerhedsfaktor

Ved beregning af TDI divideres NO(A)EL evt. LO(A)EL med de tre usikkerhedsfaktorer, der således ganges sammen.

Ved multiplikation af UF_I, UF_{II} og UF_{III} bør der imidlertid tages stilling til størrelsen af den samlede usikkerhedsfaktor,

og der bør foretages en overordnet vurdering i forhold til det givne datasæt.

En meget stor usikkerhedsfaktor, der kan medføre en meget lille TDI-værdi, betyder ikke nødvendigvis, at stoffet er lige så potent eller farligt som andre, mere velkendte stoffer med tilsvarende lave TDI-værdi. Den beregnede lave værdi må snarere ses som en følge af store usikkerheder i datagrundlaget. En samlet usikkerhedsfaktor på 10.000 og derover, bør derfor ikke anvendes.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

5 Beregning af TDI for kræftfremkaldende stoffer

5.1 Kræftfremkaldende stoffer med og uden tærskelværdi

Kræftfremkaldende stoffer anses for at kunne deles op i to grupper grundet deres virkningsmekanisme. Den ene gruppe består af stoffer, der virker kræftfremkaldende som følge af evnen til at påvirke cellernes delings- og differentieringshastighed. Dette kan ske gennem direkte eller indirekte påvirkning af cellernes receptorer. For sådanne stoffer antages der at være en nedre tærskelværdi for denne effekt. Derfor kan TDI beregnes ved hjælp af usikkerhedsfaktorer, som omtalt i forrige afsnit.

Den anden gruppe af kræftfremkaldende stoffer virker gennem kemisk interaktion med cellernes arvemasse, og den kræftfremkaldende effekt vurderes at være en følge af stoffets beskadigelse af cellernes arveanlæg (mutagen/genotoksisk aktivitet). For disse stoffer anses der ikke at være et nedre eksponeringsniveau uden øget risiko for skadevirkninger. For sådanne stoffer anvendes ikke usikkerhedsfaktorer ved beregning af TDI. I stedet beregnes TDI ved hjælp af en matematisk modelberegning, hvor man på forhånd definerer et risikoniveau man vil acceptere, og beregner så hvilken udsættelse, som giver denne risiko.

Ved vurdering af kræftfremkaldende effekt er det således af stor betydning, om det kræftfremkaldende stof falder ind under den ene eller den anden kategori. Der findes stoffer, som både er genotoksiske – dvs. kræftfremkaldende uden tærskel for effekt, og kræftfremkaldende ved andre mekanismer, hvor der anses at være en tærskel. Ved vurdering af denne type stoffer, kræver det en ekspertvurdering at afgøre, om de data der ligger til grund, bedst giver grundlag for at vurdere stoffet som tilhørende den ene eller anden kategori. Der har således i EU været arbejdet på at udvikle

nærmere vejledning i hvordan disse typer stoffer kan vurderes[14].

5.2 Vurdering af kræftfremkaldende effekt

EU, WHO IARC (International Agency for Research on Cancer) og US EPA anvender en række forholdsvis sammenlignelige procedurer/ kriterier ved vurdering af, om et stof skal i kategorien "kræftfremkaldende". Stofferne indplaceres i forskellige underkategorier alt efter dokumentationens omfang, og under hensyntagen til om dokumentationen stammer fra humandata eller dyredata.

Yderligere indgår der i vurderingerne også stillingtagen til virkningsmekanismer, fx om stoffer virker gennem en mutagen/ genotoksisk mekanisme. I EU´s klassificeringssystem for kemiske stoffer er der også kriterier for, hvornår et stof skal kategoriseres som "mutagent".

Principperne for inddeling af kræftfremkaldende stoffer i kategorier er mere udførligt beskrevet i Miljøprojekt Nr. 974 (2005) afsnit 3.6.

Ved vurdering af et stofs kræftfremkaldende effekt i forbindelse med risikokarakterisering og beregning af kvalitetskriterier tages der så vidt muligt udgangspunkt i de ovennævnte vurderinger, idet der suppleres med opdateret viden fra litteraturen. Det vurderes om der er dokumentation for at den kræftfremkaldende effekt er en følge af genotoksisk virkning, og om stoffet derfor skal betragtes som værende uden en nedre tærskel for effekt.

Hvis data taler for, at stoffet virker gennem en ikke-genotoksisk mekanisme, anses stoffet for at besidde en tærskelværdi for skadelig effekt. For en række stoffer vil data og viden vedrørende virkningsmekanisme være meget mangelfuld, og det kan være vanskeligt at sondre, om stoffet skal betragtes som værende enten med eller uden tærskelværdi. Sådanne tvivlstilfælde kan bedst håndteres ved at betragte

[14] European Food Safety Authority (2005). Opinion on a Harmonised Approach for Risk Assessment of Compounds Which are both Genotoxic and Carcinogenic.

stoffet som havende en tærskelværdi, og i den forbindelse anvende en øget usikkerhedsfaktor (UF_{III}) for at tage hensyn til usikkerheden om eksistensen af en tærskelværdi. Der bør således være et vist datagrundlag, der peger hen mod en genotoksisk mekanisme, før stoffet vurderes efter en model uden nedre grænse.

Ved vurderingen af kræftfremkaldende effekt tages stilling til om den er knyttet til bestemte eksponeringsveje, og om disse er relevante i forbindelse med et kvalitetskriterium for stoffet i jord, luft eller drikkevand.

Visse kræftformer kan være forsøgsdyrsspecifikke, og når sådanne kræftfund kan dokumenteres som ikke-relevante i human sammenhæng, tillægges disse fund kun begrænset vægt (se afsnit 3.5).

Ved vurderinger i relation til kvalitetskriterier tages der alene stilling til om stoffet skal betragtes som kræftfremkaldende, og om stoffet ud fra dets virkningsmekanisme skal anses for at have en tærskelværdi eller ej. Der foretages således ikke en mere detaljeret indplacering i forskellige kategorier i forhold til dokumentationens art og omfang, dvs. der inddeles ikke i kategorier for humane og dyreeksperimentelle kræftfremkaldende stoffer.

Et stof, som på baggrund af dokumentationen indplaceres i EU's Carc1 eller Carc 2, i IARC's gruppe 1 eller gruppe 2A/2B, og/eller i US EPA's gruppe A eller B1/B2, vil som udgangspunkt medføre, at stoffet betragtes som kræftfremkaldende, med mindre der er nyere undersøgelser eller velunderbyggede informationer og fortolkninger af data, der taler imod en sådan vurdering.

Tilsvarende gælder stoffer, som er opført på Arbejdstilsynets liste over stoffer, som anses for at være kræftfremkaldende. Det skal dog understreges, at stoffer, som hverken er vurderet af EU, IARC og/eller US EPA eller er indplaceret i en lavere kategori (EU Carc3, IARC gruppe 3 og/eller US EPA gruppe C/D), godt kan blive betragtet som kræftfremkaldende i relation til fastsættelse af kvalitetskriterier for kemikalier i jord, luft og drikkevand, hvis der er velunderbyggede data, der taler for det. Det kan for eksempel være i

tilfælde af, at der er publiceret nye undersøgelser siden en eventuel vurdering er foretaget af EU, IARC og/eller US EPA, eller der kan være stor kemisk strukturlighed med andre kendte kræftfremkaldende stoffer. I sidstnævnte tilfælde kan QSAR-modellering indgå som supplerende støtte for vurderingen.

5.3 Beregning af TDI for kræftfremkaldende stoffer uden tærskelværdi

5.3.1 Tolerabelt risikoniveau

TDI-værdien for kræftfremkaldende stoffer uden tærskelværdi fastsættes til en værdi, der repræsenterer et accepteret risikoniveau for udvikling af kræft. Denne værdi har traditionelt været fastsat til en 10^{-6} livstidsrisiko, og vil også med udsendelse af denne vejledning være det ønskede risikoniveau.

Konkret betyder dette, at man med udgangspunkt i humandata eller dyreforsøg, hvor den kræftfremkaldende effekt er påvist, ved hjælp af matematisk modellering estimerer dosis-respons kurvens forløb så langt ned i lav-eksponeringsområdet, at en eksponering svarende til en 10^{-6} livstidsrisiko kan beregnes. TDI er således den daglige gennemsnitseksponering, der ud fra *teoretiske beregninger* svarer til en forøget risiko for cancer på 1 ud af en million mennesker, som er udsat for stoffet gennem en hel livstid.

5.3.2 Metode til beregning af TDI og livstidsrisiko

Der er udviklet forskellige metoder til beregning af, hvor stor en risiko for udvikling af svulster en given eksponering for et genotoksisk kræftfremkaldende stof udgør. For alle metoder gælder, at der anvendes en eller anden form for matematisk ekstrapolation fra dosisniveauer med de kendte eksperimentelle værdier for forekomsten af svulster til de som regel meget lavere dosisniveauer, der svarer til en forekomst hos 1 ud af en million. Metoderne beskriver relationen mellem den daglige eksponering (udtrykt som dosis

eller koncentration) og den sammenhørende sandsynlighed for udvikling af svulster.

Beskrivelse af forskellige metoder og sammenligning mellem disse fremgår af Miljøprojekt Nr. 974 (2005) afsnit 5.3 og 5.4.

Den foretrukne metode til fastsættelse af TDI er T25-metoden, som også anvendes i EU-regi. Den baserer sig ligesom one-hit metoden på lineær ekstrapolation med udgangspunkt i laveste dosisniveau, hvor der optræder signifikant forøget antal svulster[15].

Metoden er en simplificeret lineær metode med udgangspunkt i en beregnet T25-dosis. T25-dosis defineres i denne sammenhæng som den kroniske eksponering (enhed: mg/kg legemsvægt per dag eller mg/m^3), som vil give 25% af forsøgsdyrene svulster i et specifikt væv, efter korrektion for den spontane hyppighed, indenfor den standardiserede levetid for den pågældende dyreart. Med udgangspunkt i denne T25-dosis foretages lineær ekstrapolation ved simpel forholdsregning ned til en dosis, der svarer til et tolerabelt risikoniveau.

Metoden og dens konkrete anvendelse i forhold til et tolerabelt 10^{-6} livstidsrisikoniveau er nøjere beskrevet i bilag 1.

5.3.3 Anvendelse af risikoestimater angivet i litteraturen

I en række tilfælde vil eksponeringsniveauet svarende til en 10^{-6} livstidsrisiko (eller "unit-risk"-estimater) på forhånd være beregnet af ekspertgrupper under fx WHO eller US EPA. For især nyere vurderinger kan det være relevant at anvende disse værdier som ligeværdigt udgangspunkt på

[15] Miljøstyrelsen har tidligere som administrativ praksis anvendt one-hit metoden. Anvendelse af T25-metoden vil i forhold til den medføre lidt højere værdier (skønsmæssigt en faktor 2-4). Denne forskel skal dog ses i et 10^{-6} perspektiv, og må anses som meget beskeden, og i praksis uden betydning i forbindelse med det tilstræbte beskyttelsesniveau.

linie med T25-metoden ved beregning af TDI og kvalitetskriterier.

Argumenterne for at vælge et risikoestimat frem for et andet kan dels være rent faglige (fx bero på konkrete virkningsmekanismer) og dels være mere pragmatiske, idet man også bør vurdere nødvendigheden af at foretage selvstændige beregninger af et risikoniveau. For eksempel kan opgaven være meget omfattende for stoffer med stor datarigdom (fx PAH-stoffer), hvor en selvstændig vurdering vil kræve ulige mange resurser og inddragelse af høj faglig ekspertise på området for at kunne leve op til kvaliteten af en international vurdering.

Derfor vil man i konkrete tilfælde kunne basere kvalitetskriterier for kræftfremkaldende stoffer på andre metoder end T25 metoden.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

European Food Safety Authority (2005). Opinion on a Harmonised Approach for Risk Assessment of Compounds Which are both Genotoxic and Carcinogenic.

6 Beregning af kvalitetskriterier for kemikalier

Dette kapitel beskriver hvorledes kvalitetskriterierne for kemikalier i jord, luft og drikkevand kan beregnes med udgangspunkt i førnævnte TDI-værdier.
Første led i beregningen er en vurdering af, om hele TDI-værdien eller kun en brøkdel heraf tildeles til beregning af det konkrete kvalitetskriterium. Dette kan dels være begrundet i eksponeringsmæssige overvejelser - den såkaldte allokering, hvor der tages hensyn til evt. andre kilder, der har betydning for eksponeringen. Eller det kan være begrundet i øvrige forhold, der kan være afgørende for, om der anvendes en reduktionsfaktor, så kun en mindre brøkdel af TDI-værdien anvendes ved beregning af kvalitetskriteriet.

Kapitlet angiver hvilke værdier for medieeksponering der anvendes ved beregning af kvalitetskriterierne. En mere uddybet forklaring på valget af disse værdier er givet i bilag 2.

6.1 Generel metode for beregning af et kvalitetskriterium

Et sundhedsmæssigt baseret kvalitetskriterium for et stof i jord, luft eller drikkevand beregnes ud fra den tolerable daglige indtagelse, ved at dividere TDI-værdien med den daglige eksponering for det relevante medie WHO/IPCS[16,17]. Det vil sige, at selve beregningen af kvalitetskriteriet ud fra TDI i princippet er ens for de tre medier, jord, luft og drikkevand

[16] WHO/IPCS (1994). Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. Environmental Health Criteria no. 170. International Programme on Chemical Safety.
[17] WHO/IPCS (1999). Principels for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria no. 210. International Programme on Chemical Safety.

og for alle stoffer, uanset om der foreligger tærskelværdi for den kritiske effekt eller ej.

Følgende generelle beregningsmetode kan opstilles:

$$KK_{l,j,v} = \frac{TDI \times V \times f}{E_{l,j,v}}$$

hvor

$KK_{l,j,v}$: kvalitetskriterium for jord, luft eller drikkevand
TDI: angives i mg/ kg legemsvægt/ d
V: legemsvægt i kg
f: allokeringsfaktor, brøkdel af TDI som ud fra eksponeringsfordeling anvendes til eksponering fra jord, luft eller drikkevand
$E_{l,j,v}$: daglig udsættelse/ forbrug af luft (m^3/d), jord (kg/d), eller drikkevand (liter/d).
(Hvis $E_{l,j,v}$ angives i (m^3/kg lgv/ d), (kg/kg lgv/d), eller (liter/kg lgv/d) udgår V i ovenstående formel).

6.1.2 Anvendelse af TDI

For stoffer hvor der ikke anses at være en tærskelværdi for effekt, dvs. de genotoksiske kræftfremkaldende stoffer, anvendes TDI-værdien (sv.t til en 10^{-6} livstidsrisikodosis) direkte til beregning af kvalitetskriteriet.
For andre stoffer, hvor der anses at være en tærskelværdi, indgår efterfølgende overvejelser vedrørende anvendelse af en allokeringsfaktor eller reduktionsfaktor.

Allokering

Allokeringsfaktoren "*f*" angiver den brøkdel af TDI som tildeles udsættelsen via det enkelte medie. Ofte tildeles mindre end 100% af et kemisk stof til beregning af et kvalitetskriterium, da der for en række kemiske stoffer vil være udsættelse gennem andre medier end det enkelte medie jord, luft og vand. I en række tilfælde vil bidrag gennem forurening af fødevarer eller gennem påvirkning fra indeklimaet udgøre hovedkilden for en persons udsættelse, og vil dermed "lægge beslag på" en betydelig del af TDI-værdien, hvorfor der kun tildeles en mindre andel til kvalitetskriteriet.

I de tilfælde hvor bidrag fra andre medier ikke forventes, fastsættes kvalitetskriterierne som udgangspunkt ved, at hele TDI allokeres til det pågældende medie. Når det vurderes, at der foreligger øvrige betydende kilder allokeres som udgangspunkt 10% af TDI til det pågældende medie, med mindre der er konkret viden om øvrige kilders bidrag, der siger noget andet. For visse stoffer, hvor langt de største bidrag kommer fra øvrige kilder, kan der således allokeres helt ned til kun 1% af TDI (dette er for eksempel gjort for visse plastblødgørere i forbindelse med et kvalitetskriterium for drikkevand).

Reduktionsfaktor

I særlige tilfælde kan andre forhold end de rent eksponeringsmæssige betragtninger medføre, at der anvendes en reduktionsfaktor, således at der kun anvendes en vis brøkdel af TDI til beregning af kvalitetskriteriet. En sådan faktor kan efter individuel vurdering komme på tale fx :

- ved særligt kritiske forhold som persistens og bioakkumulering af stoffet,
- i tilfælde, hvor kvalitetskriteriet er udarbejdet ud fra et enkelt stofs effekter, men hvor stoffet repræsenterer en hel stofgruppe, og hvor eksponeringen typisk vil være karakteriseret ved en blandingseksponering med denne stofgruppe,
- i tilfælde hvor der samtidig kan optræde eksponering fra flere stoffer, og hvor der er formodning om, at der ved de relevante niveauer vil kunne optræde kombinationseffekter (fx samvirkende effekter) fra denne blandingseksponering.

6.1.3 Eksponeringsbetragtninger

Målet for eksponeringen i nævneren i brøken til kvalitetskriterie-beregningen baseres på standardbetragtninger for daglig udsættelse.

I afsnit 6 i Miljøprojekt Nr. 974 (2005) er der foretaget en opdateret gennemgang af viden om befolkningens udsættelse for jord, luft og vand, samt angivet hvilke standardbe-

tragtninger der anvendes inden for WHO, US EPA og i EU.

Ud fra hensigten om specifikt at tage hensyn til børns eksponering er der foretaget en revision af den hidtidige praksis. Grundlaget for den reviderede praksis er anført i bilag 2.

6.2 Beregning af luftkvalitetskriteriet

Følgende fremgangsmåde anvendes til beregning af luftkvalitetskriteriet (KK_{luft}), når TDI for de(n) kritiske effekt(er) er fra studier hvor stoffet er givet via munden og angivet i enheden mg/kg legemsvægt/dag:

$$KK_{luft} = \frac{TDI \times f}{E_{luft}} \quad \text{hvor}$$

TDI: tolerabel daglig indtagelse (mg/ kg lgv/d)
f: brøkdel af TDI, der allokeres til udeluften
E_{luft}: eksponering luft, standardværdi for dagligt indåndingsvolumen: 0,5 m^3/kg lgv/d for 1-5 årige børn.

Følgende fremgangsmåde anvendes, når (nul)effektniveauer NO(A)EL eller LO(A)EL) for de(n) kritiske effekt(er) er fra studier hvor stoffet er inhaleret og angivet som en koncentration i enheden mg/m^3:

$$KK_{luft} = TK \times f$$

f: allokeringsfaktor
TK: tolerabel koncentration

$$\text{hvor} \quad TK = \frac{NO(A)EC \text{ eller } LO(A)EC}{UF_I \times UF_{II} \times UF_{III}}$$

For de fleste typer af systemiske effekter anses det at være den samlede dosis og ikke stoffets koncentration i luften, der er af betydning for udvikling af disse effekter. Ved fastsæt-

telse af luftkvalitetskriteriet i disse tilfælde foretages en omregning af det fastlagte (nul)effektniveau til et gennemsnitligt døgnniveau (kontinuert eksponering) ud fra de i studiets aktuelle eksponeringsbetingelser. Det vil sige, at der kompenseres for, at eksponeringen ikke har foregået i alle døgnets timer over en fuld uge. Hvis eksponeringen for eksempel er foretaget 6 timer per dag i 5 dage per uge, korrigeres der med en faktor 6/24 til kontinuert eksponering gennem et helt døgn og en faktor 5/7 til kontinuert eksponering gennem hele ugen.

For visse lokale effekter (effekter, der optræder lokalt i luftvejene samt direkte effekter på hud og øjne) anses det sædvanligvis at være stoffets koncentration i luften og ikke den samlede dosis som sådan, der er af betydning for udvikling af disse effekter. For sådanne stoffer kan omregning til en kontinuert eksponering sædvanligvis udelades fra en konkret vurdering.

Lugt

Nogle kemiske stoffer har en meget kraftig lugt, og hensyntagen til lugt ved fastsættelse af luftkvalitetskriteriet vil for mange stoffer (fx en række organiske opløsningsmidler) medføre lavere luftkvalitetskriterium end det sundhedsbaserede luftkvalitetskriterium, idet luftkvalitetskriteriet fastsættes til 1/3 af 50 %-lugtgrænsen.

Lugtgrænsen for et kemisk stof er generelt defineret som den koncentration i luften, hvor 50 % af et lugtpanel (kontrolleret laboratorieforsøg med bestemmelse af lugtgrænse med frivillige forsøgspersoner) kan registrere lugten.

Som et luftkvalitetskriterium vurderes en sådan 50 % lugtgrænse at kunne medføre gener hos en ikke uvæsentligt del af befolkningen.

I en analyse af en række laboratoriedata, hvor lugtgrænsen er blevet bestemt for kemiske stoffer, viser det sig, at dosis-respons sammenhængen generelt er ret stejl for et lugtpanel (DK-Teknik 2001). For personer, med intakt lugtesans (svarende til personer, der indgår i et lugtpanel), er der ge-

nerelt en forholdsvis lille spredning m.h.t. hvornår en lugt kan opfattes. Ud fra disse data vurderes, at ved et niveau på 1/3 af lugtgrænsen vil maksimalt 1-5% af befolkningen under optimale betingelser kunne fornemme lugt.

Lugtgrænser, der er angivet i litteraturen, kan variere voldsomt (flere størrelsesordner) Dette skyldes, at metoder til bestemmelse af lugtgrænser kan variere meget, alt efter hvor og hvornår de er blevet foretaget, samt at der kan være foretaget undersøgelser af forskellige stofkvaliteter, hvor stofferne ikke er entydigt definerede.

Medmindre lugtgrænsen er bestemt ud fra nye og meget velbeskrevne metoder, hvor kvalitet og pålidelighed af undersøgelsen kan vurderes, anbefales det at fastsætte luftkvalitetskriterier på baggrund af lugt fra bestemmelse af lugtgrænse foretaget af et akkrediteret laboratorium.

6.3 Beregning af jordkvalitetskriteriet

Følgende fremgangsmåde anvendes ved beregning af jordkvalitetskriteriet, når de(n) kritiske effekt(er) er en følge af gentagen udsættelse:

$$KK_{jord} = \frac{TDI \times V \times f}{E_{I,jord} \text{ (eller } E_{H,jord})} \quad \text{hvor}$$

f: procentdel af TDI, der allokeres til indtagelse af jord
V: legemsvægt, 1-3 årigt barn: 13 kg
$E_{I,jord}$: daglig eksponering (indtagelse) for jord, standardværdi:

1) 0,0002 kg/d (sv.t. 95-percentilgrænsen) i tilfælde hvor hele TDI-værdien eller hovedparten af denne anvendes til beregning af kvalitetskriteriet
2) 0,0001 kg/d (sv.t. medianudsættelse) i tilfælde hvor TDI er en 10^{-6} livstidsrisikodosis for et kræftfremkaldende stof, eller i tilfælde, hvor der anvendes en mindre del af TDI til jordkvalitetskriteriet

$E_{H,jord}$: daglig eksponering (hudkontakt) for jord, standardværdi: 0,001 kg/d for barn.

$E_{H,jord}$ anvendes i forbindelse med særligt hudgennemtrængelige stoffer, hvor systemisk bidrag fra hudoptag summeres med det orale bidrag. $E_{H,jord}$ anvendes separat hvis den kritiske effekt er relateret til den direkte hudpåvirkning af forureningskomponenten.

Følgende fremgangsmåde anvendes ved beregning af jordkvalitetskriteriet, når den kritiske effekt er akut toksicitet:

$$KK_{jord} = \frac{TD \times V}{E_{I,jord} \text{ (eller } E_{H,jord})} \quad \text{hvor}$$

TD: tolerabel enkeltdosis

$$\text{hvor} \quad TD = \frac{NO(A)EL_{akut} \text{ eller } LO(A)EL_{akut}}{UF_I \times UF_{II} \times UF_{III}}$$

V: legemsvægt, 1-3 årigt barn: 13 kg
E_I: maksimum enkeltindtag af jord (0,010 kg)
E_H: maksimal hudkontakt med jord (0,010 kg) (Anvendes for særligt hudgennemtrængende stoffer).

Ved beregning bør data for optagelse fra mave-tarmkanalen (biotilgængelighed[18]) så vidt muligt inkluderes, idet de forurenende stoffer i visse tilfælde kan binde sig kraftigt til jordpartikler og derved medføre en reduceret biotilgængelighed. Hvis data ikke foreligger, regnes med samme biotilgængelighed, som i de forsøg, der ligger til grund for TDI-beregningen (dette kan typisk være forsøg, hvor teststoffet er opblandet i foderet eller i drikkevandet).

[18] "Biotilgængelighed" refererer i denne sammenhæng til det engelske "bioaccessibility". Bioaccessibility beskriver den pool af stoffet, som jorden kan frigive, og den indeholder således også let bundne former.

Lugt, udseende

Ud over rent sundhedsmæssige aspekter tages der ved fastsættelse af jordkvalitetskriteriet hensyn til, at jorden ikke må lugte eller syne forurenet. Det vil sige, at jorden ikke ved inspektion må afgive lugt fra forureningen eller se forurenet ud (klumper af stof(fer) eller misfarvning. Der foreligger ikke nøjere retningslinier for en sådan subjektiv vurdering. Med hensyn til lugt i forbindelse med afdampning henvises til Miljøprojekt Nr. 974 (2005) afsnit 7.4.3, der beskriver anvendelsen af luftkvalitetskriteriet i forbindelse med afdampning af forurenende stoffer fra jord.

6.4 Beregning af drikkevandskvalitetskriteriet

Følgende fremgangsmåde anvendes ved beregning af drikkevandskvalitetskriteriet:

$$KK_{drikkevand} = \frac{TDI \times f}{E_{drikkevand}} \quad \text{hvor}$$

TDI: tolerabelt dagligt indtagelse (mg/ kg lgv/d)
f: er den procentdel af TDI, der allokeres til indtagelse af drikkevand
$E_{drikkevand}$: daglig eksponering for drikkevand, standardværdi:

1) 0,08 liter/ kg lgv/d (sv.t. 95-percentilen) for 1-10 årige børn. Anvendes i forbindelse med akutvirkende stoffer eller når hovedparten af TDI-værdien benyttes til beregning af drikkevandskvalitetskriteriet.
2) 0,03 liter/ kg lgv/d (sv.t. medianværdi for 1-10 årige børn) i tilfælde hvor TDI er en 10^{-6} livstidsrisikodosis for et kræftfremkaldende stof, eller i tilfælde hvor kun en mindre andel af TDI-værdien benyttes til beregning af drikkevandskvalitetskriteriet.

Kemiske stoffer, der er særligt hudoptagelige, vil kunne optages i en ikke uvæsentlig mængde i forbindelse med badning, og stoffer, der let fordamper (for eksempel mange opløsningsmidler), kan især ved brusebadning indåndes som dampe/aerosoler. Omfanget af disse eksponeringsformer via brusebadning og karbadning afhænger af, hvor letoptageligt stoffet er gennem huden, og i hvor stor udstrækning stoffet frigives ved fordampning fra vandet. Der bør derfor i konkrete tilfælde (stoffer med høj hudgennemtrængelighed og stoffer med høj flygtighed fra vandfasen), tages højde for disse bidrag ved fastsættelse af drikkevandskvalitetskriteriet, således at den samlede optagelse via drikkevand og badevand ikke overskrider den del af TDI, som er allokeret til drikkevand.

Der er ikke opstillet konkrete modeller for beregningen af sådanne bidrag, hvorfor bidragene må vurderes fra sag til sag under inddragelse af de data og de vurderinger, der er foretaget for det konkrete stof.

Lugt, smag, udseende

Drikkevandet må ikke lugte, smage eller syne forurenet og smag, lugt og udseende af drikkevandet har en væsentlig betydning, også selv om det ikke udgør en sundhedsfare i relation til indtagelse af drikkevand.

Det vil i nogle tilfælde være lugt, smag, eller udseende, og ikke stoffets sundhedsmæssige effekter, der er bestemmende for værdien af drikkevandskvalitetskriteriet.

Lugt- og smagsgrænser, der findes opgivet i litteraturen, kan variere voldsomt selvom de forskellige undersøgelser er udført med samme stof. Dette skyldes, at metoder kan variere meget alt efter hvor, og hvornår de er blevet foretaget.

Medmindre lugt- og smagsgrænsen er bestemt ud fra nye og meget velbeskrevne metoder, hvor kvalitet og pålidelighed af undersøgelsen kan vurderes, anbefales det, at fastsættelse af drikkevandskvalitetskriterier på baggrund af lugt og smag foretages ud fra bestemmelse af lugt- og smagsgrænse foretaget af et akkrediteret laboratorium.

Udgangspunktet for fastsættelse af et lugt- og smagsbaseret kvalitetskriterium bør være et nul-effekt-niveau (NOEL) i testpanelet, dvs. den koncentration hvor testpanelet som helhed ikke kan lugte eller smage stoffet.

Ofte angives lugt- og smagsgrænser i vand på tilsvarende måde som lugtgrænse i luft, dvs. det niveau, hvor 50 % af et testpanelet kan lugte/smage stoffet. Angives således lugtgrænsen i den tilgængelige litteratur som 50 %-grænsen ganges denne værdi med 1/3 (som beskrevet under luftkvalitetskriteriet), idet anvendelse af en sådan faktor skønnes at sikre, at kun en mindre andel af befolkningen vil kunne fornemme stoffet ved dette niveau.

Referencer

DK-Teknik (2001). Vurdering af lugttærskelværdier. Rapport udarbejdet for Miljøstyrelsen.

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

Bilag 1

Kvantitativ vurdering ved benyttelse af T25 ekstrapolationsmetoden.

Anbefaling af T25-metoden

Der er udviklet forskellige metoder til beregning af, hvor stor en risiko for udvikling af svulster en given eksponering for et genotoksisk kræftfremkaldende stof udgør. Ens for dem alle er at der anvendes en form for statistisk ekstrapolation fra dosisniveauer med kendte eksperimentelle værdier til de som regel meget lavere dosisniveauer, der oftest er relevante i relation til den generelle befolknings eksponering for kemiske stoffer i miljøet. Metoderne beskriver relationen mellem den administrerede daglige dosis (eller koncentration) og den resulterende hyppighed af svulster.

Som anført i afsnit 5.3.2. anbefales det at anvende T25-ekstrapolering som beskrevet i en baggrundsrapport udarbejdet af en arbejdsgruppe i forbindelse med EU´s risikovurderingsarbejde (EU-Commission 1999).

T25-metoden er blevet evalueret ved at sammenligne resultater opnået ved denne metode med resultater opnået ved anvendelse af US EPA´s LMS-metode (Linearised Multistage Model) og ved LED_{10} metoden, hvor der foretages lineær ekstrapolation ned i lavdosisområdet ud fra en benchmark-dosis på 10 % effektniveau (se afsnittene 5.3.2-4 i Miljøprojekt Nr. 974 (2005)). Ved gennemgang af resultaterne for lavdosis-estimaterne vurderede man at de er sammenlignelige.

Ved sammenligning af one-hit metoden og T25-metoden vurderes de at medføre sammenlignelige værdier, idet udgangspunktet for begge vurderingerne i princippet er at anvende laveste dosis med signifikant respons, og herfra

foretage lineær ekstrapolation ned i lavdosisområdet. I T25-metoden divideres med en skaleringsfaktor fra dyr til menneske opløftet i ¼ (sv.t. skalering i forhold til stofskiftet), mens tilsvarende skaleringsfaktor i one-hit metoden er opløftet til 1/3 (sv.t. skalering i forhold til overfladeareal). Dette forhold alene vil maksimalt bevirke en forskel på de to metoder på ca. en faktor 2.

Anvendelse af T25-metoden

T25 defineres som den kroniske dosis (enhed: mg/kg legemsvægt per dag), som vil give 25 % af forsøgsdyrene svulster i et specifikt væv, efter korrektion for den spontane hyppighed, inden for den standardiserede levetid af den pågældende art.

T25 beregnes med udgangspunkt i et langtidscancerstudie, hvor den laveste dosis, der giver en signifikant forøgelse af forsøgsdyr med svulster i et specifikt væv, som udgangspunkt anvendes ved beregningen af T25. Forekomsten af ondartede og godartede svulster sammenlægges, når de godartede svulster må mistænkes for at kunne udvikles til ondartede. Hvis der er en højere hyppighed ved en højere dosis, der giver en lavere T25, anvendes sidstnævnte, med mindre der er særlige begrundelser for ikke at tage udgangspunkt i denne. Hvis der er flere data- sæt, beregnes T25 for det mest relevante datasæt. Hvis forskellige datasæt giver T25-værdier, som ligger inden for et relativt snævert interval, anvendes gennemsnittet af disse T25-værdier. Hvis sidstnævnte procedure ikke anvendes, skal rationalet for anvendelse af en anden procedure begrundes nøje.

Beregning af T25 foretages ved at gange dosis D (mg/kg lgv/dag), hvor signifikant forøget antal svulster forekommer med faktoren 0,25/p, hvor p er den aktuelle hyppighed af svulster: $T25 = D \times 0{,}25/p$.

Ud fra denne T25 foretages lineær ekstrapolation ned i lavdosisområdet, idet dosis svarende til et givent risikoniveau beregnes ved simpel forholdsregning. En gennemsnitlig daglig dosis svarende til en øget livstidsrisiko på 10^{-6} kan således beregnes på følgende måde:

$$Dosis_{(10\text{-}6\ livstidsrisiko)} = 10^{-6} / 0{,}25 \times T25\text{-dosis}$$

Imidlertid indgår der i beregningerne også dosiskorrektion med baggrund i en skaleringsfaktor mellem dyr og mennesker, og en faktor som angiver andelen af dyrenes levetid, hvor eksponeringen har fundet sted.

Konkret kan TDI svarende til en 10^{-6} livstidsrisiko beregnes ud fra følgende formel, når T25 er beregnet ud fra forsøg med oral eller dermal eksponering hos dyr:

$$TDI = \frac{I_t \times [L_e / L]^2 \times [(T25 \times l_e) / L_e]}{0{,}25 \times [W_h / W_a]^{0,25}}$$

I_t: Den tolerable livstidsrisiko (10^{-6}).
L_e: Den aktuelle levetid for dyrene.
L: Den teoretiske gennemsnitslængde af levetiden for dyrene.
T25: Beregnet daglig dosis (mg/kg legemsvægt per dag), der medfører en 25% forøget forekomst af tumorer hos forsøgsdyrene.
l_e: Eksponeringstid.
$[W_h / W_a]^{0,25}$: Dosiskorrektion på basis af stofskifte
hvor
W_h: Menneskets vægt i kg (sættes oftest til 70 kg).
W_a: Gennemsnitsvægt af det pågældende forsøgsdyr (kg).

L, L_e l_e:
Hvis eksponeringsvarigheden er kortere end standardlevetiden for den pågældende art, eller hvis studiet afsluttes inden standardlevetiden, foretages dosiskorrektion, som beskrevet i den anførte formel. Standardlevetiden for mus, rotter og hamstre sættes med mindre andet er angivet for den specifikke stamme til 24 måneder.

Dosis (T25)
Når konkrete data ikke specifikt angiver dyrenes legemsvægt, og dosis i mg/kg lgv/d i forbindelse med dosering gennem foder eller drikkevand, anvendes følgende værdier i beregningen:

Dyreart	Legemsvægt (kg)	Indtagelse af foder (g/kg/dag)	Indtagelse af drikkevand (ml/kg/dag)
Rotte	0,10 (ung) 0,40 (ældre)	100 50	 75
Mus	0,020	150	-
Marsvin	0,75	40	-
Kanin	2,0	30	-
Hund	10,0	25	-
Abe	5,0	50	-

$(W_h/W_a)^{0,25}$

Er stoffet givet oralt eller dermalt , foretages der en dosiskorrektion for forskelle i kropsstørrelse mellem dyr og mennesker ved at omregne T25 til den tilsvarende humane dosis i mg/kg lgv./dag ved allometrisk skalering på basis af stofskiftet (afsnit 4.4.1.2). Denne skalering opnås ved at dividere T25-dosis med faktoren $(W_h/W_a)^{0,25}$

Ved beregning ud fra inhalationsundersøgelse beregnes den tolerable indåndingskoncentration TC ved anvendelse af formlen:

$$TC = \frac{I_t \times [L_e / L]^2 \times [(T25 \times l_e) / L_e]}{0{,}25}$$

I_t: Den tolerable livstidsrisiko (10^{-6}).
L_e: Den aktuelle levetid for dyrene.
L: Den teoretiske gennemsnitslængde af levetiden for dyrene.
T25: Beregnet daglig dosis (mg/m³ i indåndingsluften), der medfører en 25% forøget forekomst af tumorer hos forsøgsdyrene.
l_e: Eksponeringstid.

Ved indsættelse af T25 anvendes koncentration i indåndingsluften og der foretages sædvanligvis omregning til en gennemsnitligt indåndingskoncentration , hvis forsøget ikke er udført med kontinuerlig eksponering. Dette gøres ved at korrigere for antal timer pr. dag og antal dage pr. uge hvor eksponeringen har fundet sted.

Referencer

EU-Commission (1999). Guidelines for quantitative risk characterisation of non-threshold carcinogens in the framework of Existing Chemicals following Council Regulation (EEC)

793/93. Commission working Group on the Technical Meetings for Risk Assessment for Existing Substances. Document NO_NL/01/99_Rev.1

Bilag 2

Baggrund for anvendte eksponeringsværdier ved beregning af kvalitetskriterier

I denne vejledning ændres praksis med hensyn til de eksponeringsværdier, der anvendes ved beregning af kvalitetskriterierne for kemikalier.

Tidligere har man i forbindelse med beregning af jordkvalitetskriterier som udgangspunkt antaget, at et barn dagligt indtager 0,2 g jord/d (0,0002 kg/d), mens man ved beregning af luftkvalitetskriterier har anvendt et scenarie, hvor en voksen person dagligt indånder 20 m^3 luft. Ved beregning af kvalitetskriterier for kemikalier i drikkevand har udgangspunktet været et dagligt indtag på 2 liter vand for en voksen.

Børn
Beskyttelse af børn er de senere år i stigende grad kommet i fokus, og det har i den forbindelse været debateret i hvilken udstrækning kvalitetskriterier beregnet ud fra voksnes udsættelse i tilstrækkeligt omfang beskytter børn, da børn i forhold til deres legemsvægt generelt indtager/ indånder en større mængde drikkevand/ luft.

I miljøprojekt nr. 589 "Children and the unborn child – exposure and susceptibility to chemical subtances-" fra 2001, blev der foretaget en detaljeret gennemgang af børns særlige følsomhed og udsættelsesmønster i forbindelse med miljøforureninger og kemiske stoffer. I miljøprojektet konkluderes, at der ved fremtidig fastsættelse af kvalitetskriterier for kemikalier bør tages udgangspunkt i eksponeringsværdier for børn, for at opnå at børn er fuldt omfattet af det beskyttelsesniveau, kvalitetskriteriet repræsenterer. Denne vejledning indarbejder således ønsket om, at principperne for fastsættelse af kvalitetskriterier tager hensyn til børns særlige udsættelse.

Børns udsættelse vil således være udgangspunktet for de fremtidige standardværdier for eksponering i forbindelse med jord, luft og drikkevand.

Standardværdien for børns legemsvægt vil i fremtiden være 13 kg.

Særligt udsatte undergrupper
Almindelig biologisk variation og forskellige former for adfærd vil betyde, at nogle grupper vil være mere udsatte end andre for en given påvirkning via miljøet. Der er således også med denne vejledning taget stilling til, i hvilken udstrækning kvalitetskriterier/grænseværdier for kemikalier skal tage hensyn til de mere udsatte grupper i befolkningen.

Når kvalitetskriterier for kemikalier beregnes ved at anvende eksponeringsværdier, der svarer til befolkningsvægtede gennemsnitsværdier eller medianværdier, betyder dette, at ca. halvdelen af den befolkning, som kvalitetskriteriet skal søge at beskytte, vil kunne blive udsat for *større* eksponering end udgangspunktet for beregningen (fx en TDI-værdi). Hvis sigtet med et kvalitetskriterium er, at størstedelen af befolkningen skal være omfattet at det ønskede beskyttelsesniveau, vil det således være nødvendigt at anvende en øvre fraktilværdi for befolkningens udsættelse.

Det skal nævnes, at de standardværdier der hidtil har været anvendt for børn (jord) og voksne (luft og drikkevand) jf. beskrivelsen af eksponeringsværdier, svarer til sådanne øvre percentilgrænser.

Kvalitetskriterier for kemikalier skal fortsat sigte mod at beskytte flertallet af befolkningen, dvs. også de mere udsatte undergrupper. I de tilfælde hvor befolkningsfordelingen af eksponeringerne kendes, vil valg af øvre eksponeringsværdier typisk kunne foretages ved at tage udgangspunkt i værdier svarende til 90- eller 95-percentilerne.

For at sikre særligt udsatte grupper anvendes følgende principper :

a) I situationer, hvor man ved beregning af kvalitetskriteriet har tildelt hele TDI-værdien, hovedparten eller der er eksakt

viden om størrelsen af udsættelsen via det aktuelle medie, vil det være nødvendigt at anvende øvre percentilgrænser for eksponering. Herved sikres at kun en mindre andel af befolkningen vil blive eksponeret over TDI-niveau.

b) *I andre tilfælde, hvor der anvendes en reduceret TDI-værdi som følge af anvendelse af en allokeringsfaktor (evt. en særlig reduktionsfaktor), anvendes derimod medianværdier for udsættelse med mediet. Dette forhold begrundes med, at anvendelse af en allokeringsfaktor eller reduktionsfaktor sædvanligvs betyder anvendelse af runde værdier (10 % eller 1% af TDI), som ofte vil være på "den sikre side". I sådanne tilfælde, hvor der i forvejen er indbygget en øget grad af sikkerhed, vil det ikke være påkrævet også at anvende en øvre percentilværdi for eksponering. Anvendelse af en medianværdi vurderes her, at kunne opfylde målet om at beskytte størstedelen af befolkningen.*

c) *For kræftremkaldende stoffer uden tærskelværdi for effekt foretages som tidligere nævnt ingen allokering af TDI. I disse tilfælde anvendes medianværdier for eksponering ved beregning af kvalitetskriteriet, idet TDI-værdien repræsenterer en daglig dosis for en befolkningsvægtet gennemsnitlig livstidsrisikoforøgelse (en ekstrarisiko på 1 ud af 1 million udsatte over livstid), hvorfor en gennemsnits-/medianværdi for befolkningens udsættelse må være udgangspunktet.*

Luft, daglig standardeksponering

I forbindelse med valg af standardeksponering for luft tages der udgangspunkt i afsnit 6.1.1 i Miljøprojekt Nr. 974 (2005). I dette afsnit vurderes US EPA´s eksponeringsvurderinger at udgøre det bedste grundlag. I EU´s risikovurderingprogram for kemiske stoffer henvises ligeledes til de amerikanske værdier.

Uddrag af tabel 6.1.1

Alder	Legemsvægt (kg)	V_R – gennemsnit (m^3/dag)	V_R – inaktiv[a] (m^3/dag)	V_R - aktiv (m^3/dag)
Børn:				
Under 1 år	7.6	4.5	2.35	6.35
1 – 2 år	13	6.8	4.16	9.15

3 – 5 år	18	8.3	4.98	10.96
6 – 8 år	26	10	5.95	13.09

V_R = Respirations Volumen som henholdsvis gennemsnit, inaktivitet og aktivitet.

Værdierne er alders- og kønsopdelte, men angivelserne omfatter ikke spredningen (fordelingen) i de enkelte aldersgrupper. Det er således ikke ud fra de forliggende data muligt at aflæse en 90- eller 95 percentilværdi for eksponeringen af de forskellige aldersgrupper.

Som ovenfor nævnt vil der ved fastsættelsen af luftkvalitetskriterier blive taget hensyn til eksponeringen af børn.

Fra tabellen ses, at børn under 1 år i gennemsnit over et døgn indånder 4,5 m^3 svarende til 0,59 m^3/kg lgv/dag, mens børn i aldersgruppen 1-2 år og 3-5 år indånder henholdsvis 6,8 m^3 og 8,3 m^3 (svarende til henholdsvis 0,52 og 0,46 m^3/kg lgv/dag). Især de 1-5 årige må anses at være udeaktive.

På denne baggrund vil der ved beregning af luftkvalitetskriterier fremover blive anvendt en standardværdi for 1-5 årige børn på 0,5 m^3 luft/ kg lgv.

Til sammenligning kan nævnes at luftkvalitetskriteriet hidtil er blevet beregnet ud fra et dagligt indåndingsvolumen på 0,3 m^3/ kg lgv., idet udgangspunktet her var en voksen person (70 kg), der dagligt indåndede 20 m^3 luft.

Jord, daglig standardeksponering

Miljøprojekt Nr. 974 (2005) sammenfatter i afsnit 6.1.2 den seneste viden med hensyn til børns udsættelse for jord samt anfører forskellige organisationers vurdering.

Ud fra en sammenfattende vurdering kan følgende eksponeringsværdier for børn opstilles:

Eksponeringsvej	Eksponeringsværdi
Oralt, maksimum enkeltindtag	10 g

Oralt, dagligt gennemsnit	0,1 g/d
Oralt, 95-percentil	0,2 g/d
Hudkontakt, dagligt gennemsnit	1 g/d
Hudkontakt, maksimum	10 g/d

For børns indtag gennem munden fastsættes jordkvalitetskriteriet for akut toksiske stoffer ud fra et enkeltindtag på 10 g jord.

I tilfælde, hvor hele TDI eller hovedparten af TDI anvendes til beregning af jordkvalitetskriteriet, tages der specifikt hensyn til særligt udsatte børn, idet der ved beregning af jordkvalitetskriteriet tages udgangspunkt i 95-percentilen for udsættelse dvs. 0,2 g jord/d for herved at minimere risikoen for at overskride TDI-værdien.

I andre tilfælde, hvor der kun anvendes en mindre del af TDI-værdien til jordkvalitetskriteriet, anvendes ved beregningen af jordkvalitetskriteriet 0,1 g jord/d som et gennemsitligt standardindtag.

For kræftfremkaldende stoffer, hvor TDI svarer til en gennemsnitlig 10^{-6} livstidsrisikodosis blandt børn, anvendes 0,1 g jord/dag som udgangspunkt for beregning af jordkvalitetskriteriet.

Drikkevand, daglig standardeksponering

I Miljøprojekt Nr. 974 (2005) i afsnit 6.1.3 gennemgås viden om forskellige aldersgruppers indtagelse af drikkevand, idet indtagelse både opgives for forskellige aldersgrupper og fordelingen inden for disse (gennemsnitsværdier og 90-/95 percentiler). Beskrivelse i baggrundsrapporten omfatter vurdering og anbefalingerne fra US EPA´s Exposure Factors Handbook.

Baggrundsrapporten henviser i forbindelse med børns indtag af drikkevand til tabel 6.1.3 D, der angiver US EPA´s anbefalede værdier:

Alder	Gennemsnit (mean)	50 Percentil	90 Percentil	95 percentil

Under 1 år	0,30 liter/dag 44 ml/kg/dag	0,24 liter/dag 35 ml/kg/dag	0,65 liter/dag 102 ml/kg/dag	0,76 liter/dag 127 ml/kg/dag
Under 3 år	0,61 liter/dag		1,5 liter/dag	
3 – 5 år	0,87 liter/dag		1,5 liter/dag	
1 – 10 år	0,74 liter/dag 35 ml/kg/dag	0,66 liter/dag 31 ml/kg/dag	1,3 liter/dag 64 ml/kg/dag	1,5 liter/dag 79,4 ml/kg/dag

For spædbørn angives medianindtaget at være 35 ml/ kg lgv/d mens 95-percentilen angives at være på 127 ml/ kg lgv/d. For børn i aldersgruppen 1-10 år anføres tilsvarende en medianværdi på 31 ml/kg lgv./d og en 95-percentil på 79,4 ml/kg lgv./d.

Som standardværdi vurderes det mest relevant at anvende værdien for 1-10 årige børn, idet evt. forskelle i vaner m.h.t. amning i USA og Danmark kan have stor indflydelse på drikkevandsindtagelse for spædbørn. Endelig dækker TDI-begrebet alene moderens direkte eksponering og derved barnets indirekte udsættelse gennem modermælken, og TDI er således ikke formelt set beregnet til at dække spædbarnets direkte udsættelse. Gennemsnitligt er indtagene dog direkte sammenlignelige.

Ved beregning af drikkevandskvalitetskriteriet, hvor der ved en given forureningskomponent er foretaget en allokering på 100 % eller hovedparten af TDI til drikkevandet, eller i situationer hvor den kritiske effekt er en akut toksisk effekt, anvendes 95-percentilværdien for 1-10 årige børns indtag, dvs. et dagligt indtag på 0,08 liter/ kg lgv/d. I disse situationer, vil drikkevandskvalitetskriteriet således også omfatte spædbørns direkte udsættelse, idet dette også afspejler indtaget for børn under 1 år, som har en høj direkte udsættelse.

Ved beregning i andre situationer, hvor der er foretaget en allokering på en mindre del af TDI til drikkevandskvalitetskriteriet (eller der er anvendt en reduktionsfaktor) anvendes medianværdien for 1-10-åriges forbrug af drikkevand sv.t. 0,03 liter/ kg lgv/d. Allokeringen vil også betyde at spædbørns direkte udsættelse er omfattet af kvalitetskriteriet.

For kræftfremkaldende stoffer, hvor TDI svarer til en gennemsnitlig 10^{-6} livstidsrisikodosis blandt børn, anvendes

0,03 liter/ kg lgv/d som udgangspunkt for beregning af drikkevandskvalitetskriteriet.

Bilag 3

Anvendte forkortelser

BMD – BenchMark Dosis
D – dag
ED – Eksponerings Dosis
EEC – European Economic Community
EU – Europæiske Union
GLP – Good Laboratory Practice
IARC – International Agency for Research on Cancer
IPCS – International Programme on Chemical Safety
KK – kvalitetskriterie
LED – Linear Ekstrapolations Dosis
Lgv – legemsvægt
LMS – Linear Multistage Model
LOAEL – Lowest Observed Adverse Effect Level
LOEL – Lowest Observed Effect Level
NOAEL – No Observed Adverse Effect Level
NOEL – No Observed Effect Level
OECD – Organisation for Economic Cooperation and Development
PAH – Poly Aromatiske Hydrocarbon
QSAR – Quantitative Structure Activity Relationships
TDI – Tolerabel Daglig Indtag (Tolerabel Daglig Eksponering, el. Tolerabel Daglig Dosis)
TK – Tolerabel Koncentration
UF – Usikkerheds Faktor
US EPA – United States Environmental Protection Agency
V_R – indåndingsvolumen
WHO – World Health Organisation
W – Kropsvægt

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Late lessons from early warnings: science, precaution, innovation

Summary

ISSN 1725-9177

European Environment Agency

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Preface

An investment in knowledge pays the best interest
— Benjamin Franklin '*The Way to Wealth*' (1758).

There is something profoundly wrong with the way we are living today. There are corrosive pathologies of inequality all around us — be they access to a safe environment, healthcare, education or clean water. These are reinforced by short-term political actions and a socially divisive language based on the adulation of wealth. A progressive response will require not only greater knowledge about the state of the planet and its resources, but also an awareness that many aspects will remain unknown. We will need a more ethical form of public decision-making based on a language in which our moral instincts and concerns can be better expressed. These are the overall aims of Volume 2 of *Late lessons from early warnings*.

Volume 1 of *Late lessons from early warnings* was published at a time when the world was experiencing an economic slowdown, China had joined the World Trade Organization and western Europe was still a 15-member Union. Global grain production had declined for the third time in four years due mainly to droughts in North America and Australia, and the world saw major recalls of contaminated meat, foot and mouth disease and bovine spongiform encephalopathy (mad cow disease). Global temperatures continued to climb and many bird populations were in decline, but the United States of America had rejected the Kyoto Protocol. We were seeing ourselves through the lens of the first human genome sequence, yet we were trying to manage chemicals known to be harmful to humans and ecosystems, through international conventions and treaties such as the Basel Convention to deal with toxic waste dumping in the developing world; the OSPAR/HELCOM Conventions to reduce the discharges, emissions and the loss of hazardous substances into the sea and the Montreal Protocol, to phase out ozone-depleting substances. The destruction of the World Trade Center had just happened.

Since then, we have witnessed a period of extraordinary hubris. Most visibly, the financial profligacy of the first decade of the century led inexorably to the crises of 2007–2009 whereby the major components of the international financial system were weakened to the extreme by indebtedness, mispriced products, lax monetary policies and mis-engineered protection against risks and uncertainty. The world experienced more not less volatility. Political systems became silted up by vested interests and a determination by citizens to protect assets accumulated in easier times, and beneath it all lay a deeper environmental crisis epitomised by climate change and biodiversity loss.

There was also a collapse of trust, not only in financial institutions but in big companies, as they abandoned staff, pensions and health care schemes. Recent evidence from social psychology has shown that despite rising levels of education and innovation in products and services, people trust only those they know and not strangers. As Stephen Green said in *Good value: reflections on money, morality, and an uncertain world* in 2009:

> 'There has been a massive breakdown of trust: trust in the financial system, trust in bankers, trust in business and business leaders, trust in politicians, trust in the media, trust in the whole process of globalisation — all have been severely damaged, in rich countries and poor countries alike'.

The scientific elites have also been slowly losing public support. This is in part because of the growing number of instances of misplaced certainty about the absence of harm, which has delayed preventive actions to reduce risks to human health, despite evidence to the contrary.

Suddenly, our problems have grown into what Charles W. Churchman in 1967 termed *wicked problems* — difficult or impossible to solve because of incomplete, contradictory and changing requirements, difficult to recognize, resistant to resolution because of the complexity of their interdependencies and needing to be tackled not by one but via many forms of social power. Solving

them requires a new combination of hierarchical power, solidarity and individualism.

What could this mean, for example, for the 100 thousand chemicals currently in commercial use?

To begin with we have more conventions and treaties in place than a decade ago: the 2004 Rotterdam Convention on the Prior Informed Consent (PIC) Procedure covering international trade of 24 pesticides, four severely hazardous pesticide formulations and 11 industrial chemicals; the 2004 Stockholm Convention on Persistent Organic Pollutants to protect human health and the environment from substances which are highly toxic, persistent, bio-accumulative and move long distances in the environment, such as DDT, PCBs, various industrial chemicals, and a set of unintentional chemical by-products such as dioxin. But these conventions only address the top-down hierarchical approach to power.

At the same time Europe has put in place legislation to achieve a global regulatory influence including the EU Cosmetic Directive banning the use of chemicals known or strongly suspected of being carcinogens, reproductive toxins, or mutagens causing cancer, mutation or birth defects; the EU Restriction of Hazardous Substances Directive, which restricts the use of hazardous materials in the manufacture of various types of electronic and electrical equipment including lead, mercury, cadmium, hexavalent chromium, the flame retardents polybrominated biphenyls and polybrominated diphenyl ethers, and which encourages the substitution to safe/or safer alternatives in the electric and electronic equipment industry; the closely linked 2006 EU Waste Electrical and Electronic Equipment Directive for collection, recycling and recovery of electrical goods; the 2006 Strategic Approach to International Chemicals Management (SAICM); and the 2007 EU Registration, Evaluation and Authorisation of Chemicals, widely known as REACH, to assign greater responsibility to industry to manage the risks from chemicals and to provide safety information on substances. The effects of these regulatory tools are described in different chapters, but once again point to the main economic actors rather than communities or individuals.

One thing that has become clearer over the past decade is that certain chemical substances are highly stable in nature and can have long-lasting and wide ranging effects before being broken down into a harmless form. The risk of a stable compound is that it can be bio-accumulated in fatty tissues at concentrations many times higher than in the surrounding environment. Predators, such as polar bears, fish and seals, are known to bio-magnify certain chemicals in even higher concentrations with devastating consequences for both humans and ecosystems. So exposure to toxic chemicals and certain foodstuffs are at risk of causing harm, especially to vulnerable groups such as foetuses in the womb or during childhood when the endocrine system is being actively built. Even with small dose exposures, the consequences can in some instances be devastating with problems ranging from cancer, serious impacts on human development, chronic diseases and learning disabilities. Here the power to act could be more properly set by well-informed individuals and communities.

The relationship between knowledge and power lies at the heart of Volume 2. In many chapters, the implicit links between the sources of scientific knowledge about pollutants, changes in the environment and new technologies, and strong vested interests, both economic and paradigmatic, are exposed. A number of authors also explore in greater depth, the short-sightedness of regulatory science and its role in the identification, evaluation and governance of natural resources, physical and chemical hazards. By creating a better understanding of these normally invisible aspects, it is hoped that this volume will enable communities and people to become more effective stakeholders and participants in the governance of innovation and economic activities in relation to the associated risks to humans and the planet.

Much of what we are able to learn from the histories of past environmental and public health mistakes is also directly applicable to the better regulation and governance of global institutions and financial and economic risks. Robin G. Collingwood argued in his *Autobiography* (1939), that:

> 'History can offer something altogether different from [scientific] rules, namely insight. The true function of insight is to inform people about the present…we study history in order to see more clearly into the situation in which we are called upon to act… the plane on which, ultimately, all problems arise is the plane of 'real' life: that to which they are referred for their solution is history.'

In this volume, we go further. Whilst still drawing lessons from such widely accepted tragedies as leaded petrol, mercury poisoning in Japan's

Minamata Bay and older pesticides which sterilised many men who used it, we have ventured into the uncertainties of potential yet contested harm, from genetically modified products; nanotechnologies; chemicals such as Bisphenol A; new pesticides and mobile phones. There is also an examination of the 80 or so potential 'false positives' where there had been indications of harm but where it was subsequently claimed that there were in fact no risks to prevent: these cases too can provide information that can help to improve future decision-making about innovation and emerging technologies.

A major part of effective decision-making lies in the way issues are framed. In the case of climate change, the first order question is whether it is worth worrying about at all. US Vice President Al Gore chose to make the question a matter of choice between believers and sceptics. However, problems arose when the public was asked to make a scientific decision when too few people had the qualifications to make any kind of reasoned judgement. They were in fact asked to make a false choice. Instead the question should have been framed around which areas should people and governments make decisions and which should be delegated to experts.

In the end there are few certain and enduring truths in the ecological and biological sciences, nor in the economics, psychologies, sociologies and politics that we use to govern them. One, however, comes from the work of Elinor Ostrom, a late and widely missed colleague, who showed from her work on managing fisheries and ecosystems that complex problems can be solved if communication is transparent and open, visions are shared, trust is high and communities are activated to work from the bottom-up as well as from the top down.

As we navigate the Anthropocene, the epoch named in recognition of our impact on the planet, we will need to encourage more people to become involved in solving the wicked problems of our times. Whether through gathering local information or becoming more aware of the many uncertainties and unpredictabilities in our surroundings, the power structures of knowledge will need to change. And if we are to respond more responsibly to the early warning signals of change, we will need to re-design our style of governance to one which reflects a future defined by the local and specific rather than only the global and the average. We hope that Volume 2 of *Late lessons from early warnings* with its many lessons and insights can help us all meet such a challenge.

Professor Jacqueline McGlade,
Executive Director

1 Introduction

Why further late lessons from early warnings?

The 2013 *Late lessons from early warnings* report is the second of its type produced by the European Environment Agency (EEA) in collaboration with a broad range of external authors and peer reviewers.

Volume 1 of *Late lessons from early warnings: the precautionary principle 1896–2000* published in 2001, looked at the history of a selection of occupational, public health and environmental hazards and asked whether we could have been better at taking action early enough to prevent harm. Twelve key lessons for better decision-making were drawn from cases where public policy was formulated against a background of scientific uncertainty and 'surprises' — and where clear evidence of hazards to people and the environment was often ignored (see box on page 11).

The 14 case studies and 12 key lessons from the 2001 report remain highly pertinent today, and underline four main reasons for a second report. The first relates to expanding the late lessons approach to consider long-known, important additional issues with broad societal implications such as lead in petrol, mercury, environmental tobacco smoke and DDT, as well as issues from which lessons have emerged more recently such as the effects of the contraceptive pill on feminisation of fish and the impacts of insecticides on honeybees.

The second concerns filling an acknowledged gap in the 2001 report, by analysing the issue of false positives where government regulation was undertaken based on precaution but later turned out to be unnecessary. Most of the cases examined in the *Late lessons from early warnings* reports are 'false negatives' — instances where early warnings existed but no preventive actions were taken.

The third reason is to address the rapid emergence of new society-wide challenges such as radiation from mobile phones, genetically-modified products, nanotechnologies and invasive alien species as well as if, how and where precautionary actions can play a role.

The final reason relates to how precautionary approaches can help manage the fast-changing, multiple, systemic challenges the world faces today, what new insights can be drawn in this context and how these can underpin opportunities for sustainable innovations and, supported by information technologies, greater public participation in their selection.

Overall approach

As for Volume 1, the approach in Volume 2 has been to include a wide range of relevant case studies produced by external authors along with chapters written by members of the report's editorial team (see acknowledgements section for details). The relevant topics for case study treatment were selected on the basis of advice from the editor, in collaboration with the editorial team and an advisory board, members of the EEA Scientific Committee and the Collegium Ramazzini ([1]).

The chapters in Volume 2 are grouped into five parts: A. Lessons from health hazards; B. Emerging lessons from ecosystems; C. Emerging issues; D. Costs, justice and innovation; and E. Implications for science and governance.

The chapters have been written by authors who, to varying degrees, have had substantial involvement in the subject area being addressed. Indeed they would not have been approached if

([1]) The Collegium Ramazzini is an independent, international academy founded in 1982 by Irving J. Selikoff, Cesare Maltoni and other eminent scientists. Its mission is to advance the study of occupational and environmental health issues and to be a bridge between the world of scientific discovery and the social and political centers, which must act on the discoveries of science to protect public health.

they had not already extensively studied the case that they were asked to write about. All of them, as respected experts in their fields and in line with their professional scientific training, were expected to be as objective as possible in answering the questions put to them by EEA. To support this, and to develop consistency between chapters, the authors were provided with seven structuring questions to be followed when building their chapter.

The case studies have been peer-reviewed by recognised experts in the respective fields who gave of their time freely and provided their feedback within a set of editorial guidelines provided by the EEA.

Scope

The report has been designed, structured and written in order to, inter alia, help politicians, policymakers and the public to:

i understand better the ways in which **scientific knowledge** is financed, created, evaluated, ignored, used and misused in taking timely and precautionary decisions about how to reduce harms, whilst stimulating benign innovations and generating useful employment;

ii learn from some **very expensive 'mistakes' in the past** so as to help societies make fewer mistakes now, and in the future, especially with some of the relatively new, largely unknown, yet already widespread technologies like nanotechnology and mobile phones;

iii be aware of less visible, important factors such as the skewed ways in which the **costs of actions and inactions** for hazardous technologies have been estimated, and the role that **some businesses** have played in ignoring early warnings and in manufacturing doubt about the science supporting such warnings;

iv consider how the law, or administrative arrangements, could be better used to deliver **justice, to those people (and ecosystems) that have been, or could be, harmed** by poorly designed, or badly deployed, innovations;

v explore how best to **engage the public** in helping to make **strategic choices over innovations**, and their technological and social pathways, as well as their involvement in **ecosystems management** and in long term monitoring through **citizen science**.

Part A of the report commences with an analysis of 'false positives' showing that these are few and far between as compared to false negatives and that carefully designed precautionary actions can stimulate innovation, even if the risk turns out not to be real or as serious as initially feared. The remaining nine chapters address false negatives — lead in petrol, perchlorethylene contaminated water, Minamata disease, occupational beryllium disease, environmental tobacco smoke, vinyl chloride, dibromochloropropane (DBCP), Bisphenol A and dichlorodiphenyltrichlorethane (DDT) — from which three common themes emerge: there was more than sufficient evidence for much earlier action; slow and sometimes obstructive behaviour by businesses whose products endangered workers, the public and the environment; and the value of independent scientific research and risk assessments.

Part B focuses on emerging lessons from the degradation of natural systems and their wider implications for society — booster biocides, the pill and the feminisation of fish, climate change, floods, insecticides and honeybees as well as ecosystem resilience more broadly. It considers, like its predecessor, the issues of scientific evidence as the basis for action/inaction, the multiple, often complex factors and feedback loops in play, many of which are not fully understood, as well as the interfaces between science, policy and society and how all actors can move together towards necessary actions in the context of heightened systemic risks, and substantial unknowns.

Part C analyses some newly emerging and large-scale products, technologies and trends, which potentially offer many benefits but also potentially much harm to people and ecosystems and thereby ultimately economic development. Cases addressed include the Chernobyl and Fukushima nuclear accidents; genetically modified agricultural crops and agroecology; the growing threat of invasive alien species; mobile phones and the risk of brain tumours; and nanotechnologies. There is often little science, and very little direct hindsight, to assist in the management of these emerging technologies but the lessons from the historical case studies need to be applied if hazards are to be avoided.

The evidence from the chapters in Part C is that, by and large, societies are not making the most use of the costly lessons that can be gleaned from their histories. A key question is how this can be improved given the many reasons identified from the case studies why taking actions have been delayed including: the novel

and challenging nature of the issues themselves; poorly or inconsistently evaluated information; strong opposition by the corporate and scientific establishments of the day; and the tendency by the decision making institutions, practices and cultures to favour the status quo and the short term perspective. This section also illustrates the value of bottom up as well as top down approaches to innovations in ensuring that the directions of technological pathways, the equitable distributions of benefits, costs and knowledge ownership, and the diversity of locally sensitive technological options are relevant to the food, energy and ecosystems crises.

The historical chapters illustrate numerous harms which for the most part have been caused by irresponsible corporations. This fact, coupled with shortcomings in how decisions are made by governments on when to act on early warnings, and in the law when it comes to compensating victims of harm, are analysed in three chapters in Part D of the report. Each chapter analyses the reasons behind prevailing practice and then goes on to offer insights, for example, on how cost calculation methods can be improved; on how insurance schemes could be used to compensate future victims of harm; and on the reasons why businesses frequently ignore early warnings.

The cases in Parts A–D form the basis for considering in Part E the governance implications for science, public policy and public engagement, and how current practices could be improved to enable society to maximise the benefits of innovations while minimising harms. The main insights are that science could be more relevant for precautionary decision-making; that the wider use of the precautionary principle can avert harm and stimulate innovation; and that the late lessons of history and precautionary approaches are highly pertinent to today's multiple and inter-connected crises — such as those arising from finance, economics, the use of ecosystems, climate change, and the use and supply of energy and food.

Finally, many of the historical and recent case studies illustrate the value of engaging the public in broadening the knowledge base and stimulating robust innovations.

Twelve late lessons

Based on the case studies of Volume 1 of *Late lessons from early warnings* (EEA, 2001), twelve key lessons for better decision-making were drawn:

1	Acknowledge and respond to ignorance, as well as uncertainty and risk, in technology appraisal and public policymaking
2	Provide adequate long-term environmental and health monitoring and research into early warnings
3	Identify and work to reduce 'blind spots' and gaps in scientific knowledge
4	Identify and reduce interdisciplinary obstacles to learning
5	Ensure that real world conditions are adequately accounted for in regulatory appraisal
6	Systematically scrutinise the claimed justifications and benefits alongside the potential risks
7	Evaluate a range of alternative options for meeting needs alongside the option under appraisal, and promote more robust, diverse and adaptable technologies so as to minimise the costs of surprises and maximise the benefits of innovation
8	Ensure use of 'lay' and local knowledge, as well as relevant specialist expertise in the appraisal
9	Take full account of the assumptions and values of different social groups
10	Maintain the regulatory independence of interested parties while retaining an inclusive approach to information and opinion gathering
11	Identify and reduce institutional obstacles to learning and action
12	Avoid 'paralysis by analysis' by acting to reduce potential harm when there are reasonable grounds for concern

Source: EEA, 2001, *Late lessons from early warnings: the precautionary principle 1986–2000,* Environmental issues report No 22, European Environment Agency.

2 The precautionary principle and false alarms — lessons learned

Steffen Foss Hansen and Joel A. Tickner

Most of the cases examined in the *Late lessons from early warnings* reports are 'false negatives' — instances where early warnings existed but no preventive actions were taken. In debates surrounding the precautionary principle it is often claimed that widespread application of the principle will lead to a large number of regulatory false positives — over-regulation of minor risks and regulation of non-existent risks, often due to unwarranted public 'fears'. Understanding and learning from past false positives as well as false negatives is essential for improving decision-making about public health and the environment.

This chapter reviews incidents of 'false positives', where government regulation was undertaken based on precaution but later turned out to be unnecessary. In total 88 cases were identified to be alleged false positives, however, following a detailed analysis most of them turned out to be either real risks, or cases where 'the jury is still out', or unregulated alarms, or risk-risk trade-offs, rather than false positives.

The analysis revealed four regulatory false positives: US swine flu, saccharin, food irradiation, and Southern leaf corn blight. Numerous important lessons can be learned from each, although there are few parallels between them in terms of when and why each risk was falsely believed to be real. This is a lesson in itself: each risk is unique, as is the science and politics behind it and hence a flexible approach is therefore needed, adapted to the nature of the problem. The costs of the false positives identified were mainly economic, although the actions taken to address swine flu in 1976 did lead to some unintended deaths and human suffering, and diverted resources from other potentially serious health risks. Determining the net costs of mistaken regulatory action, however, requires a complete assessment of the impacts of the regulation, including the costs and benefits of using alternative technologies and approaches.

Overall, the analysis shows that fear of false positives is misplaced and should not be a rationale for avoiding precautionary actions where warranted. False positives are few and far between as compared to false negatives and carefully designed precautionary actions can stimulate innovation, even if the risk turns out not to be real or as serious as initially feared. There is a need for new approaches to characterising and preventing complex risks that move debate from the 'problem' sphere to the 'solutions' sphere. By learning from the lessons in this chapter, more effective preventive decisions can be made in the future.

The scarcity of genuine false positives compared to the large number of 'mistaken false positives' could partly be the result of a deliberate strategy in risk communication. Several references and leaked documents have shown that some regulated parties have consciously recruited reputable scientists, media experts and politicians to call on if their products are linked to a possible hazard. Manufacturing doubt, disregarding scientific evidence of risks and claiming over-regulation appear to be a deliberate strategy for some industry groups and think tanks to undermine precautionary decision-making.

3 Lead in petrol 'makes the mind give way'

Herbert Needleman and David Gee

This chapter addresses the widespread use of lead in petrol. It focuses on the period 1925–2005, when leaded petrol was first widely marketed in the US and then spread to the rest of the world before being gradually phased out from the 1970s. In Europe, the Aarhus Protocol (www.unece.org/env/pp/treatytext.html) initiated the phase-out of leaded petrol in the period 1998–2005.

The neurotoxic effects of lead were recognised as far back as Roman times. And in 1925, at the 'one day trial' of leaded petrol in the US, many experts warned of the likely health impacts of adding lead to petrol. Yet, despite the availability of an equally effective alcohol additive which was assessed by experts to be cleaner, the leaded route to fuel efficiency was chosen in the US and then exported to the rest of the world.

For several decades after the introduction of leaded petrol, virtually no independent research was carried out and the main source of information was industry and industry-sponsored researchers. Not until the 1960s and 1970s did independent scientists from outside this group show, for example, that body burdens of lead arising from human activities were not 'normal', as industry claimed, but were hundreds of times higher than before the industrial revolution and were therefore likely to be harmful.

At its peak in the mid-1970s, leaded petrol released about 200 000 tonnes of lead into the atmosphere annually in both the US and Europe. Following the subsequent phase-out, blood lead levels in children (the most sensitive group exposed) quickly fell, in line with the decrease in air concentrations. The lessons nevertheless remain relevant globally today. Although nearly all countries worldwide had phased out leaded petrol by 2012, lead concentrations in soils and sediments remain high. Meanwhile, electronic wastes containing lead and other contaminants also cause elevated blood lead levels.

Supplementary panel texts focus on the events leading up to the US choice of leaded petrol as the primary fuel source in 1925 and more recent accounts of EU policymaking on lead in petrol and the road to phase-outs in Germany and the United Kingdom.

4 Too much to swallow: PCE contamination of mains water

David Ozonoff

PCE (perchlorethylene, also known as 'perc' or tetrachloroethylene), was used in the production of plastic linings for drinking water distribution pipes in the late 1960s and 1970s. This new and relatively untested type of distribution pipe was used in over 700 miles of New England's water distribution systems. Not until 1976 was it discovered that PCE had been leaching into the water from the pipe lining, causing widespread contamination of water supplies that still today require continuous remediation.

Before the pipes were put into production there was a substantial amount of scientific information available about the potential hazards of PCE. This did not include current concerns about PCE's carcinogenicity, teratogenicity and other health consequences of relatively low-level exposure upper most among today's concerns, but many early warnings suggested the need for caution in introducing PCE-based mains pipe linings.

PCE had been used to treat hookworm and data on side effects were in the literature, while later a variety of occupational users were studied, including aircraft workers, small companies in countries where biological monitoring was required, and dry-cleaning firms. Several environmental studies were also conducted to see if drinking water contaminated with PCE or its close relative, TCE (trichloroethylene), was associated with cancer. Results were mixed and the chemical industry consistently denied that PCE was a human carcinogen.

This case study explores the early (pre 1970) history researching the toxicity of the chemical. It also focuses on the failure of one manufacturer, Johns-Manville Corporation, to recognise the warning signals about using a suspected toxic substance. It examines why a new product was deployed without thought to the public health consequences and why evidence of the potential hazard was ignored.

The science has not been hidden. It has been ineffective in guiding and catalysing action. Whether the problem is a failed duty of care or a lack of clarity about what evidence will trigger action, the contemporary argument over how to interpret the scientific evidence is irresolvable within science itself. There are no overarching criteria from the philosophy of science that can dictate a solution.

This chapter also includes two supplementary texts. A panel that analyses the differences between the conclusions of risk assessments based on the same data, focusing in particular on assessments of PCE and TCE. A further panel describes the opportunities to switch to wet-cleaning technologies to reduce the current use of PCE in dry cleaning.

5 Minamata disease: a challenge for democracy and justice

Takashi Yorifuji, Toshihide Tsuda and Masazumi Harada

Minamata disease, which can induce lethal or severely debilitating mental and physical effects, was caused by methylmercury-contaminated effluent released into Minamata Bay by Chisso, Japan's largest chemical manufacturer. It resulted in widespread suffering among those who unknowingly ate the contaminated fish. This chapter documents the story in three phases.

The disease first came to prominence in the 1950s. It was officially identified in 1956 and attributed to factory effluent but the government took no action to stop contamination or prohibit fish consumption. Chisso knew it was discharging methylmercury and could have known that it was the likely active factor but it chose not to collaborate and actively hindered research. The government concurred, prioritising industrial growth over public health. In 1968 Chisso stopped using the process that caused methylmercury pollution and the Japanese government then conceded that methylmercury was the etiologic agent of Minamata disease.

The second part of the story addresses the discovery that methylmercury is transferred across the placenta to affect the development of unborn children, resulting in serious mental and physical problems in later life. Experts missed this at first because of a medical consensus that such transfer across the placenta was impossible.

The third phase focuses on the battle for compensation. Initially, Chisso gave token 'sympathy money' under very limited criteria. In 1971 the Japanese government adopted a more generous approach but after claims and costs soared a more restrictive definition was introduced in 1977, justified by controversial 'expert opinions'. Legal victories for the victims subsequently made the government's position untenable and a political solution was reached in 1995–1996. In 2003, the 'expert opinions' were shown to be flawed and the Supreme Court declared the definition invalid in 2004.

In September 2011 there were 2 273 officially recognised patients. Still, the continuing failure to investigate which areas and communities were affected means that the financial settlement's geographic and temporal scope is still not properly determined. Alongside deep-seated issues with respect to transparency in decision-making and information sharing, this indicates that Japan still faces a fundamental democratic deficit in its handling of manmade disasters.

This chapter is followed by three short updates on the effects of mercury poisoning since Minamata; on attempts to contain it, including the 2009 global agreement to phase mercury out of economic activity; and on the need for better information about contaminant exposures to enable policymakers to make informed choices that balance the benefits of fish consumption against the assumed adverse effects of low-level methylmercury exposures.

6 Beryllium's 'public relations problem'

David Michaels and Celeste Monforton

Scores of workers employed in nuclear weapons production have been diagnosed with chronic beryllium disease (CBD), a progressive and irreversible inflammatory lung disease. This chapter presents a history of knowledge and public policy about preventing beryllium-related disease, focusing primarily on the United States beryllium industry's role in shaping US regulatory policy.

Over several decades increasingly compelling evidence accumulated that CBD was associated with beryllium exposure at levels below the existing regulatory standard. The beryllium industry had a strong financial incentive to challenge the data and decided to be proactive in shaping interpretation of scientific literature on beryllium's health effects. It hired public relations and 'product defence' consulting firms to refute evidence that the standard was inadequate. When the scientific evidence became so great that it was no longer credible to deny that workers developed CBD at permitted exposure levels, the beryllium industry responded with a new rationale to delay promulgation of a new, more protective exposure limit.

This case study underscores the importance of considering the hazards from toxic materials throughout the entire product life cycle. While primary producers of beryllium products may be able to control exposures in their own facilities, it is unlikely that many secondary users and recyclers have the expertise, resources and knowledge necessary to prevent beryllium disease in exposed workers and residents in nearby communities.

The primary lessons of this chapter are widely applicable to many environmental health controversies. In particular, it illustrates the practice of 'manufacturing uncertainty' — a strategy used by some polluters and manufacturers of hazardous products to prevent or delay regulation or victim compensation.

This chapter is followed by an analysis of the rationale for corporate behaviour in the regulation of beryllium. It is argued that the availability of occasional and limited opportunities for companies to change course without suffering onerous consequences would encourage them to rethink their position and create an obligation on shareholders to take the responsible course. Although this may be perceived as letting them 'get away with it', the end result may be better public policy and corporate responsibility.

7 Tobacco industry manipulation of research

Lisa A. Bero

This chapter differs in some ways from the others in Volume 2 of *Late lessons from early warnings*. The history of 'second hand', 'passive' or 'environmental tobacco smoke' (ETS), to which non-smokers are exposed overlaps with the history of active smoking. Those affected include the partners and children of smokers, and the bartenders and other workers who have to work in smoky environments.

The focus in this chapter is on the strategies used by the tobacco industry to deny, downplay, distort and dismiss the growing evidence that, like active smoking, ETS causes lung cancer and other effects in non-smokers. It does not address the history of scientific knowledge about tobacco and how it was used or not used to reduce lung cancer and other harmful effects of tobacco smoke. There is much literature on this and a table at the end of the chapter summarises the main dates in the evolution of knowledge in this area.

The chapter concentrates on the 'argumentation' that was used to accept, or reject, the growing scientific evidence of harm. Who generated and financed the science used to refute data on adverse health effects? What were the motivations? What kind of science and information, tools and assumptions were used to refute data on the adverse health of tobacco?

The release of millions of internal tobacco industry documents due to law suits in the US has given insights into the inner workings of the tobacco industry and revealed their previously hidden involvement in manipulating research. However, this insight is not available for most corporate sectors. The chapter discusses the possibilities of 'full disclosure' of funding sources and special interests in research and risk assessment in order to secure independence and prevent bias towards particular viewpoints.

While smoking bans are now being introduced in more and more countries, other industries are drawing inspiration from tobacco company strategies, seeking to maintain doubt about harm in order to keep hazardous products in the marketplace.

The chapter also includes a summary of the tobacco industry's role in shaping risk assessment in the US and Europe to serve its own interests.

8 Vinyl chloride: a saga of secrecy

Morando Soffritti, Jennifer Beth Sass, Barry Castleman and David Gee

This chapter is about how early warnings in the 1950s and 1960s concerning the short-term harm of vinyl chloride (VC) to the skin and bones of workers, and to the livers of laboratory animals, were initially hidden from other workers and regulators. This was despite some early misgivings by company experts whose advice was initially ignored by their employers. This pattern was repeated when the later, more devastating news of a rare liver cancer in workers was revealed by long-term animal studies and by an attentive and concerned company physician.

Unlike many other histories, however, this story features a very prompt response from the global chemical industry to the publication of the liver cancer evidence, a response that included funding cancer testing and later compliance with a large reduction in the permissible exposure limits. The case also provides early evidence of reproductive effects of vinyl chloride monomer (VCM).

Other features of this story presage the later and common responses of the corporate world to heightened public awareness and pressure from non-governmental organisations (NGOs) and trade unions, including greatly exaggerated estimates of the likely costs of complying with tighter pollution controls; a frequent mismatch between the position of the trade association and that of many, more progressive companies within the association; but also some relatively quick corporate responses to public, NGO and regulatory pressure.

The chapter also features two legal aspects, which, though more common in the US, are also valuable for Europeans. First, the potentially positive role that judicial review of regulatory proposals can play in providing a societal judgement about the behaviour of corporations. This can embrace not just moral judgements but also judgements about the state of the science and what society should do with it.

Second, the role that document discovery in legal compensation cases can play in revealing the real and until then secret activities of corporations. Any proposals to promote justice for victims of environmental and health harms via no fault administrative arrangements need to be accompanied by other measures to extract information about corporate behaviour.

The chapter is followed by a panel analysing the value of animal testing for identifying carcinogens.

9 The pesticide DBCP and male infertility

Eula Bingham and Celeste Monforton

Dibromochloropropane (DBCP) is a pesticide used against nematodes (roundworms or threadworms) that damage pineapples, bananas and other tropical fruits. It was introduced into US agriculture in 1955 and approved for use as a fumigant in 1964. By 1961 laboratory experiments had shown that it made the testicles of rodents shrink and significantly reduced the quantity and quality of sperm. Nonetheless, the compound was widely marketed and became a commercial success.

In 1977, workers at a production plant became worried that they were unable to father children. An emergency study by a US government agency discovered that in many cases the workers were suffering from deficient or absent sperm. While controls were improved at US facilities, the product continued to be marketed and sprayed in Latin America, the Philippines, some African countries, and elsewhere.

By the 1990s, tens of thousands of plantation workers in these countries had allegedly suffered adverse reproductive effects from DBCP use.

The story continues today with contentious legal claims for compensation, contamination of drinking water and industry attempts to prevent a Swedish documentary on the issue from being screened.

This chapter looks at the knowledge available about the hazards and the actions taken, or not taken, to avert them. The DBCP story is significant as it is the first clear example of reproductive damage to workers who manufactured and used a synthetic chemical. This is one of many examples supporting the growing concerns about increasing rates of reproductive and developmental disease, and about the endocrine disrupting chemicals that seem to be playing a role in these disorders.

Protecting production workers, users, consumers and the environment from chemicals that may damage reproduction demands closer integration of scientific disciplines, as well as government action. The lessons of DBCP may help in ensuring timely protection from harm, based on precautionary approaches to scientific evidence.

10 Bisphenol A: contested science, divergent safety evaluations

Andreas Gies and Ana M. Soto

Bisphenol A (BPA) is currently one of the world's best-selling chemicals and primarily used to make polycarbonate plastics. It is widely used in common products such as baby bottles, household electronics, medical devices and coatings on food containers. BPA is known to mimic the female hormone oestrogen and has been found to leach from the materials where it is used.

Studies have suggested that even exposure to low doses of BPA may cause endocrine disrupting effects. As with other hormones, it appears that an organism is most sensitive during development but that effects are often not observed until much later in the lifecycle. This means that at the time when the effects become detectable, the chemical exposure has vanished. This makes it extremely difficult to link exposure to effects in humans.

This chapter maps some of the findings in studies of rodents and humans. It also discusses the challenges of evaluating scientific findings in a field where industry-sponsored studies and independent scientific research seem to deviate strongly. The authors offer suggestions for ways to uncouple financial interests from scientific research and testing.

A widely used and dispersed industrial chemical like Bisphenol A is a controversial example of an endocrine disrupting substance that has implications for policymakers. Different approaches to risk assessment for BPA by US and European authorities are presented. It throws light on the ways in which similar evidence is evaluated differently in different risk assessments and presents challenges for applying the precautionary principle.

The intense discussion and scientific work on BPA have slowly contributed to a process of improving test strategies. While traditional toxicology has relied on a monotonic increasing dose-response relationship as evidence that the effect is caused by the test agent, studies on BPA and other endocrine disruptor chemicals (EDCs) have demonstrated the limitations of this approach and adjustments have been made in some cases.

It has also been widely accepted that effects cannot be predicted by simply thinking of BPA as a weak oestrogen and extrapolating from what is observed for more potent endogenous oestrogens. This lesson is particularly evident in the intense pharmaceutical interest in selective oestrogen response modifiers (SERMs).

The chapter is followed by a panel analysing the value of animal testing for identifying carcinogens.

11 DDT: fifty years since *Silent Spring*

Henk Bouwman, Riana Bornman, Henk van den Berg and Henrik Kylin

'There was a strange stillness. The birds for example — where had they gone? Many people spoke about them, puzzled and disturbed. The feeding stations in the backyards were deserted. The few birds seen anywhere were moribund: they trembled violently and could not fly. It was a spring without voices ... only silence lay over the fields and woods and marsh.'

The book *Silent Spring* by Rachel Carson is mainly about the impacts of chemicals (in particular dichlorodiphenyltrichlorethane also known as DDT) on the environment and human health. Indeed, the close association between humans and birds remains very apt. Representing the only two warm-blooded groups of life on Earth, mammals and birds share the same environments and threats.

Carson's claim that she lived in 'an era dominated by industry, in which the right to make a dollar at whatever cost is seldom challenged' still resonates strongly with the problems that societies face all over the world. One chapter heading, 'The obligation to endure', derived from the French biologist and philosopher Jean Rostand's famous observation that, 'the obligation to endure gives us the right to know'. United States President John F. Kennedy responded to the challenge posed by Carson by investigating DDT, leading to its complete ban in the US. The ban was followed by a range of institutions and regulations concerned with environmental issues in the US and elsewhere, driven by public demand for knowledge and protection.

DDT was the primary tool used in the first global malaria eradication programme during the 1950s and 1960s. The insecticide is sprayed on the inner walls and ceilings of houses. Malaria has been successfully eliminated from many regions but remains endemic in large parts of the world. DDT remains one of the 12 insecticides — and the only organochlorine compound — currently recommended by the World Health Organization (WHO), and under the Stockholm Convention on Persistent Organic Pollutants, countries may continue to use DDT. Global annual use of DDT for disease vector control is estimated at more than 5 000 tonnes.

It is clear that the social conscience awakened by Rachel Carson 50 years ago gave momentum to a groundswell of actions and interventions that are slowly but steadily making inroads at myriad levels. Chapter 17 of her book, 'The other road' reminds the reader of the opportunities that should have been seized much earlier. With more than 10 % of bird species worldwide now threatened in one way or another, it is clear that we missed early warnings or failed to act on them. Will we continue to miss signposts to 'other roads'? Are our obligations to endure met by our rights to know? As Carson said 50 years ago: 'The choice, after all, is ours to make.'

12 Booster biocide antifoulants: is history repeating itself?

Andrew R. G. Price and James W. Readman

Tributyltin (TBT) was widely used as an effective antifouling agent in paints for ships and boats until the European Community restricted its use in 1989 because of its proven harm to the environment and shellfisheries. Thereafter, booster biocides were introduced to enhance the performance of antifouling paints. They were believed to be less damaging to aquatic life than TBT. Subsequently, however, it has been established that booster biocides can also create significant environmental risks.

This chapter outlines the background to booster biocide use, the early warnings about their potential physiological and ecological impacts on non-target species, and the actions taken in response. The science that set some alarm bells ringing is described, along with lessons that could influence the future of an industry still searching for less environmentally invasive solutions.

Booster biocide antifouling agents threaten a variety of habitats — from coral reefs and seagrass beds to open moorings — within the EU and globally. Their primarily herbicidal properties mean that coral zooxanthellae, phytoplankton and periphyton are particularly vulnerable. Compared to TBT, an antifouling agent with a quite specific action, booster biocides have more broad-spectrum impacts. The wider ecological effect of shifting to booster biocides remain poorly understood but of considerable concern because they may affect the base of marine food chains.

From a toxicological viewpoint, booster biocides do not threaten to have endocrine disrupting properties similar to TBTs. At current environmental concentrations, however, some can damage primary producers and some are persistent. While legislation has been introduced to control their use, the rigour of regulations varies between countries. These geographical disparities need to be addressed, and future biocidal products and novel approaches to antifouling should be better appraised.

For policymakers, the challenge is to protect non-target biological communities from selective change resulting from booster biocide use. Persistence, bioaccumulative and toxic (PBT) criteria can be used to evaluate the relative potential impact from the available biocides, and consequently target appropriate legislation. Nevertheless, lateral thinking, aiming to identify novel materials and strategies to address antifouling, could pay dividends in the future.

13 Ethinyl oestradiol in the aquatic environment

Susan Jobling and Richard Owen

Many decades of research have shown that when released to the environment, a group of hormones known as oestrogens, both synthetic and naturally occurring, can have serious impacts on wildlife. This includes the development of intersex characteristics in male fish, which diminishes fertility and fecundity. Although often sublethal, such impacts may be permanent and irreversible.

This chapter describes the scientific evidence and regulatory debates concerning one of these oestrogens, ethinyloestradiol (EE2), an active ingredient in the birth control pill. First developed in 1938, it is released to the aquatic environment via wastewater treatment plants. Although it is now clear that wildlife species are exposed to and impacted by a cocktail of endocrine disrupting chemicals, there is also reasonable scientific certainty that EE2 plays a significant role, and at vanishingly low levels in the environment.

In 2004 the Environment Agency of England and Wales accepted this, judging the evidence sufficient to warrant consideration of risk management. In 2012, nearly 75 years after its synthesis, the European Commission proposed to regulate EE2 as a EU-wide 'priority substance' under the Water Framework Directive (the primary legislation for protecting and conserving European water bodies). This proposal was subsequently amended, delaying any decision on a regulatory 'environmental quality standard' until at least 2016.

This is in part because control of EE2 will come at a significant price. Complying with proposed regulatory limits in the environment means removing very low (part per trillion) levels of EE2 from wastewater effluents at considerable expense.

Is this a price we are willing to pay? Or will the price of precautionary action be simply too high — a pill too bitter to swallow? To what extent is society, which has enjoyed decades of flexible fertility and will also ultimately pay for the control and management of its unintended consequences, involved in this decision? And what could this mean for the many thousands of other pharmaceuticals that ubiquitously infiltrate our environment and which could have sublethal effects on aquatic animals at similarly low levels?

14 Climate change: science and the precautionary principle

Hartmut Grassl and Bert Metz

The first scientifically credible early warning about the possible dangers of climate change due to carbon dioxide (CO_2) emissions from burning fossil fuels came in 1897. While the basic physical principles of global warming are simple, however, the more detailed science of climate change is exceedingly complicated. Even now, more than a hundred years since the first early warning, many important details of climate change cannot be predicted with certainty. It is therefore unsurprising that the science of climate change and questions about the true value of burning fossil fuels have fostered sustained scientific and political controversy.

When the first volume of *Late lessons from early warnings* was drafted there appeared to be too much legitimate controversy about climate change for the issue to be included. A case study could have led to arguments that distracted attention from the valuable and robust lessons from more established issues such as asbestos, polychlorinated biphenyls (PCBs), chlorofluorocarbons (CFCs) and the ozone-hole, X-rays and acid rain. This decision was taken despite the then widespread acceptance that 'the balance of evidence suggests a discernible human influence on global climate' (*Contribution of Working Group I to the Second Assessment Report of the Intergovernmental Panel on Climate Change*, IPCC, 1995).

Over a decade later and after two more reviews by the Intergovernmental Panel on Climate Change (IPCC) of a much greater volume of climate change science it seemed appropriate to include climate change in this volume, despite some continuing controversy. The evidence that human activities are having a dangerous impact on the climate has strengthened since 1995. By 2007, the IPCC was able to conclude with 'very high confidence that the global net effect of human activities since 1750 has been one of warming'. Given the size and irreversibility (on human time scales) of many of the harmful effects of human-induced climate change, there is an urgent need for action to reduce CO_2 emissions and other greenhouse gases. Some contrarian views persist, however, as the authors illustrate.

This chapter summarises the history of growing knowledge about human-induced climate change and of the main actions, or inactions that accompanied it. Like many other chapters, it reflects the lifelong commitment of both authors to trying to understand and mitigate the effects of human-induced climate change. It concludes with some lessons and insights that are relevant to many other environmental and health issues.

Also included is a panel text describing how the IPCC's approach to assessing uncertainty evolved between its first to its fifth assessment reports.

15 Floods: lessons about early warning systems

Zbigniew W. Kundzewicz

Floods are an increasingly acute problem. Intense precipitation has become more frequent and more intense, growing manmade pressure has increased the magnitude of floods that result from any level of precipitation, and flawed decisions about the location of human infrastructure have increased the flood loss potential.

Unlike most other case studies presented in this report, this chapter focuses on flooding as a phenomenon and the requirements for effective early warning systems, rather than addressing a particular event and the lessons that can be learned.

Flooding cannot be wholly prevented. The occurrence of a flood need not be considered a 'failure' and, conversely, minimisation of losses may constitute a 'success'. There are lessons to be learned from every flood and it is important to use them in preparing for the next flood. Once we accept that no flood protection measures can guarantee complete safety, a general change of paradigm is needed to reduce human vulnerability to floods. The attitude of 'living with floods' and accommodating them in planning seems more sustainable than hopelessly striving to eradicate them.

Flood forecasting and warning systems fail because links in the chain perform poorly or fail completely. A single weak point in a system that otherwise contains excellent components may render the overall system performance unsatisfactory. A successful system requires sufficient integration of components and collaboration and coordination between multiple institutions.

The chapter deals primarily with the challenges of fluvial (river) floods. It is complemented by three short supplementary texts. The first highlights the complex, dynamic and diverse ecosystems of river floodplains, which are often degraded during construction of flood defences. Despite their huge economic value, near-natural floodplains are among the most threatened ecosystems globally.

The second discusses uncertainties in anticipating rainfall patterns and intensity, and their relationship to flood levels during extreme flows. Such uncertainties present challenges for scientists and decision-makers alike.

The third addresses the increasing risks of coastal flooding due to factors such as climate change and sea-level rise, and reviews European experience with precautionary action.

16 Seed-dressing systemic insecticides and honeybees

Laura Maxim and Jeroen van der Sluijs

In 1994 French beekeepers began to report alarming signs. During summer, many honeybees did not return to the hives. Honeybees gathered close together in small groups on the ground or hovered, disoriented, in front of the hive and displayed abnormal foraging behaviour. These signs were accompanied by winter losses.

Evidence pointed to Bayer's seed-dressing systemic insecticide Gaucho®, which contains the active substance imidacloprid. This chapter presents the historical evolution of evidence on the risks of Gaucho® to honeybees in sunflower and maize seed-dressing in France, and analyses the actions in response to the accumulating evidence regarding these risks.

The social processes that ultimately lead to application of the precautionary principle for the ban of Gaucho® in sunflower and maize seed-dressing are described, with a focus on the ways in which scientific findings were used by stakeholders and decision-makers to influence policy during the controversy.

Public scientists were in a difficult position in this case. The results of their work were central to a social debate with high economic and political stakes. In certain cases their work was not judged according to its scientific merit but based on whether or not it supported the positions of some stakeholders. This situation tested the ability and courage of researchers to withstand pressure and continue working on imidacloprid.

Other European countries also suspended neonicotinoid seed-dressing insecticides. Evidence of the toxicity of neonicotinoids present in the dust emitted during sowing of coated seeds supported such decisions. Most important, the French case highlighted the major weaknesses of regulatory risk assessment and marketing authorisation of pesticides, and particularly neonicotinoids. These insights were recently confirmed by work by the European Food Safety Authority.

From this case study eight lessons are drawn about governance of controversies related to chemical risks. The study is followed by two additional texts. A first panel presents Bayer Crop Science's comments on the analysis in this chapter. A second contains the authors' response to the Bayer comments.

17 Ecosystems and managing the dynamics of change

Jacqueline McGlade and Sybille van den Hove

A decade after Rachel Carson's *Silent Spring* was published, describing the toxic legacy of the twentieth century, Annie Dillard in her Pulitzer prize winning book *Pilgrim at Tinker Creek*, opened up a different way of looking at the world. It presaged a twenty first century in which the global economy would be based on a more thorough understanding of nature, its functioning and material wealth. Wholly descriptive, yet increasingly relevant, her book captured the very essence of what this chapter is about: that amongst the observations which routinely help to predict the evolution of the natural world are the seeds of surprise — surprise of the unusual and surprise as a portent of future change. Our systemic failure to anticipate such surprises forms the core of this chapter. A series of case studies from fisheries, forests, savannah and aquatic systems are used to underline how early warnings about changes in these natural systems emerged but were not used.

The chapter highlights how the division of knowledge into political, disciplinary and geographic silos has led to the 'recurring nightmares' of short-term interests outcompeting long-term vision; situations where competition replaces co-operation; fragmentation of values and interest; fragmentation of authority and responsibility; and fragmentation of information and knowledge leading to inadequate solutions or even additional problems. In addition, the lack of institutional fit has often confounded the effectiveness of the stewardship of ecosystem services, and led to unexpected surprises, excessive rent seeking and high transaction costs.

Using counterfactual thinking (i.e. the dependence of *whether, when* and *how* one event occurs on *whether, when* and *how* another event occurs and the possible alteration of events), built around the four interconnected concepts of *planetary boundaries, tipping points, panarchy* and *resilience*, the chapter provides an analytical lens through which to explore why many of the warning signals were not seen. The chapter concludes by suggesting why ecosystems are likely to be even more at risk in the future and why we will need to observe and interpret the dynamics of both nature and institutions ever more closely if we are to avoid sudden irreversible ecological changes.

18 Late lessons from Chernobyl, early warnings from Fukushima

Paul Dorfman, Aleksandra Fucic and Stephen Thomas

The nuclear accident at Fukushima in Japan occurred almost exactly 25 years after the Chernobyl nuclear accident in 1986. Analysis of each provides valuable late and early lessons that could prove helpful to decision-makers and the public as plans are made to meet the energy demands of the coming decades while responding to the growing environmental costs of climate change and the need to ensure energy security in a politically unstable world.

This chapter explores some key aspects of the Chernobyl and Fukushima accidents, the radiation releases, their effects and their implications for any construction of new nuclear plants in Europe. There are also lessons to be learned about nuclear construction costs, liabilities, future investments and risk assessment of foreseeable and unexpected events that affect people and the environment.

Since health consequences may start to arise from the Fukushima accident and be documented over the next 5–40 years, a key lesson to be learned concerns the multifactorial nature of the event. In planning future radiation protection, preventive measures and bio-monitoring of exposed populations, it will be of great importance to integrate the available data on both cancer and non-cancer diseases following overexposure to ionising radiation; adopt a complex approach to interpreting data, considering the impacts of age, gender and geographical dispersion of affected individuals; and integrate the evaluation of latency periods between exposure and disease diagnosis development for each cancer type.

Given the degree of uncertainty and complexity attached to even the most tightly framed and rigorous nuclear risk assessment, attempts to weight the magnitude of accident by the expected probability of occurrence have proven problematic, since these essentially theoretical calculations can only be based on sets of pre-conditioning assumptions. This is not an arcane philosophical point but rather a very practical issue with significant implications for the proper management of nuclear risk. With its failure to plan for the cascade of unexpected beyond design-base accidents, the regulatory emphasis on risk-based probabilistic assessment has proven very limited. An urgent reappraisal of this approach and its real-life application seems overdue.

Whatever one's view of the risks and benefits of nuclear energy, it is clear that the possibility of catastrophic accidents and consequent economic liabilities must be factored into the policy and regulatory decision-making process. In the context of current collective knowledge on nuclear risks, planned pan-European liability regimes will need significant re-evaluation.

19 Hungry for innovation: from GM crops to agroecology

David A. Quist, Jack A. Heinemann, Anne I. Myhr, Iulie Aslaksen and Silvio Funtowicz

Innovation's potential to deliver food security and solve other agriculture-related problems is high on the agenda of virtually all nations. This chapter looks at two different examples of food and agricultural innovation: genetically modified (GM) crops and agroecological methods, which illustrate how different innovation strategies affect future agricultural and social options.

GM crops are well suited to high-input monoculture agricultural systems that are highly productive but largely unsustainable in their reliance on external, non-renewable inputs. Intellectual property rights granted for GM crops often close down, rather than open up further innovation potential, and stifle investment into a broader diversity of innovations allowing a greater distribution of their benefits.

Science-based agroecological methods are participatory in nature and designed to fit within the dynamics underpinning the multifunctional role of agriculture in producing food, enhancing biodiversity and ecoystem services, and providing security to communities. They are better suited to agricultural systems that aim to deliver sustainable food security than high external input approaches. They do, however, require a broader range of incentives and supportive frameworks to succeed. Both approaches raise the issue of the governance of innovation within agriculture and more generally within societies.

The chapter explores the consequences of a 'top-down transfer of technology' approach in addressing the needs of poor farmers. Here innovation is often framed in terms of economic growth in a competitive global economy, a focus that may conflict with efforts to reduce or reverse environmental damage caused by existing models of agriculture, or even deter investment into socially responsible innovation.

Another option explored is a 'bottom-up' approach, using and building upon resources already available: local people, their knowledge, needs, aspirations and indigenous natural resources. The bottom-up approach may also involve the public as a key actor in decisions about the design of food systems, particularly as it relates to food quality, health, and social and environmental sustainability.

Options are presented for how best to answer consumer calls for food quality, sustainability and social equity in a wide sense, while responding to health and environmental concerns and securing livelihoods in local small-scale agriculture. If we fail to address the governance of innovation in food, fibre and fuel production now, then current indications are that we will design agriculture to fail.

20 Invasive alien species: a growing but neglected threat?

Sarah Brunel, Eladio Fernández-Galiano, Piero Genovesi, Vernon H. Heywood, Christoph Kueffer and David M. Richardson

Biological invasions are one of the five major causes of biodiversity loss as global human travel and trade have moved, and continue to move, thousands of species between and across continents. Some species of alien origin have a high probability of unrestrained growth which can ultimately lead to environmental damage.

An alien species — animal, plant or microorganism — is one that has been introduced, as a result of human activity, either accidentally or deliberately, to an area it could not have reached on its own. A common definition of the term 'invasive' focuses on its (negative) impact, while other definitions consider only rate of spread and exclude considerations of impact.

Despite the growing amount of legislation being adopted at the global scale, biological invasions continue to grow at a rapid rate, with no indication yet of any saturation effect. Decision-making in this area is very challenging. The overall complexity of the problem, its interdisciplinarity, the scientific uncertainties and the large number of stakeholders that need to be informed and involved, together demand governance actions that are difficult to see emerging at the regional scale (as in the EU), let alone globally.

It is widely agreed that preventing biological invasions or tackling them at a very early stage is the most efficient and cost-effective approach. Harmless species can be confused with harmful invasive species, however, leading to a waste of resources. Even more seriously, harmful invaders can be mistaken for innocuous species — so-called 'invaders in disguise' — and no appropriate action may be taken to counter the threats they pose.

Even with a very good risk assessment system, new outbreaks of invasive alien species could still occur, necessitating a system of rapid early warning and effective eradication response. The decision on where to draw the line on the acceptable environmental risks versus the introduction of new species or new communities that may carry invasive alien species then becomes a value judgement.

There is lively debate within the scientific community regarding the most appropriate strategies for managing invasive alien species. Governments and institutions charged with making decisions have access to considerable knowledge on the topic, but the lack of rules of interactions between multiple parties regularly thwarts effective decision-making.

21 Mobile phones and brain tumour risk: early warnings, early actions?

Lennart Hardell, Michael Carlberg and David Gee

In 2011 the World Health Organization's International Agency for Research on Cancer (IARC) categorised the radiation fields from mobile phones and other devices that emit similar non-ionizing electromagnetic fields (EMFs), as a Group 2B i.e. 'possible' human carcinogen. Nine years earlier IARC gave the same classification to the magnetic fields from overhead electric power lines.

The IARC decision on mobile phones was principally based on two sets of case-control human studies of possible links between mobile phone use and brain tumours: the IARC Interphone study and the Hardell group studies from Sweden. Both provided complementary and generally mutually supportive results. This chapter gives an account of the studies by these two groups — and others coming to different conclusions — as well as reviews and discussions leading up to the IARC decision in 2011. The chapter also describes how different groups have interpreted the authoritative IARC evaluation very differently.

There are by now several meta-analyses and reviews on mobile phones and brain tumours, which describe the challenges of doing epidemiology on this issue, the methodological limitations of the major studies published so far and the difficulties of interpreting their results.

It has been suggested that national incidence data on brain tumours could be used to qualify or disqualify the association between mobile phones and brain tumours observed in the case-control studies. However, in addition to methodological shortcomings, there might be other factors that influence the overall incidence rate such as changes in exposure to other risk factors for brain tumours that are unknown in descriptive studies. Cancer incidence depends on initiation, promotion and progression of the disease. As the mechanism for radiofrequency electromagnetic fields carcinogenesis is unclear, it supports the view that descriptive data on brain tumour incidence is of limited value.

The chapter points to mobile phone industry inertia in considering the various studies and taking the IARC carcinogenic classification into account and a failings from the media in providing the public with robust and consistent information on potential health risks. The IARC carcinogenic classification also appears not to have had any significant impact on governments' perceptions of their responsibilities to protect public health from this widespread source of radiation.

The benefits of mobile telecommunications are many but such benefits need to be accompanied by consideration of the possibility of widespread harms. Precautionary actions now to reduce head exposures would limit the size and seriousness of any brain tumour risk that may exist. Reducing exposures may also help to reduce the other possible harms that are not considered in this case study.

Evidence is increasing that workers with heavy long-term use of wireless phones who develop glioma or acoustic neuroma should be compensated. The first case in the world was established on 12 October 2012. The Italian Supreme Court affirmed a previous ruling that the Insurance Body for Work (INAIL) must grant worker's compensation to a businessman who had used wireless phones for 12 years and developed a neuroma in the brain.

22 Nanotechnology — early lessons from early warnings

Steffen Foss Hansen, Andrew Maynard, Anders Baun, Joel A. Tickner and Diana M. Bowman

Nanotechnology is the latest in a long series of technologies heralded as ushering in a new era of technology-driven prosperity. Current and future applications of nanotechnology are expected to lead to substantial societal and environmental benefits, increasing economic development and employment, generating better materials at lower environmental costs, and offering new ways to diagnose and treat medical conditions. Nevertheless, as new materials based on nanoscale engineering move from the lab to the marketplace, have we learnt the lessons of past 'wonder technologies' or are we destined to repeat past mistakes?

This chapter first introduces nanotechnology, clarifies the terminology of nanomaterials and describes current uses of these unique materials. Some of the early warning signs of possible adverse impacts of some nanomaterials are summarised, along with regulatory responses of some governments. Inspired by the EEA's first volume of *Late lessons from early warnings*, the chapter looks critically at what lessons can already be learned, notwithstanding nanotechnology's immaturity.

Nanotechnology development has occurred in the absence of clear design rules for chemists and materials developers on how to integrate health, safety and environmental concerns into design. The emerging area of 'green nanotechnology' offers promise for the future with its focus on preventive design. To gain traction, however, it is important that research on the sustainability of materials is funded at levels significant enough to identify early warnings, and that regulatory systems provide incentives for safer and sustainable materials.

Political decision-makers have yet to address many of the shortcomings in legislation, research and development, and limitations in risk assessment, management and governance of nanotechnologies and other emerging technologies. As a result, there remains a developmental environment that hinders the adoption of precautionary yet socially and economically responsive strategies in the field of nanotechnology. If left unresolved, this could hamper society's ability to ensure responsible development of nanotechnologies.

23 Understanding and accounting for the costs of inaction

Mikael Skou Andersen and David Owain Clubb

In political decision-making processes, the burden of proof is often distributed such that policymakers only respond to early warning signals from environmental hazards once the costs of inaction have been estimated.

This chapter revisits some key environmental issues for which estimates of costs of inaction have been carefully developed over many years of research. The aim is to consider the methodological challenges involved in producing estimates that are credible and appropriate rather than present specific estimates for these costs.

The case studies also provide insights into how early warning signals might provide a basis for estimating the costs of inaction, when the science base is less consolidated. For example, the case of nitrates in drinking water illustrates that a precautionary approach to the costs of inaction is quite conceivable. The phase-out of ozone-depleting substances, where early-warning scientists successfully alerted the world to the damaging effects of chlorofluorocarbons (CFCs), provides another important case because additional impacts for global warming actually cause the costs of inaction to be considerably higher than initially believed. This is a reminder that figures for the costs of inaction have often been grossly underestimated.

Finally, in the case of air pollution, making use of different estimates for mortality risk avoidance will help decision-makers to see that there are higher- and lower-bound estimates for the costs of inaction. Even if the lower-bound estimates are perhaps too conservative, with a bias towards health effects, they will in many situations encourage more rather than less abatement effort. Reducing emission loads will also tend to bring relief for the intangible assets of biodiversity and nature.

Making the best use of environmental science and modelling helps to make environmental protection and precaution a priority. Producing cost estimates should not be left to economists alone, but should rather be seen as a starting point for a broader discussion, featuring also the relevant expertise in health, ecology, demography, modelling and science. Well researched estimates, based on interdisciplinary collaboration, can strengthen some of those scattered and diffuse interests, which during the ordinary processes of policy-making have difficulty making their voices heard.

24 Protecting early warners and late victims

Carl Cranor

Many *Late lessons from early warnings* chapters provide examples of early warning scientists who were harassed for bringing inconvenient truths about impending harm to the attention of the public and regulators. There is also some evidence that young scientists are being discouraged from entering controversial fields for fear of such harassment. In addition, where warnings have been ignored and damage has ensued, it has often proven difficult in the past to achieve prompt and fair compensation for the victims. Some ideas for reform, building on some current institutional models are explored here.

This chapter first explores the idea of extending whistleblowing laws to help encourage and protect early-warning scientists and others who identify evidence of impending harm. Complementary measures, such as greater involvement of professional societies and the use of recognition awards, as for example in Germany, could also be helpful.

Next, the chapter explores improved mechanisms for compensating victims of pollution and contamination. The chapter on the Minamata Bay disaster provides an extreme example of long delays in getting adequate compensation for the victims of methylmercury poisoning. It was almost fifty years, between 1956 and 2004, before the victims attained equitable levels of compensation and legal recognition of responsibility. Other case studies illustrate similar examples of long delays in receiving adequate compensation.

Options are examined for providing justice to any future victims of those emerging technologies such as nanotechnology, genetically modified crops and mobile phone use, which currently can provide broad public benefits but potentially at a cost to small groups of victims. The potential for widespread exposure and uncertain science could justify 'no-fault' administrative schemes that provide more efficient and equitable redress in situations where the benefit of scientific doubt would be given to victims. The use of anticipatory assurance bonds to help minimise and meet the costs of future environmental damage from large scale technologies is also explored.

A supplementary panel text describes cases of asbestos and mesothelioma, where the senior courts in the United Kingdom have developed innovative ways of dealing with both joint and several liability, and the foreseeability of subsequent asbestos cancers, after the initial recognition of the respiratory disease, asbestosis. Such legal developments in the field of personal injury could illustrate the future direction of long-tail liability in both environmental damage and personal injury.

25 Why did business not react with precaution to early warnings?

Marc Le Menestrel and Julian Rode

In the past, companies have frequently neglected early warning signals about potential hazards for human health or the environment associated with their products or operations. This chapter reviews and analyses relevant interdisciplinary literature and prominent case studies — in particular those documented in both volumes of *Late lessons from early warnings* — and identifies main factors responsible for the disregard of early warning signals.

The chapter shows how economic motives often drive non-precautionary business decisions. In virtually all reviewed cases it was perceived to be profitable for industries to continue using potentially harmful products or operations. However, decisions are also influenced by a complex mix of epistemological, regulatory, cultural and psychological aspects. For instance, characteristics of the research environment and the regulatory context can provide business actors with opportunities to enter into 'political actions' to deny or even suppress early warning signals. Also, business decision-makers face psychological barriers to awareness and acceptance of the conflicts of values and interests entailed by early warning signals. Cultural business context may further contribute to the denial of conflicts of values.

The chapter concludes with a set of reflections on how to support more precautionary business decision making. A prominent policy response to the conflicting interests of business and society is introducing regulations that attempt to steer business rationality towards internalising external effects. Innovative solutions such as assurance bonding should be considered.

There is a need to better understand and expose why business actors do not respond voluntarily to early warning signals with precautionary actions. Blaming business, in particular with hindsight, tends to be common reaction that may not always be constructive. It often misses the complex or even contradictory set of motives and drivers that business actors face.

Public institutions could support progressive business by analysing and publically disclosing the dilemmas and temptations entailed by early warning signals, for example for different industries and for the specific societal and regulatory context of decisions. Rigorous and explicit exposition of the dilemmas will create further incentives for responsible actors to share and communicate their precautionary responses.

An additional reflection centres on the role of political actions of business actors, in particular those actions aimed at suppressing early warning signals. Regulatory efforts that make the political actions of business more transparent can help to sustain a sound balance of power, thereby maintaining our ability to benefit from early warning signals and reducing the likelihood of health and environmental hazards.

26 Science for precautionary decision-making

Philippe Grandjean

The goals of academic researchers may differ from those of regulatory agencies responsible for protecting the environment. Thus, research must take into account issues such as feasibility, merit and institutional agendas, which may lead to inflexibility and inertia.

A large proportion of academic research on environmental hazards therefore seems to focus on a small number of well studied environmental chemicals, such as metals. Research on environmental hazards should therefore to a greater extent consider poorly known problems, especially the potential hazards about which new information is in particular need.

Misinterpretation may occur when results published in scientific journals are expressed in hedged language. For example, a study that fails to document with statistical significance the presence of a hazard is often said to be negative, and the results may be misinterpreted as evidence that a hazard is absent. Such erroneous conclusions are inspired by science traditions, which demand meticulous and repeated examination before a hypothesis can be said to be substantiated.

For prioritising needs for action, research should instead focus on identifying the possible magnitude of potential hazards. Research is always affected by uncertainties and many of them can blur a real association between an environmental hazard and its adverse effects, thereby resulting in an underestimated risk. Environmental health research therefore needs to address the following question: are we sufficiently confident that this exposure to a potential hazard leads to adverse effects serious enough to initiate transparent and democratic procedures to decide on appropriate intervention?

The choice of research topics must consider societal needs for information on poorly known and potentially dangerous risks. The research should be complementary and extend current knowledge, rather than being repetitive for verification purposes, as required by the traditional science paradigm. Research findings should be openly available and reported so that they inform judgments concerning the possible magnitude of suspected environmental hazards, thereby facilitating precautionary and timely decision-making.

27 More or less precaution?

David Gee

Despite its presence in a growing body of EU and national legislation and case law, the application of the precautionary principle has been strongly opposed by vested interests who perceive short term economic costs from its use. There is also intellectual resistance from scientists who fail to acknowledge that scientific ignorance and uncertainty, are excessively attached to conventional scientific paradigms, and who wait for very high strengths of evidence before accepting causal links between exposure to stressors and harm.

The chapter focuses on some of the key issues that are relevant to a more common understanding of the precautionary principle and to its wider application. These include different and confusing definitions of the precautionary principle and of related concepts such as prevention, risk, uncertainty, variability and ignorance; common myths about the meaning of the precautionary principle; different approaches to the handling of scientific complexity and uncertainty; and the use of different strengths of evidence for different purposes.

The context for applying the precautionary principle also involves considering the 'knowledge to ignorance' ratio for the agent in focus: the precautionary principle is particularly relevant where the ratio of knowledge to ignorance is low, as with emerging technologies.

A working definition of the precautionary principle is presented that aims to overcome some of the difficulties with other definitions, such as their use of triple negatives; a failure to address the context of use of the precautionary principle; no reference to the need for case specific strengths of evidence to justify precaution; and overly narrow interpretations of the pros and cons of action or inaction.

The chapter also points to the need for greater public engagement in the process of framing and decision-making about both upstream innovations and their downstream hazards, including the specification of the 'high level of protection' required by the EU treaty. A precautionary and participatory framework for risk analysis is proposed, along with some 'criteria for action' to complement criteria for causation.

The capacity to foresee and forestall disasters, especially when such action is opposed by powerful economic and political interests, appears to be limited, as the case studies in *Late lesson from early warnings* illustrate. The chapter argues that with more humility in the face of uncertainty, ignorance and complexity, and wider public engagement, societies could heed the lessons of past experience and use the precautionary principle, to anticipate and minimise many future hazards, whilst stimulating innovation. Such an approach would also encourage more participatory risk analysis; more realistic and transparent systems science; and more socially relevant and diverse innovations designed to meet the needs of people and ecosystems.

28 In conclusion

The first volume of *Late lessons from early warnings* highlighted the difficulties of balancing precaution with technological innovation and ended with a call to action for policymakers. How much progress has been made since then?

First, there is growing evidence that precautionary measures do not stifle innovation, but instead can encourage it, in particular when supported by smart regulation or well-designed tax changes. Not only has the body of knowledge become richer since 2001, but also the number of stakeholders involved in decision-making has become larger and more diverse. There has also been increasing attention to communicating scientific uncertainty, especially in the fields of climate change, food safety, and emerging risks.

However, there has been less progress in other areas: for example, many of the political and scientific 'bureaucratic silos' still remain, despite frequent calls for policy integration and inter-departmental coordination. This has led to the unintended destruction of stocks of natural capital in some parts of the world and in other instances, the global spread of technologies, despite warnings of impending hazards. The result has been widespread damage, with most polluters still not paying the full costs of pollution.

Yet, more encouragingly, new transformative approaches are emerging to manage the systemic and interconnected challenges the world faces e.g. economic/financial, climate/energy, ecosystems/food. These relate, inter alia, to the increasing use of digital communications and networking by consumers, citizens and shareholders to demand and foster increased participation, more social responsibility, greater levels of accountability and higher transparency, especially in determining future pathways for energy and food production. There is a greater understanding of the complexity of the environment, of scientific ignorance and uncertainties, the irreversibility of many harmful impacts and on the broader risks to the long term interests of society if political and financial institutions remain unchanged. Also some corporations are fundamentally embracing sustainable development objectives in their business models and activities.

The case studies across both volumes of *Late lessons from early warnings* cover a diverse range of chemical and technological innovations, and highlight a number of systemic problems. These include a lack of institutional and other mechanisms to respond to early warning signals; a lack of ways to correct market failures either caused by misleading market prices or where costs and risks to society and nature are not properly internalised; and the fact that key decisions on innovation pathways are made by those with vested interests and/or by a limited number of people on behalf of many. The insights and lessons drawn from the case histories certainly provide the seeds for some of the answers. They also provide knowledge for a series of key actions that are outlined below.

Of course, many questions remain. For example: how can the precautionary principle be used further to support decision-making in the face of uncertainties and the inevitable surprises that come from complex systems?; how can societies avoid a lack of 'perfect' knowledge being used as a justification for inaction in the face of 'plausible' evidence of serious harm?; how can conflicting interests be balanced during the phases of development and use?; and how can the benefits of products and technologies be more equitably distributed?

Reduce delays between early warnings and actions

The majority of the case studies in *Late lessons from early warnings* Volumes 1 and 2 illustrate that if the precautionary principle had been applied on the basis of early warnings, justified by 'reasonable grounds for concern' many lives would have been saved and much damage to ecosystems avoided. It is therefore very important that large scale emerging technologies, such as biotechnologies, nanotechnologies and information

and communication technologies, apply the precautionary principle based on the experiences and lessons learned from these and other case studies.

Precautionary actions can be seen to stimulate rather than hinder innovation; they certainly do not lead to excessive false alarms. As the analysis in Volume 2 shows, of 88 cases of claimed 'false positives', where hazards were wrongly regulated as potential risks, only four were genuine false alarms. The frequency and scale of harm from the mainly 'false negative' case studies indicate that shifting public policy towards avoiding harm, even at the cost of some false alarms, would seem to be worthwhile, given the asymmetrical costs of being wrong in terms of acting or not acting based on credible early warnings.

However, the speed and scale of today's technological innovations can inhibit timely action. This is often because by the time clear evidence of harm has been established, the technology has been modified, thereby allowing claims of safety to be subsequently re-asserted. Even where the technological change has been marginal, the large, often global, scale of investment can lead to widespread technological lock-in, which is then difficult and expensive to alter.

These features of current technological innovation strengthen the case for taking early warning signals more seriously and acting on lower strengths of evidence than those normally used to reach 'scientific causality'. Most of the historical case studies show that by the time such strong evidence of causality becomes available, the harm to people and ecosystems has become more diverse and widespread than when first identified, and may even have been caused by much lower exposures than those initially considered dangerous.

The case studies have also shown that there are many barriers to precautionary action, including: the short-term nature of most political and financial horizons; the existence of technological monopolies; the conservative nature of the sciences involved, including the separate 'silos' within which they operate; the power of some stakeholders; and the cultural and institutional circumstances of public policymaking that often favour the status quo.

Acknowledge complexity when dealing with multiple effects and thresholds

Increasing scientific knowledge has shown that the causal links between stressors and harm are more complex than was previously thought and this has practical consequences for minimising harm. Much of the harm described in Volumes 1 and 2, such as cancers or species decline, is caused by several co-causal factors acting either independently or together. For example, the reduction of intelligence in children can be linked to lead in petrol, mercury and polychlorinated biphenyls (PCBs) as well as to socio-economic factors; bee colony collapse can be linked to viruses, climate change and nicotinoid pesticides; and climate change itself is caused by many complex and inter-linked chemical and physical processes.

In some cases, such as foetal or fish exposures, it is the timing of the exposure to a stressor that causes the harm, not necessarily the amount; the harm may also be caused or exacerbated by other stressors acting in a particular timed sequence. In other cases, such as radiation and some chemicals such as bisphenol A (BPA), low exposures can be more harmful than high exposures; and in others, such as asbestos with tobacco, and some endocrine disrupting substances, the harmful effects of mixtures can be greater than from each separate stressor. There are also varying susceptibilities to the same stressors in different people, species and ecosystems, depending on pre-existing stress levels, genetics and epigenetics. This variation can lead to differences in thresholds or tipping point exposures, above which harm becomes apparent in some exposed groups or ecosystems but not others. Indeed there are some harmful effects which occur only at the level of the system, such as a bee colony, which cannot be predicted from analysing a single part of the system, such as an individual bee.

Our increased knowledge of complex biological and ecological systems has also revealed that certain harmful substances, such as polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichlorethane (DDT) can move around the world via a range of biogeochemical and physical processes and then accumulate in organisms and ecosystems many thousands of kilometres away.

The practical implications of these observations are threefold. First, it is very difficult to establish very strong evidence that a single substance or stressor 'causes' harm to justify timely actions to avoid harm; in many cases only reasonable evidence of co-causality will be available. Second, a lack of consistency between research results is not a strong reason for dismissing possible causal links: inconsistency is to be expected from complexity. Third, while reducing harmful exposure to one co-causal factor may not necessarily lead to a large reduction in the overall harm caused by many other

factors, in some cases the removal of just one link in the chain of multi-causality could reduce much harm.

A more holistic and multi-disciplinary systems science is needed to analyse and manage the causal complexity of the systems in which we live.

Rethink and enrich environment and health research

Environment and health research overly focuses on well-known rather than unknown hazards at the expense of emerging issues and their potential impacts. For example the ten most well-known substances, such as lead and mercury, account for about half of all articles on chemical substances published in the main environmental journals over the last decade. Over the past decade, public research funding in the European Union on nanotechnology, biotechnology as well as Information and Communications Technology (ICT) is heavily biased towards product development with about 1 % being spent on their potential hazards. A more equal division of funding between known and emerging issues, and between products and their hazards, would enrich science and help avoid future harm to people and ecosystems and to the long term economic success of those technologies.

Funding more holistic systems science would also help achieve a greater integration among the different branches of science and counteract problems such as: peer review predominantly within and not across disciplines; short-term interests outcompeting long-term vision; competition replacing cooperation because of conflicts of interest; contradictions amongst paradigms; fragmentation of values and authority; as well as fragmentation of information and knowledge. These can all lead to inferior solutions and provide increased opportunities for those with vested interests to manufacture doubt.

Scientific methods can also be improved. For example, much higher strengths of evidence are required overall before causality is accepted, compared to the evidence being used to assert safety. The assertion that there is *no evidence of harm* is then often assumed to be *evidence of no harm*, even though the relevant research is missing. Historically there has been an over-reliance on the statistical significance of point estimates compared to confidence limits based on multiple sampling. There has also been a bias towards using models that grossly simplify reality rather than using long-term observations and trend data of biological and ecological systems. These approaches have sometimes led to the production of false positives. More importantly the governance of scientific ignorance and unknown unknowns has been neglected.

Finally, many case studies highlight the problems faced by early warning scientists who have been harassed for their pioneering work, including bans on speaking out or publishing, loss of funding, legal or other threats, and demotion. One obvious conclusion is that scientists in these situations should receive better protection either via an extension of 'whistle blowing' and discrimination laws, or by independent acknowledgement of the value of their work.

Improve the quality and value of risk assessments

The majority of the case studies in *Late lessons from early warnings* indicate that risk assessment approaches need to better embrace the realities of causal and systems complexity (rather than use a narrow conception of 'risk') with the inevitable features of ignorance, indeterminacy and contingency. In a number of case studies, for example BPA, where low doses are more harmful than high doses, or tributyltin antifoulants (TBT) and synthetic oestrogen diethylstilboestrol (DES) where the timing of the dose is what makes it harmful, simplistic assumptions are inadequate. Variability in exposures and varying susceptibilities in populations and species exposed also need to be more realistically factored into risk assessments.

This is equally true for technological risk assessments. As the Fukushima Investigation Committee concluded in 2011:

> '…the accidents present us with crucial lessons on how we should be prepared for 'incidents beyond assumptions'. With its failure to plan for the cascade effects beyond design–base accidents 'the regulatory emphasis on risk based probabilistic risk assessment has proven very limited'.

In other words, narrow risk assessment approaches are now outstripped by the realities which they cannot address, recognise and communicate. Too often this contributes to the effective denial of those risks that do not fit the risk assessment frame. It is therefore urgent that risk assessment practices

be transformed to make them broader-based, more inclusive, transparent and accountable. There should also be more communication on the diversity of scientific views, especially on emerging issues where ignorance and uncertainties are high and genuine differences of scientific interpretations are likely, desirable, and defensible. In this sense, recognising the pedigree of knowledge, i.e. the consistency of views amongst peers and the level of convergence coming from different branches of research, is essential for effective decision making and action to support the wellbeing of people and the environment.

The case studies show that evaluations of evidence in risk assessments can be improved by including a wide range of stakeholders when framing the risks and options agenda; broadening the scope and membership of evaluation committees; increasing the transparency of committee approaches and methods, particularly in identifying uncertainties and ignorance; and ensuring their independence from undue influence through using appropriate funding sources and applying robust policies on conflicts of interest.

Public confidence would be increased if all the evidence used in risk assessments was made publicly accessible and open to independent verification, including data submitted by industries to authorities.

As experiences from mercury, nuclear accidents, leaded petrol, mobile phones, BPA, and bees show, there can be a significant divergence in the evaluations of the same, or very similar, scientific evidence by different risk assessment committees. In such instances, differences in the choice of paradigm, assumptions, criteria for accepting evidence, weights placed on different types of evidence, and how uncertainties were handled, all need to be explained. Risk assessors and decision makers also need to be aware that complexity and uncertainty have sometimes been misused to shift the focus away from precautionary actions by 'manufacturing doubt' and by waiting for 'sound science' approaches that were originally developed by the tobacco industry to delay action.

Foster cooperation between business, government and citizens

Policy formulation should start from a broad concept of technological innovation to include non-technological, social, institutional, organisational and behavioural innovation. In this framework, governments have at least three roles: providing direction by putting in place smart regulations and consistent market signals; ensuring that the distributional consequences of innovations are balanced between risks and rewards across society, fostering a diversity of innovations so that the wider interests of society; and take precedence over narrower interests.

Numerous case studies show that decisions to act without precaution often come from businesses. There are, however, several impediments to businesses acting in a precautionary manner, including a focus on short-term economic value for shareholders alongside psychological factors that lead to a so-called 'ethical blindness' or a 'self-serving bias' whereby people largely interpret ambiguous situations in their own interests. Governments and businesses could collaborate more with citizens on publicly disclosing the potential value conflicts entailed in acting on early warning signals. A culture of transparency can in turn promote positive business attitudes and innovations.

Involving the public can also help in choosing between those innovation pathways to the future; on prioritising relevant public research; on providing data and information in support of monitoring and early warnings; improving risk assessments; on striking appropriate trade-offs between innovations and plausible health and environmental harms; and, making decisions about risk-risk trade-offs.

Correcting market failures using the polluter pays and prevention principles

When evidence of initial harm emerges, the costs should be internalised retroactively into the prices of polluting products, via taxes and charges, in line with the polluter pays principle and emerging practice across the world. The revenues could then be devoted partly to stimulating research into less hazardous alternatives, and partly to reform tax systems by reducing taxes and charges on 'societal goods' like employment.

The pollution taxes/charges would rise or fall in line with new scientific knowledge about increasing/decreasing harm, and this would help to level the playing field for less-polluting alternative products. Tax shifts from employment to pollution and the inefficient use of resources can bring multiple benefits such as increased employment, a stimulus to innovation, a more stable tax base in the light of expected demographic changes, and a more efficient tax collection system.

More broadly, firms and governments need to extend their economic accounting systems to incorporate the full impacts of their activities on people's health and on ecosystems. Governments need to anticipate this in their policies, by providing the right blend of fiscal instruments to both protect the public and ensure that firms internalise the true costs of potential harm.

A number of case studies also demonstrate the long time lags between evidence of harm and the additional injustice and time of forcing victims to pursue their cases through civil compensation claims. Prompt and anticipatory no-fault compensation schemes and assurance bonds, could be set up and financed in advance of potential harm by the industries that are producing novel and large-scale technologies, thereby helping to offset any potential market failure. Such schemes can also be designed to increase the incentives for innovating companies to carry out more *a priori* research into the identification and elimination of hazards.

Governance of innovation and innovation in governance

The *Late lessons from early warnings* reports demonstrate the complexities of developing not only the right kind of science and knowledge but also handling the interactions between the many actors and institutions involved — governments, policymakers, businesses, entrepreneurs, scientists, civil society representatives, citizens and the media.

Alongside many other analyses produced across the world today, the reports also stress the need to act to transform our ways of thinking and of doing, and urgently so in the face of unprecedented global changes, challenges and opportunities. Many lessons have been learnt, yet have not been acted upon. Any calls for action will need to reflect on today's global socio-economic setting and support, among other things, the drive to:

- rebalance the prioritisation of economic and financial capital over social, human and natural capitals through the broader application of the policy principles of precaution, prevention and polluter-pays, and environmental accounting;

- broaden the nature of evidence and public engagement in choices about key innovation pathways by directing scientific efforts more towards dealing with complex, systemic challenges and unknowns and complementing this with professional, lay, local and traditional knowledge; and,

- build greater adaptability and resilience in governance systems to deal with multiple systemic threats and surprises, through strengthening institutional structures and deploying information technologies in support of the concept of responsible information and dialogues.

The governance of innovation will remain at the level of good intentions unless it is translated into innovations in science practices, institutional arrangements and public engagements as well as transformations in prevailing business attitudes, practice and influence. These are the tasks that lie ahead.

In memory of Masazumi Harada, 1935–2012

Masazumi Harada, a physician involved for many years in the study of the mercury poisoning Minamata disease, died in June 2012 of acute myelocytic leukemia at his home in Kumamoto City. He was 77.

Harada conducted medical examinations on the disease's sufferers for the first time in the summer of 1961 in Minamata city in Kumamoto Prefecture while he was a student at Kumamoto University's graduate school.

Shocked by their miserable lives, Harada devoted himself to the study of the disease from that time. Harada published a thesis on congenital Minamata disease in 1964. The work had a significant impact as it disproved the conventional belief at the time that the placenta does not pass poisons. He received an award from the Japanese Society of Psychiatry and Neurology for the thesis in 1965.

He then established the Open Research Center for Minamata Studies at the university in 2005, becoming the center's head. He continued to lead the disease's research from non-medical perspectives as well. Harada visited Brazil, China and native Indian communities in Canada to discover those suspected of suffering from the disease.

Author of many books, Harada wrote 'Minamata Byo' (Minamata Disease), which raised awareness on the issue around the world.

Dr. Masazumi Harada first came to Asubpeeschoseewagong (Grassy Narrows) and Wabaseemoong (White Dog) First Nations in Canada in the early 1970s. Harada's death comes at the end of River Run 2012, five days of actions by members and supporters of Grassy Narrows in Toronto, who are seeking to have Minamata disease recognized in Canada and Ontario. Harada's final report for the Grassy Narrows community was released on 4 June 2012 after 30 years of research, showing mercury deposited in the river by the Dryden paper mill in the 1970s is impacting those who were not yet born when the dumping ceased.

In memory of Poul Harremoës, 1934–2003

Poul Harremoës was a key player in environmental issues in Denmark and internationally for more than 30 years until his death, at 69, in 2003. In that time, those who worked closely with him benefited from a continuous, almost daily flow of excellent ideas for new research projects.

He was a member of the Danish Pollution Council, which prepared the first framework national law on environmental protection and advised on the establishment of a Ministry of Environment from 1971. He was a key participant in numerous settings, including the first Scientific Committee of the European Environment Agency from 1995.

He had a civil engineering degree from the Technical University of Denmark. He specialised early on in geo-technics and constructed dams on the Faroe Islands. While teaching geo-technics he wrote a textbook that was used for more than 40 years. However, he was able to quickly change his research direction and develop new areas of excellence. So, for example, he got a grant to study at Berkeley, California, from where he received a M.Sc. degree in environmental engineering.

In 1972, he became professor in environmental engineering at the Technical University of Denmark where he originally worked with wastewater discharge to the sea and the biological processes of wastewater treatment. He became a world leading scientist in the theories of biofilms for removal of organics and nitrogen from wastewater before turning to sewer design and modelling. In 2000, Poul was awarded the *Heineken prize for Environmental Sciences* for his contributions to the theory of biofilm kinetics in relation to biological waste water treatment and for his successful organisation of the international scientific community in water pollution research and control.

As a result of his work with sewers and storm water he went into the area of risk analysis and the role of the precautionary principle. In a short time he became an international expert in this field and was highly demanded for lectures in all parts of the world. A key outcome of his interest was his contributions as chairman of the editorial team for the first volume of *Late lessons from early warnings* published in 2001.

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MILJØMINISTERIET

Miljøstyrelsen

Metoder til fastsættelse af kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand med henblik på at beskytte sundheden

Vejledning fra Miljøstyrelsen **Nr. 5** 2006

Indhold

Forord

Principperne for vurdering og fastsættelse af kvalitetskriterier for kemikalier med henblik på at beskytte sundheden er beskrevet tidligere[1]. Men i forbindelse med Miljøministerens redegørelse om jordforureningsloven i 2003, besluttede regeringen at nedsætte en arbejdsgruppe, som fik til opgave at "*vurdere, hvorvidt de sundhedsmæssige kvalitetskriterier ligger på det rigtige niveau i relation til international praksis, samt give forslag til ændringer*.

Arbejdsgruppen udpegede en række politiske valg, der indgår i principperne for fastsættelse af kvalitetskriterier. På den baggrund blev der i december 2005 indgået en aftale mellem Miljøministeren og et bredt udsnit af Folketingets partier, som bl.a. fastlægger følgende generelle principper for fastsættelsen af forebyggende kriterier :

- Hovedparten af befolkningen skal fortsat beskyttes (dvs. hensyntagen til særligt udsatte skal også inddrages).
- Der skal tages specifikt hensyn til børn.
- Det fastholdes, at der højst anvendes en samlet usikkerhedsfaktor på 10.000, når resultater fra dyreforsøg overføres til mennesker.
- Der accepteres fortsat en livstidsrisiko på 10^{-6}.

Denne vejledning indarbejder disse principper og gennemgår de metoder, som skal anvendes ved fastsættelse kvalitetskriterier for jord, luft og drikkevand med henblik på at beskytte sundheden[2].

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[1] Miljøstyrelsens Vejledning nr. 1 "Sundhedsmæssig vurdering af kemiske stoffer i drikkevand ", 1992, og i bilag til Miljøstyrelsens Vejledning nr. 6, 1990 "Begrænsning af luftforurening fra virksomheder" samt i Miljøprojekt nr. 12, 1995 "Toksikologiske kvalitetskriterier for jord og drikkevand".

[2] Principper for fastsættelse af kvalitetskriterier med henblik på at beskytte miljøet er tilsvarende beskrevet i Miljøstyrelsens vejled-

Vejledningen henvender sig til centrale og lokale myndigheder, herunder embedslægeinstitutionerne, konsulenter, rådgivere og virksomheder.

Miljøstyrelsen fastsætter løbende kvalitetskriterier for konkrete kemiske stoffer i jord, luft og drikkevand med henblik på at beskytte sundheden, jf. Miljøbeskyttelsesloven § 14, stk. 1,. Kvalitetskriterierne bliver fastsat på baggrund af videnskabelige rapporter og efter drøftelse i en styregruppe med deltagelse af bl.a. Fødevarestyrelsen, Arbejdstilsynet og Sundhedsstyrelsen.

Denne vejledning og detaljeringsgraden, ikke mindst i den tilhørende faglige rapport[3], skal give grundlag for større forståelse for, hvordan kvalitetskriterier skal udarbejdes.

Kvalitetskriterierne anvendes dels til at vurdere alvoren af en allerede given forurening, og dels når myndighederne stiller krav i forbindelse med udledning af konkrete stoffer til omgivelserne.

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ning nr. 4 (2004): Principper for fastsættelse af vandkvalitetskriterier for stoffer i overfladevand.

[3] Miljøprojekt Nr. 974, Miljøstyrelsen (2005). Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand. Rapporten er udarbejdet af Institut for Fødevaresikkerhed og Ernæring, Fødevaredirektoratet (nu Danmarks Fødevareforskning) i samarbejde med Miljøstyrelsen.

1 Indledning

1.1 Hvad er kvalitetskriterier

1.1.1 Formål med fastsættelse af kvalitetskriterier for kemikalier

Denne vejledning beskriver den administrative praksis ved fastsættelse af kvalitetskriterier for kemikalier med henblik på at beskytte sundheden.

Kvalitetskriterier danner basis for en række vurderinger af administrativ karakter, som vedrører både jord, luft og drikkevand.

Målsætningen ved fastsættelsen af kvalitetskriterier for kemikalier er, at de skal medvirke til *at forebygge forurening og skader på sundheden*. Kvalitetskriterierne ses som et element i lovens overordnede politiske målsætning om at oppebære et højt beskyttelsesniveau for befolkningens sundhed og sikre en bæredygtig udvikling, herunder fremme af renere teknologi.

Kvalitetskriterierne angiver et højt beskyttelsesniveau, hvor ingen effekt kan forventes, selv ved udsættelse gennem et helt liv, eller hvis der er tale om et stof uden tærskelværdi, en teoretisk forøget risiko for kræft hos én ud af en million mennesker, som er udsat for stoffer gennem et helt liv på 70 år.

Kvalitetskriterierne bruges af myndighederne i forbindelse med vurdering af alvoren af et givet forureningsniveau. Det kan være relevant, hvis der er tale om en eksisterende forurening (fortidens synder), eller hvis der skal fastsættes kravværdier for udledning af konkrete stoffer i miljøet (forebyggende). For begge situationer kan andre forhold end de sundhedsmæssige spille ind, såsom baggrundsniveauer og tekniske/økonomiske overvejelser. Ved indsatsen i forhold til eksisterende forureninger kan der også være behov for at

vurdere, hvad der er sundhedsmæssigt forsvarligt, når der tages hensyn til overordnede samfundsmæssige prioriteringer. Med udgangspunkt i de sundhedsmæssigt fastsatte kvalitetskriterier udarbejdes således en række administrativt fastsatte kriterier.

Eksempler på administrative kriterier som er baseret på kvalitetskriterier er:

1. Luftkvalitetskriteriet anvendes som udgangspunkt til at fastsætte bidrags værdier (B-værdier), der anvendes i forbindelse med regulering af virksomheders udslip af kemiske stoffer til udeluften.
2. Luftkvalitetskriteriet anvendes til at vurdere afdampning af kemiske stoffer fra jordforureninger. Det anvendes også ved kortlægning og offentlig oprydning af forurenede grunde.
3. Jordkvalitetskriterier anvendes som udgangspunkt ved fastsættelse af administrative kriterier, der anvendes ved de lokale myndigheders kortlægning af forurenede grunde og i forbindelse med vurderinger af arealanvendelse, samt ved offentlig oprydning af forurenede grunde[4].
4. Jordkvalitetskriteriet anvendes endvidere som udgangspunktet for fastsættelse af afskæringskriteriet for visse immobile stoffer. Afskæringskriteriet anvendes ved lettere forurenede områder som skillelinie mellem det niveau, hvor det er nødvendigt at fjerne jorden og det niveau, hvor det er tilstrækkeligt at iværksætte særlige forholdsregler i forbindelse med følsom anvendelse af områderne, som fx private haver eller børneinstitutioner.
5. Drikkevandskvalitetskriteriet anvendes af de centrale og decentrale myndigheder i forbindelse med håndtering af konkrete sager med kemisk forurening af drikkevand, som et supplement til de kravværdier, der er fastsat i bekendtgørelsen om vandkvalitet.
6. Drikkevandskvalitetskriterierne anvendes som udgangspunkt ved fastsættelse af grundvandskvalitetskriterier i tilknytning til nedsivning fra jordforureninger,

[4] Kvalitetskriterierne omfatter normalt ikke en vurdering af om arealer kan anvendes til dyrkning af nytteplanter eller til afgræsning for husdyr.

idet grundvandet skal være af en kvalitet, så de fastsatte drikkevandskrav/ -kvalitetskriterier kan overholdes.

Der eksisterer en række vejledninger og lister, som nærmere beskriver baggrunden for, hvordan et kvalitetskriterium kan anvendes.[5]

1.1.2 Generelle principper for fastsættelse af kvalitetskriterier for kemikalier

Kvalitetskriterierne fastsættes på et niveau, hvor udsættelse gennem et helt liv ikke fører til skadevirkninger i befolkningen. De fastsættes på baggrund af den eksisterende viden og under hensyntagen til de mangler, der ligger i datagrundlaget.

For at minimere risici for skadelig påvirkning af befolkningen indgår beskyttelse af særligt følsomme grupper fx børn, gravide, syge, ældre og svækkede ved fastsættelse af kvalitetskriterier.

Viden om et kemisk stofs sundhedsskadelige egenskaber og om bestemte befolkningsgruppers særlige følsomhed er sjældent så eksakt, at der kan fastsættes et kvalitetskriterium, der præcist definerer skillelinien (hvis en sådan overhovedet findes) mellem et ufarligt og farligt niveau. Kvalitetskriterierne kan således ikke opfattes som en streg i sandet, hvor enhver overskridelse er farlig. Ved fastsættelse af kvalitetskriterier for kemikalier skal anvendes en forsigtighedstilgang, da målet er at sikre et højt beskyttelsesniveau for alle ved udsættelse over et helt liv.

Et kvalitetskriterium skal således opfattes som en sikkerhedsgrænse og ikke en faregrænse. En overskridelse er "det gule lys", som advarer om, at her er noget, som måske kan blive et reelt problem.

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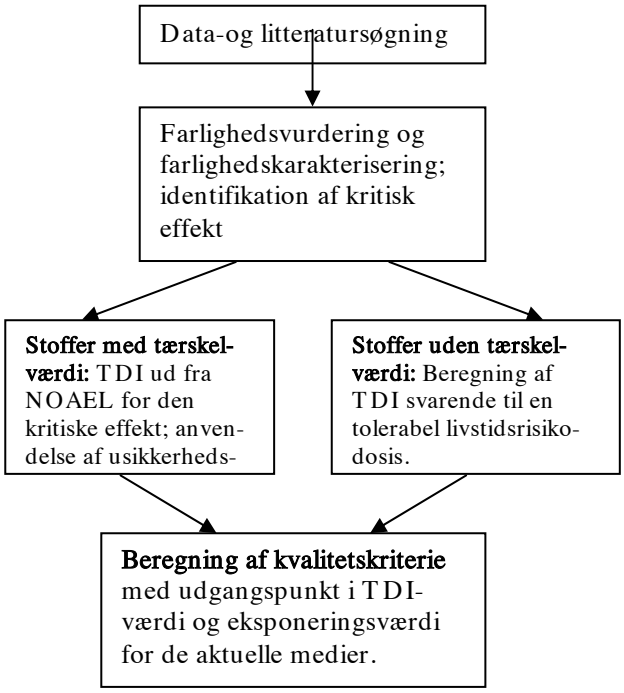
[5] Luftvejledningen, Miljøstyrelsens Vejledning nr. 2, 2001; Oprydning på forurenede lokaliteter

1.2 Vejledningens opbygning og indhold

Vejledningen udstikker retningslinier for den faglige risikovurdering og for de metoder/ principper der anvendes. Dernæst beskrives proceduren for beregning af kvalitetskriterierne (se figur 1).



Figur 1.
Fremgangsmåden ved beregning af kvalitetskriterium (TDI=Tolerabel Dagligt Indtag)

Det videnskabelige grundlag for fastsættelse af sundhedsmæssigt baserede kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand består af en farlighedsvurdering, en dosis-respons (effekt) vurdering (farlighedskarakterisering), samt en eksponeringsvurdering. Farlighedsvurderingen og farlighedskarakteriseringen tager udgangspunkt i undersøgelser af det pågældende stofs toksikologiske effekter i mennesker og i dyr.

I kapitel 2 omtales *Datagrundlaget* som anvendes som udgangspunkt for arbejdet. Data hentes primært fra internationale og nationale dokumenter, via litteratursøgning i internationale databaser, samt fra originalartikler.

Kapitel 3 behandler de faglige metoder, der anvendes i forbindelse med farlighedsvurderingen og farlighedskarakteriseringen. Dosis-effekt og dosis-responssammenhænge og udpegning af NOAEL (No Observed Adverse Effect Level) og LOAEL (Lowest Observed Adverse Effect Level) beskrives.

I kapitel 4 beskrives hvordan farlighedskarakteriseringen udmunder i udpegning af en kritisk effekt, som danner udgangspunkt for fastsættelse af en *tolerabel daglig indtagelse, TDI*. Her omtales, hvordan anvendelsen af usikkerhedsfaktorer indgår i beregningerne.

I kapitel 5 omtales, hvordan TDI beregnes for kræftfremkaldende stoffer uden tærskelværdi. Risikoniveauet for TDI-værdien defineres, og der gives retningslinier med hensyn til valg af metode til, hvordan beregningen af dette risikoniveau foretages.

Kapitel 6 beskriver, hvordan den videre beregning af kvalitetskriterier for kemiske stoffer i jord, luft og drikkevand foretages ud fra de fastsatte TDI-værdier. I bilag 2 til vejledningen omtales rationalet for at anvende konkrete standardbetragtninger m.h.t. personers udsættelse for forureninger gennem jord, luft og drikkevand. Endvidere omtales hvilke andre faktorer (fx hensyntagen til lugt eller smag) end de rent sundhedsmæssige, der i visse tilfælde kan have indflydelse på kvalitetskriteriet.

1.3 Ændringer i forhold til tidligere praksis

Ved fastsættelsen af kvalitetskriterier anvendes der internationalt anerkendte principper. I denne forbindelse skal fremhæves de principper og metoder, der er beskrevet i to publikationer af WHO/ IPCS[6,7] om udarbejdelsen af vejledende grænseværdier og risikovurdering af kemisk udsættelse. WHO´s publikationer[8,9] omhandlende risikovurdering og fastsættelse af vejledende grænseværdier for en række kemiske stoffer i drikkevand og luft har også indgået i arbejdet. På tilsvarende vis er der en tæt sammenhæng med de principper og metoder, der anvendes i forbindelse med EU´s risikovurderingsprogram for kemiske stoffer[10].

På visse områder indebærer denne vejledning, at den hidtidige praksis for fastsættelse af kvalitetskriterier justeres. Dette gælder fx for beregning af kvalitetskriterier for kemikalier i luft og drikkevand, idet der nu som udgangspunkt anvendes standardværdier for børns udsættelse (se kapitel 6 og bilag 2).

I forbindelse med ekstrapolering af tolerabelt risikoniveau for genotoksiske kræftfremkaldende stoffer (dvs. kræftfremkaldende stoffer hvor der ikke anses at være en tærskelværdi for effekt), anvendes lineær ekstrapolation ud fra den så-

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[6] WHO/IPCS (1994). Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. Environmental Health Criteria no. 170. International Programme on Chemical Safety.

[7] WHO/IPCS (1999). Principals for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria no. 210. International Programme on Chemical Safety.

[8] WHO (1996). Guidelines for drinking-water quality 2nd edition, vol 2. Health Criteria and other supporting information. International Programme on Chemical Safety.

[9] WHO (2000). Air Quality Guidelines for Europe, 2nd edition. WHO Regional Publications, European Series, no. 91.

[10] EEC (2003). Technical Guidance Document in support of Commission Directive 93/67/EEC on risk assessment for new notified substances and Commission Regulation (EC) No. 1488/94 on risk assessment for existing substances and Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market.

kaldte T25- metode udviklet i forbindelse med EU´s risikovurderingsprogram (kapitel 5).

Hvis datagrundlaget er til stede, anbefales det at anvende benchmark-metoden ved fastsættelse af TDI (kapitel 3).

Det skal bemærkes, at denne vejledning anvender begrebet usikkerhedsfaktorer, hvor man tidligere anvendte begrebet sikkerhedsfaktorer. Dels beskriver ordet *usikkerhed* i højere grad faktorernes anvendelse, idet de netop tager højde for usikkerheder. Dels imødegås den misforståelse, at en stor sikkerhedsfaktor for et stof medfører større sikkerhed for dette stof i forhold til en lille anvendt sikkerhedsfaktor for et andet stof. Faktorerne anvendes derimod for at opnå et ensartet beskyttelsesniveau, og størrelsen af faktorerne afspejler usikkerheder som følge af usikker viden og manglende datagrundlag.

De angivne metoder i vejledningen anvendes fremover ved fastsættelse af kvalitetskriterier.

De ændringer der er sket i forhold til tidligere praksis betyder ikke, at de eksisterende kvalitetskriterier skal "laves om", idet anvendelse af de nye metoder ikke vurderes at påvirke beskyttesesniveauet væsentligt. Når et kvalitetskriterium tages op til revurdering bør dette ske både m.h.t. til revurdering af de sundhedsmæssige data og af de eksponeringsmæssige forhold. En revurdering foretages derfor fuldt ud med alle de vurderingsmæssige og beregningsmæssige faser, der er beskrevet i vejledningen.

Referencer

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Miljøstyrelsen (1991). Orientering om ny miljøbeskyttelseslov. Orientering nr. 6, 1991.

2 Datagrundlag for vurdering af farlighed

Fastsættelse af kvalitetskriterier for et kemisk stof sker på baggrund af eksisterende viden om stoffets sundhedsskadelige egenskaber.

Kvalitetskriterierne fastsættes med henblik på beskyttelse af menneskers sundhed, og det ideelle datagrundlag for fastsættelse af et kvalitetskriterium er derfor viden, hvor mennesker har været udsat for det konkrete stof. For langt de fleste kemiske stoffer er der kun begrænset viden om veldefineret udsættelse og effekter hos mennesker, og kvalitetetskriterierne vil som oftest være baseret på viden opnået fra dyreforsøg med mere veldefineret udsættelse eller *in vitro* data[11].

For en mere detaljeret beskrivelse af nedenstående afsnit henvises til kapitel 2, Miljøprojekt Nr. 974 (2005).

2.1 Data fra mennesker

Fordelen ved at anvende data, hvor mennesker har været udsat for et kemisk stof, er, at man undgår at skulle overføre data fra dyreforsøg, og estimere hvad en tilsvarende udsættelse betyder hos mennesker. Erfaringer med menneskers udsættelse kan stamme fra en række forskellige typer undersøgelser og afrapporteringer, der groft kan deles op på følgende måde:

- *case reports og kliniske undersøgelser*
- *befolkningsundersøgelser (fx arbejdsmiljø eller udvalgte dele af befolkningen)*
- *undersøgelser af frivillige forsøgspersoner*

[11] Data fra reagensglasforsøg.

Ved *case reports og kliniske undersøgelser* opnås data fra forgiftningstilfælde eller erfaringer fra undersøgelser i klinikken, hvor personer fx i behandlingsøjemed udsættes for stoffer for at vurdere eventuelle allergiske reaktioner. Fordelen ved disse typer data er, at man her umiddelbart kan se en sammenhæng mellem udsættelse og (akutte) effekter.

I *befolkningsundersøgelser* vurderes en større gruppe af mennesker mere systematisk med hensyn til sammenhængen mellem udsættelse og sundhedsskader. Disse undersøgelser kan fx omfatte sundhedsovervågning af særlige grupper i arbejdsmiljøet, hvor personerne er karakteriseret ved en særlig udsættelse, eller man kan undersøge, om der hos personer, der har udviklet nogle konkrete sygdomme, er nogle fælles karakteristika med hensyn til kemiske påvirkninger. Fordelen ved at anvende data fra denne type undersøgelser er, at man er meget tæt på den målgruppe, man ønsker at beskytte med kvalitetskriterierne. Samtidig ses effekterne i sammenhæng med den dagligdag, som mennesker nu engang fungerer i, hvor man udsættes for en kompleks blanding af livsstilsfaktorer og miljøfaktorer. Ulempen er, at det ofte er vanskeligt at vurdere omfanget af udsættelsen af en given komponent, og at det pga. mange andre samvirkende faktorer kan være svært at påvise sammenhænge, som kan være sløret af al "støjen" fra andre faktorer. Ligesom undersøgelserne kun sjældent kan dokumentere en årsagsvirkningssammenhæng, vil de også kun uhyre sjældent kunne anvendes til at dokumentere manglende sammenhæng, dvs. frikende stoffer.

I *undersøgelser med frivillige forsøgspersoner* udsætter man i reglen forsøgspersonerne i en kortere varighed for et konkret stof for at vurdere effekterne. Disse undersøgelser er meget sammenlignelige med dyreforsøg, hvor man tilsvarende har en meget veldefineret udsættelse. De naturlige og etiske begrænsninger ved disse undersøgelser betyder, at der hos mennesker kun kan undersøges for lettere grader af akutte effekter og i sammenhæng med kortere tids forsøgsudsættelse. Endvidere er forsøgspersonerne sjældent særligt følsomme, som visse undergrupper i befolkningen kan være.

Man skal være meget opmærksom på det etiske aspekt m.h.t. anvendelse af frivillige forsøgspersoner. I forbindelse

med vurdering af konkrete humane undersøgelser i risikovurderingssammenhæng, bør anvendelsen af disse data vurderes nøje såvel ud fra etiske som kvalitetsmæssige hensyn. Især ældre undersøgelser kan være udført under stærkt kritisable forhold og med store undersøgelsesmæssige mangler.

Kvalitetskriterier for kemikalier fastsættes ud fra eksisterende data, og det frarådes generelt at igangsætte humanforsøg for at opnå øget viden om konkrete stoffers skadelige effekter.

2.2 Dyreeksperimentelle undersøgelser

For de fleste kemiske stoffer foreligger der ikke data fra menneskers udsættelse, hvorfor kvalitetskriterier hyppigst baseres på data fra dyreeksperimentelle undersøgelser. Resultaterne fra dyreforsøgene anvendes således som model og anvendes til at forudsige hvilke effekter, der kan forventes hos mennesker. Dyreeksperimentelle data kan også benyttes som supplement til humane data, der ikke er entydige, eller til at udpege de aktive stoffer, når mennesker har været udsat og reageret over for blandinger af stoffer.

Fordelene ved dyreforsøg er, at der er tale om standardiserede forsøgsbetingelser, og at der er mulighed for at afsløre væsentlig flere effekter hos forsøgsdyr end hos mennesker, da organer og væv kan undersøges efter forsøgets afslutning. Der er også mulighed for at undersøge virkningsmekanismer og detaljerede dosis-effekt og dosis-responssammenhænge for enkeltstoffer.

Ideelt set ønskes der ved fastsættelsen af kvalitetskriterier for et kemikalie et fuldt datasæt bestående af dyreeksperimentelle undersøgelser til vurdering af en række toksikologiske egenskaber: Toksikokinetik (optagelse og udskillelse), akut toksicitet, irritation, sensibilisering (allergi), toksicitet ved gentagen administration af stoffet, mutagenicitet og genotoksicitet (påvirkning af arvematerialet), kræftfremkaldende effekter, samt effekter på reproduktion og fosterudvikling.

Dyreforsøg har imidlertid også begrænsninger, da nogle effekter kan være vanskelige at afsløre fx lettere grader af slimhindeirritation i øjne og luftveje, lettere grader af påvirkning af centralnervesystemet og visse typer nerveskader.

Endelig kan nogle dyrearter udvikle artspecifikke effekter over for visse stoffer, hvor relevansen i forhold til udsættelse af mennesker er meget omdiskuteret (se afsnit 3).

Det er endvidere vigtigt at vurdere kvaliteten af de dyreeksperimentelle undersøgelser, der anvendes som udgangspunkt for fastsættelse af kvalitetskriterier. Undersøgelser af høj kvalitet, som er udført efter eller på niveau med OECD´s eller EU´s retningslinier for forsøgsdyrstestning, bør foretrækkes. Ved risikovurderingen i forbindelse med fastsættelse af kvalitetskriterier er der dog ikke nogle formelle kvalitetskrav til undersøgelserne, idet en lang række undersøgelser i den videnskabelige litteratur ofte vil være udført i forskningsøjemed uden at undersøgelserne er udført efter en officiel forsøgsguideline eller i overensstemmelse med GLP-reglerne.[12] Sådanne undersøgelser, der ofte er kvalitetssikret i peer-reviewede tidsskrifter, kan indeholde væsentlig information. Forsøgets kvalitet og validitet må i de aktuelle tilfælde vurderes, og der tages stilling til om forsøget kan være af betydning ved fastsættelse af et kvalitetskriterium.

På baggrund af de etiske aspekter ved dyreforsøg arbejdes der internationalt med at udvikle alternative *in vitro* metoder.

2.3 Andre typer data

Ud over forsøg på levende dyr foreligger der ofte undersøgelser der er udført på udtagne organer, væv eller isolerede celler. Disse *in vitro* metoder finder især anvendelse til vurdering af stoffers toksiske effekt på organ-/celleniveau særligt m.h.t. mutagene og genotoksiske effekter.

[12] GLP står for Good Laboratory Practice, og er et regelsæt udviklet af OECD for at sikre kvaliteteten af undersøgelserne.

Normalt kan *in vitro* forsøg ikke anvendes til at fastlægge tærskel for effekt (NOAEL e.l.), men de vil indgå i bedømmelsen af stoffets farlighed. In vitro-data kan således styrke mistanken om skadeeffekter og derved påvirke valget af usikkerhedsfaktorer (se afsnit 4).

Ved mangel på data for et konkret stof kan data fra nært beslægtede stoffer indgå i vurderingen ud fra betragtninger om kemisk strukturlighed og sammenfaldende effekter. Sådanne data kan have indflydelse på den rent kvalitative, men også i visse tilfælde i den kvantitative vurdering, hvis det konkret vurderes, at der er grundlag for meget snævert koblede analogislutninger.

Endvidere kan der udføres en mere systematisk analyse vedrørende kvantitative struktur-aktivitets relationer (engelsk: Quantitative Structure Activity Relationships – QSARs). Anvendelse af denne form for computerbaserede modeller har i en række tilfælde vist sig som et alternativ til dyreforsøg med henblik på forudsigelse af toksikologiske egenskaber (MST 2001[13]). Vurderingerne kan sjældent anvendes som udgangspunkt for beregning af kvalitetskriteriet, men vil i visse tilfælde kunne anvendes til styrkelse af mistanken om konkrete effekter. QSAR vil således kunne påvirke valget af usikkerhedsfaktorer ved beregning af kvalitetskriteriet.

2.4 Indhentning af data

Som udgangspunkt for udarbejdelse af kvalitetskriterier anvendes i udstrakt grad internationalt anerkendte stofmonografier og dokumenter, hvor det aktuelle stof er vurderet og beskrevet m.h.t. dets sundhedsskadelige effekter. En række af disse værker (kilder) er nævnt i afsnit 2.5.1 i Miljøprojekt Nr. 974 (2005) . Sådanne dokumenter, der ofte er baseret på en grundig faglig vurdering ved en særlig nedsat ekspertgruppe, vil typisk kunne anvendes til at udpege de relevante undersøgelser og data, der skal anvendes til den videre beregning af kvalitetetskriteriet. Det anbefales gene-

[13] Miljøstyrelsen (2001). Report on the advisory list for selfclassification of dangerous substances. Environmental Project No. 636 2001. http://www.mst.dk/udgiv/publications/2001/87-7944-694-9/html/

relt, at data, der skal danne udgangspunkt for beregning af kvalitetskriteriet, altid hjemskaffes som originallitteratur til vurdering af den konkrete undersøgelses kvalitet og relevans.

Endvidere søges i en række relevante databaser (se afsnit 2.5.2 i den faglige rapport). Sådanne søgninger er især relevante, hvis en international stofmonografi for det konkrete stof ikke foreligger, eller hvis vurderingen ligger nogle år tilbage.
For visse stoffer kan man komme ud for at datasøgning giver så ringe resultat, at der ikke er tilstrækkeligt datagrundlag til at foretage en vurdering af stoffet. For eksempel hvis der kun er akutte studier til rådighed, eller ingen studier fastlægger NO(A)EL/LO(A)EL.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier i jord, luft og drikkevand.

3 Farlighedsvurdering og farlighedskarakterisering

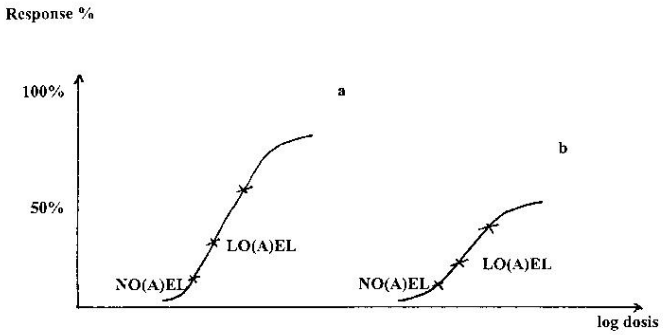
Farlighedsvurderingen og farlighedskarakteriseringen har til formål at beskrive stoffets farlige egenskaber. Der opstilles i videst muligt omfang dosis-effekt og dosis-respons sammenhænge, der danner baggrund for identifikation af den kritiske effekt. Den kritiske effekt er den effekt, der anses for at være den afgørende ved den sundhedsmæssige vurdering. Nuleffekt niveauet (evt. laveste effektniveau) for denne effekt anvendes til beregning af TDI (tolerabelt dagligt indtag/ tolerabelt daglig eksponering), som i den videre proces benyttes til beregning af kvalitetskriteriet.

3.1 Dosis-effekt og dosis-respons sammenhænge

Udsættelse for et kemisk stof kan medføre forskellige typer effekter afhængig af eksponeringsvej, eksponeringens størrelse og varighed. Lettere grader af effekter kan være forbigående genevirkninger i form af fx slimhindeirritation, mens alvorlige effekter kan være dødeligt forløbende akutte forgiftninger eller udvikling af kroniske sygdomme som kræft.

En længerevarende dyreeksperimentel undersøgelse vil således kunne give viden om forskellige typer effekter ved forskellige eksponeringsniveauer (dosis-effekt), og om hvor lang tid det tager, før de optræder i forhold til eksponeringen.

Yderligere vil man fra undersøgelsen kunne se, hvor stor en andel af de doserede dyr der er ramt af effekten (dosis-respons). Visse effekter kan være opdelt i forskellige sværhedsgrader eller stadier, hvor der så for hver af disse kan foreligge dosis-responssammenhænge.



Figur 1 Eksempler på dosisrespons-sammenhænge

Ovenstående figur viser, hvordan dosisrepons-kurver for forskellige effekter kan optegnes ud fra konkrete forsøgsdata med forskellige eksponeringsniveauer og observerede effekter ved disse (markeret med krydser). Kurve a angiver fx dosis-respons forløbet for luftvejsirritation hos forsøgsdyrene, og er her karakteriseret ved at være meget stejl (alle dyr påvirkes inden for et forholdsvist lille dosisinterval, dvs. lille spredning i følsomhed). Kurve b angiver forekomsten af kræftsvulster og viser en dosisrespons sammenhæng ved højere eksponeringsniveauer, og beskriver en noget fladere dosis-respons sammenhæng (dvs. dyrenes følsomhed over for udvikling af kræft er mere spredt). NOAEL-markeringerne på figuren repræsenterer eksponeringsniveauer, hvor der ikke blev fundet statistisk signifikant flere dyr i eksponeringsgruppen end i kontrolgruppen, der udvikler den pågældende effekt, mens LOAEL-værdierne repræsenterer det laveste eksponeringsniveau, der har medført en signifikant forøget forekomst af effekten.

Den faglige baggrundsrapport (Miljøprojekt Nr. 974 (2005)) angiver i afsnit 3.1 mere detaljerede beskrivelser og tolkningen af forskellige dosis-respons forløb.

3.2 Fastsættelse af nul-effektniveau og laveste effektniveau

For langt de fleste typer effekter vurderes der at være en tærskelværdi, der adskiller effektniveauer fra ikke-

effektniveauer, dvs. eksponeringen skal overskride en vis tærskelværdi før dosis er tilstrækkelig til at udløse effekt. Niveauet umiddelbart under denne teoretiske tærskelværdi betegnes nuleffektniveauet, der er den højeste dosis, der ikke medfører effekt. Dosisniveauet umiddelbart over tærskelværdien betegnes laveste effektniveau, da dette er det laveste dosisniveau, der lige netop udløser effekten.

I praksis anvendes for nuleffektniveauet det såkaldte *no observed adverse effect level*, *NOAEL*, der er den højeste af de i forsøget anvendte doser, hvor der i et konkret forsøg ikke er *observeret* den givne effekt. For laveste effekt-niveau anvendes det såkaldte *lowest observed adverse effect level, LOAEL*, der er den laveste dosis i forsøget hvor der er observeret den givne effekt.

I litteraturen anvendes betegnelserne NOAEL og NOEL (*no observed effect level)* samt LOAEL og LOEL (*lowest observed effect level).* Når betegnelserne anvendes korrekt, er det for at sondre mellem om de effektniveauer eller nuleffektniveauer der beskrives, er i forhold til *skadelige (adverse)* effekter eller effekter generelt, hvor også lettere grader af effekter som påvirkning af enzymniveauer og andre effekter af ikke direkte skadelig karakter er omfattet.

Man må dog være opmærksom på, at denne skelnen mellem skadelige og ikke-skadelige effekter i mange tilfælde ikke er gjort konsekvent i litteraturen, og at betegnelserne ofte benyttes i flæng. Det kan således i konkrete tilfælde være vanskeligt at afgøre, hvor grænsen går for, om en effekt skal tolkes som skadelig eller ej, og dermed om et LOEL snarere skal tolkes som et LOAEL.

Se endvidere afsnit 3.2 i den faglige rapport (1).

3.3 Benchmark-metoden

NO(A)EL/ LO(A)EL metoden til udpegning af et eksponeringsniveau er afhængig af, hvilke eksponeringsniveauer man har valgt ved udførelse af et forsøg, og i nogle tilfælde har man måske ikke engang fundet et NO(A)EL. Dette har medført, at en nyere metode, benchmark-metoden, visse

steder har vundet indpas i forbindelse med risikovurderinger, idet der ikke anvendes en NO(A)EL eller LO(A)EL men en benchmark-dosis for den videre beregning af TDI. Ved denne metode foretages en computerbaseret modellering af dosis-responskurven ud fra de tilgængelige data. På dosis-respons kurven findes ED_5 – eller ED_{10}-niveauet (dvs. det eksponeringsniveau, der medfører respons hos 5% eller 10% af de eksponerede). Denne dosis vælges derpå som udgangspunkt (benchmark-dosis, BMD_5 eller BMD_{10}) for den videre beregning på tilsvarende måde som man anvender en NO(A)EL- eller LO(A)EL-værdi.

Metoden anvendes af de canadiske miljømyndigheder og af US EPA, og også af WHO i forbindelse med "Air quality Guidelines for Europe" (2). WHO angiver i denne forbindelse, at en BMD_5 ud fra en gennemsnitsbetragtning kan sammenlignes med et NO(A)EL, mens en BMD_{10} kan sammenlignes med et LO(A)EL.

WHO omtaler også anvendelsen af benchmark-metoden som en alternativ metode i forbindelse med publikation om risikovurdering fra 1999 (3) og i forbindelse med publikation om fastsættelse af vejledende grænseværdier fra 1994 (4).

På nuværende tidspunkt haves der kun sparsom erfaring med anvendelsen af metoden herhjemme. Som det faglige miljøprojekt anfører, kan benchmark-metoden ikke umiddelbart anvendes for alle typer data, og metoden kræver for de relevante effektområder, hvor den kan anvendes, ofte flere dosis-niveauer end der sædvanligvis haves.

Benchmark-metoden er, når der foreligger tilstrækkelige data, et supplement til den traditionelle NO(A)EL/ LO(A)EL metode i forbindelse med fastsættelse af kvalitetskriterier. Uanset hvilken metode man vælger, bør man vurdere og begrunde den fra gang til gang, dvs. man bør referere til og begrunde de anvendte beregningsmetoder eller henvise til de originalreferencer, hvor den benyttede benchmark-dosis er beregnet.

3.4 Udpegning af kritisk effekt

Ved den samlede vurdering af dosis-effekt og dosis-respons sammenhænge foretages udpegning af den kritiske effekt samt udpegning af den NO(A)EL/ LO(A)EL værdi (evt. benchmarkdosis), der vurderes som mest relevant ved fastsættelse af kvalitetskriteriet. Ofte, men ikke nødvendigvis, vil dette være den laveste NO(A)EL/ LO(A)EL værdi, der er rapporteret vedrørende en skadelig effekt eller potentielt skadelig effekt.

Ved udpegning af den kritiske effekt vurderes relevansen af eksponeringsmåden (eksponeringsvejen i en given undersøgelse) i forhold til eksponeringsvejen for det medie (jord, luft eller vand) kvalitetskriteriet skal udarbejdes for. Alvorligheden af forskellige effekter sammenholdes i forhold til, hvor langt de ligger fra hinanden i eksponeringsniveau. Det vurderes fx om en alvorlig effekt kan være en følge af mindre alvorlige effekter, som således kan ses som en forløber og indikator for udvikling af en egentlig skadeeffekt. Endvidere vurderes i det konkrete tilfælde, om der optræder visse artsspecifikke effekter hos forsøgsdyrene, og om dokumentationen er tilstrækkelig til, at disse fund kan vurderes at være irrelevante i forhold til mennesker (se afsnit 3.5).

Ved tvivlstilfælde om udpegning af kritisk effekt og NO(A)EL/LO(A)EL værdi kan det være nødvendigt at udpege flere NO(A)ELs/LO(A)ELs for de potentielt kritiske effekter og anvende disse sideløbende ved den efterfølgende beregning af TDI/ kvalitetskriterier. Dette er for at vurdere, hvordan anvendelse af usikkerhedsfaktorer og andre beregningsmæssige forhold får indflydelse på kvalitetskriteriet, når der anvendes forskellige udgangspunkter m.h.t. kritisk effekt og NO(A)EL/LO(A)EL.

3.5 Relevans af visse effekter i forsøgsdyr

Som udgangspunkt anvendes dyremodeller som troværdige modeller ved forudsigelse af stoffets egenskaber hos mennesker. Visse effekter, der optræder i forsøgsdyr, har vist sig at være tæt knyttet til bestemte dyrearter. I tilfælde, hvor der foreligger dokumentation for at effekterne alene er forårsa-

get af en sådan artsspecifik virkningsmåde, anses disse for at være af meget begrænset relevans ved den videre vurdering af stoffet.

Især inden for kræftområdet har man fundet flere typer svulster (tumorer), hvor relevansen i forhold til mennesker i de enkelte tilfælde bør vurderes nøjere. I Miljøprojekt Nr. 974 (2005) er en række af disse beskrevet i afsnit 3.7.

Referencer

(1) Miljøprojekt Nr. 974 (2005). Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

(2) WHO (2000). Air Quality Guidelines for Europe. WHO regional Publications, European Series, No. 91.

(3) WHO (1999). Principles for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria 210. International Programme on Chemical Safety.

(4) WHO (1994). Assessing human health risks of chemicals: derivation of guidance values for health-based exposure limits. Environmental Health Criteria 170. International Programme on Chemical Safety.

4 Beregning af TDI for stoffer med tærskelværdi

4.1 TDI-begrebet

Det sidste trin i farlighedskarakteriseringen er beregning af tolerabelt dagligt indtag, *TDI (*eller tolerabel koncentration, *TK)*. Ved beregning for stoffer, hvor der anses at være en nedre tærskel for effekt anvendes usikkerhedsfaktorer:

$$TDI\,(TK) = \frac{NO(A)EL^{a}}{UF_{I} \times UF_{II} \times UF_{III}}$$

[a] *Alternativt kan et LO(A)EL eller en Benchmark-dosis* BMD_{x} *anvendes, se afsnit 3.3.*

Den tolerable daglige indtagelse (TDI) er et udtryk for den daglige gennemsnitsdosis (fra alle kilder), som befolkningen vurderes at kunne udsættes for (tolerere) gennem et helt livsforløb, uden at der forventes at opstå sundhedsskadelige effekter.
TDI angives sædvanligvis i enheden mg/kg legemsvægt per dag.

Analogt til TDI kan betegnelsen tolerabel koncentration (TK) defineres som den koncentration af et stof i jord, luft eller drikkevand som befolkningen vurderes at kunne udsættes for (tolerere) gennem et helt livsforløb, uden at der forventes at opstå sundhedsskadelige effekter. TK angives fx i enheden mg/m^3 (luft), mg/l (drikkevand), eller i mg/kg (jord).

For nogle stoffer er det nødvendigt at fastsætte en PTWI – værdi (Provisional Tolerable Weekly Intake – PTWI), der angiver det tolerable ugentlige indtag, i stedet for TDI. PTWI benyttes sædvanligvis over for stoffer, hvor det er

vigtigt at understrege, at det er den samlede eksponering over længere tid, der er af betydning for forekomst af effekter (fx bly og cadmium).

Begrundelse for anvendelse af usikkerhedsfaktorer omtales kort nedenfor, idet en mere detaljeret beskrivelse er givet i Miljøprojekt Nr. 974 (2005) afsnit 4.4

4.2 Anvendelse af usikkerhedsfaktorer

I kommentarerne til Miljøbeskyttelsesloven angives at "Miljøministeriet ved udstedelse af regler og vejledninger kan operere med fx sikkerhedsfaktorer ved fastsættelse af grænseværdier eller retningslinier for forureningsmæssige beregninger på de områder, hvor der ikke foreligger et tilstrækkeligt eksakt vidensgrundlag"

Tidligere har man anvendt begrebet sikkerhedsfaktorer. Det har imidlertid vist sig, at denne betegnelse kan misforstås, således at man forventer større sikkerhed, jo større sikkerhedsfaktor der anvendes. Dette er ikke i overensstemmelse med, hvordan faktorerne anvendes, idet de benyttes for at tage hensyn til usikkerheder og manglende viden i datagrundlaget, dvs. jo større usikkerhed jo større faktor. Ved brugen af disse faktorer tilstræbes det, at der opnås et ensartet beskyttelsesniveau udtrykt ved TDI-værdien. På denne baggrund er det derfor mere korrekt at anvende betegnelsen usikkerhedsfaktorer.

Også i udenlandsk litteratur anvendes forskellige termer for denne faktor fx: safety factor, uncertainty factor, Sicherheitsfaktor, assessment factor, bedömningsfaktor.

Nedenfor angives hvilke elementer og hensyn, der indgår i de tre usikkerhedsfaktorer.

4.2.1 Usikkerhedsfaktor I

Usikkerhedsfaktor I (UF_I) anvendes for at tage højde for, at mennesker kan være mere følsomme over for et givent stof end forsøgsdyr. Denne faktor har historisk været sat til 10.

Ekstrapolation af data fra dyr til mennesker kan opfattes som omhandlende to forskellige aspekter:

1) korrektion af dosis for forskelle i kropsstørrelse mellem forsøgsdyr og mennesker, såkaldt allometrisk skalering, og
2) andre former for forskelle mellem forsøgsdyr og mennesker, som ikke nødvendigvis afspejles i forskellene i kropsstørrelse.

Miljøprojekt Nr. 974 (2005) har gennemgået den eksisterende viden og understøtter, at der fortsat som udgangspunkt anvendes en standardværdi på 10 for UF_I, hvilket også er i overensstemmelse med international praksis, når dosis hos dyr omsættes til human dosis med samme enhed.

Hvis der foreligger veldokumenteret viden om toksikokinetiske og/eller toksikodynamiske forskelle mellem det givne forsøgsdyr og mennesker, anbefales det at tage udgangspunkt i denne viden med henblik på fastsættelse af en dataspecifik faktor i stedet for anvendelse af en standardværdi på 10. I den forbindelse kan 10-faktoren evt. opdeles i underfaktorer, hvor størrelsen af disse "delfaktorer" må vurderes konkret fra gang til gang.

Ved beregning af tolerabel koncentration (TK), hvor udgangspunktet er dyreforsøg, hvor eksponeringen foregår via inhalation, bør ekstrapolering til humaneksponering ved inhalation foretages direkte ud fra eksponeringsniveauet angivet i mg stof /m^3 (omregnet til kontinuerlig gennemsnitskoncentration per dag), frem for at foretage omregning fra indåndet dosis til mg/kg lgv/d for forsøgsdyrene. Ved at anvende indåndingskoncentrationen direkte svarer dette til at doseringen foretages i forhold til stofskiftet, hvilket anses for at være den mest relevante metode, når der skal korrigeres for forskelle i kropsstørrelse. Anvendelse af en UF_I skal ses i lyset heraf og bør derfor i denne situation ligge lavere end 10. En værdi på en halv tierpotens ($10^{0,5}$) kan som udgangspunkt anvendes til resterende forskelle m.h.t. artsforskelle i kinetik og dynamik, medmindre konkrete data tilsiger højere eller lavere værdi.

Der bør i videst muligt omfang tages hensyn til en række mætningsfænomener (fx mætning af absorption, metabolisme eller udskillelse), der kan forekomme i dyreforsøg ved høj dosering, idet disse mætningsfænomener ofte ikke vil være relevante ved lavere miljømæssigt relevante niveauer. I sådanne tilfælde bør der om muligt omregnes til "effektiv" dosis/ koncentration som dyrene har været udsat for.

4.2.2 Usikkerhedsfaktor II

Usikkerhedsfaktor II (UF_{II}) anvendes for at tage højde for, at nogle individer i befolkningen kan være mere følsomme over for et givent stof end den generelle befolkning (for eksempel børn, gravide, ældre, svækkede, kronisk syge). Denne faktor har oftest været sat til 10. Forskellene i følsomhed skyldes den biologiske variation, der findes mellem mennesker. Faktorer som alder, køn, graviditet, genotype, helbred, og livsstil kan være medvirkende til en øget biologisk følsomhed, som afspejler dels forskelle i toksikokinetik og dels i toksikodynamik.

Miljøprojekt Nr. 974 (2005) henviser til en række analyser, der har vurderet variationen mellem mennesker og dermed størrelsen af UF_{II}. Sammenfattende understøtter disse analyser anvendelse af en standardværdi på 10 for denne variation.

Denne usikkerhedsfaktor skal derfor som udgangpunkt sættes til 10. En alternativ værdi kan anvendes, hvis udgangspunktet for NO(A)EL/LO/A)EL værdien specifikt er relateret til data for særligt følsomme personer eller den kritiske effekt er en effekt, hvor man har særligt kendskab til variationsbredden i følsomhed.

4.2.3 Usikkerhedsfaktor III

Usikkerhedsfaktor III (UF_{III}) anvendes for at tage højde for manglende kvalitet og relevans af de tilgængelige data. I relation til fastsættelse af kvalitetskriterier i jord, luft og drikkevand har denne faktor typisk varieret fra 1 til 100 afhængigt af datagrundlaget for de pågældende stoffer.

I UF_{III} indgår bl.a. følgende elementer :

- kvaliteten af datasættet (fx ekstrapolation fra subkronisk nuleffektniveau til kronisk nuleffektniveau),,
- ekstrapolation fra en eksponeringsvej til en anden ("route to route" ekstrapolation fx omregning fra oral dosis til inhalationsdosis),
- ekstrapolation fra LO(A)EL til NO(A)EL,
- og alvorligheden af effekterne (fx kræftfremkaldende effekter).

Det er ikke muligt at pege på en specifik størrelsesorden for en standardværdi, hverken for de enkelte delelementer af UF_{III} eller for den samlede UF_{III}.

Der henvises til Miljøprojekt Nr. 974 (2005) for en nærmere beskrivelse af UF_{III} i rapportens afsnit 4.4.3.

Nedenfor anføres mere kortfattet retningslinierne for fastsættelse af UF_{III}.

Kvalitet og relevans af data

UF_{III} skal tage højde for, om der er kvalitetsmæssige eller datamæssige mangler. Vurdering af kvalitet, omfatter vurdering af om de enkelte undersøgelser (og især undersøgelserne der refererer til de kritiske effekter og effektniveauer) er udført og afrapporteret på en måde, så resultaterne anses for relevante og pålidelige. Selve omfanget af datasættet vurderes for data vedrørende alle relevante effektområder i relation til kortvarig og langvarig eksponering, og det vurderes om der i datasættet er væsentlige mangler i forhold til vurdering af kritisk effekt og estimering af TDI. En faktor på 1 anvendes ved datasæt, som vurderes at være fuldt tilstrækkeligt for det givne stof, mens der ved mindre eller større mangler har været anbefalet faktorer i størrelsesordenen 3-10. I visse tilfælde med store mangler kan en faktor på helt op til 100 komme på tale. Fastlæggelse af UF_{III} må ofte baseres på en ekspertvurdering og foretages under hensyntagen til det enkelte stofs toksikologiske profil.

Et særligt aspekt, er manglende viden om børn og ufødtes følsomhed for konkrete kemiske påvirkninger. For at beskyt-

te børn og ufødte er det vigtigt ved fastsættelse af UF_{III} at vurdere om der er tilstrækkelig viden om disse forhold ud fra reproduktionsforsøg og flergenerationsforsøg. Er disse forhold ikke tilstrækkeligt belyst bør mangel på data have indflydelse på valget af UF_{III}. Størrelsen af en sådan delfaktor må ses i sammenhæng med stoffets toksikologiske profil og hvilke øvrige data, der haves.

Ekstrapolation fra en eksponeringsvej til en anden

Ved mangel på data for den relevante eksponeringsvej kan det blive nødvendigt at foretage en "route-to-route" ekstrapolation. De analyser, der er foretaget med hensyn til ekstrapolation fra oralt NO(A)EL til et NO(A)EC ved inhalation, peger generelt på, at en ekstrapoleret værdi ofte vil være væsentligt højere end en observeret værdi (dvs toksiciteten undervurderes). Det modsatte gør sig generelt gældende når der ekstrapoleres fra oralt NO(A)EL til NO(A)EL ved hudkontakt, hvor den ekstrapolerede værdi ofte vil være lavere end en observeret værdi (dvs. toksiciteten overvurderes). Der kan imidlertid ikke peges på en konkret størrelse for usikkerhedsfaktor III ved disse ekstrapolationer, der hviler på et meget usikkert grundlag. Vurderingen af om en "route-to route" ekstrapolation skal foretages, og i hvilken udstrækning der skal anvendes en usikkerhedsfaktor i tilknytning hertil, må bero på en ekspertvurdering i det konkrete tilfælde.

For stoffer, hvor der kun findes subakutte eller subkroniske undersøgelser, er det ikke muligt at fastsætte et NO(A)EL for livstidseksponering, som generelt må forventes at ligge lavere. Der har været foretaget flere analyser af forholdet mellem NOAELs og LOAELs opnået i studier af forskellige eksponeringsvarigheder. Med baggrund i disse synes der at være belæg for en usikkerhedsfaktor af størrelsesorden minimum 10. En af de nyere analyser har således vist, at en faktor på 10 vil være tilstrækkelig i knap 90% af tilfældene, når der var tale om ekstrapolation fra subkroniske data, mens der ved ekstrapolation fra subakutte data skulle anvendes en væsentlig højere faktor (>20).

Ved ekstrapolation fra LO(A)EL til NO(A)EL kan der ikke peges på en generel faktorstørrelse, som afspejler en generel

usikkerhed. Praksis inden for området og de tilgængelige analyser af problemstillingen peger på, at en værdi omkring en faktor 10 i langt de fleste tilfælde er tilstrækkelig. Vurderingen må imidlertid bero på en ekspertvurdering i hvert enkelt tilfælde.

Med baggrund i vægtning af ovenstående aspekter anvendes en UF_{III} på 10 som udgangspunkt. Denne værdi kan så korrigeres, når der er grundlag for dette i konkrete tilfælde.

Denne problemstilling kan i visse situationer omgås, når det ud fra data er muligt at beregne en benchmark-dosis, således at denne anvendes i stedet for LO(A)EL-værdien.

Alvorligheden af effekter

Det har været praksis ved fastsættelse af kvalitetskriterier, at særligt alvorlige effekter (fx kræftfremkaldende effekt eller fosterbeskadigende effekter) afspejles i usikkerhedsfaktor III. Det gælder især i de tilfælde, hvor den alvorlige effekt optræder ved lave dosisniveauer. Her kan anvendes *en ekstra faktor* på op til 10 for at tage hensyn til dette. For at få den endelige værdi for UF_{III} ganger man de enkelte værdier sammen.

Sammenfattende kan det konkluderes, at det ikke er muligt at sætte en standardværdi for en samlet UF_{III}. Usikkerhederne inden for de forskellige områder må vægtes, når der tages stilling til en samlet faktor for UF_{III}, da en multiplikation af mange delfaktorer i UF_{III} kan give en meget høj værdi, og i visse tilfælde give et skævt billede af det samlede videngrundlag og kvaliteten heraf. Fastsættelse af UF_{III} bør således i høj grad bero på en ekspertvurdering, hvor valget af faktoren og elementerne heri tydeligt begrundes.

4.2.4 Samlet usikkerhedsfaktor

Ved beregning af TDI divideres NO(A)EL evt. LO(A)EL med de tre usikkerhedsfaktorer, der således ganges sammen.

Ved multiplikation af UF_I, UF_{II} og UF_{III} bør der imidlertid tages stilling til størrelsen af den samlede usikkerhedsfaktor,

og der bør foretages en overordnet vurdering i forhold til det givne datasæt.

En meget stor usikkerhedsfaktor, der kan medføre en meget lille TDI-værdi, betyder ikke nødvendigvis, at stoffet er lige så potent eller farligt som andre, mere velkendte stoffer med tilsvarende lave TDI-værdi. Den beregnede lave værdi må snarere ses som en følge af store usikkerheder i datagrundlaget. En samlet usikkerhedsfaktor på 10.000 og derover, bør derfor ikke anvendes.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

5 Beregning af TDI for kræftfremkaldende stoffer

5.1 Kræftfremkaldende stoffer med og uden tærskelværdi

Kræftfremkaldende stoffer anses for at kunne deles op i to grupper grundet deres virkningsmekanisme. Den ene gruppe består af stoffer, der virker kræftfremkaldende som følge af evnen til at påvirke cellernes delings- og differentieringshastighed. Dette kan ske gennem direkte eller indirekte påvirkning af cellernes receptorer. For sådanne stoffer antages der at være en nedre tærskelværdi for denne effekt. Derfor kan TDI beregnes ved hjælp af usikkerhedsfaktorer, som omtalt i forrige afsnit.

Den anden gruppe af kræftfremkaldende stoffer virker gennem kemisk interaktion med cellernes arvemasse, og den kræftfremkaldende effekt vurderes at være en følge af stoffets beskadigelse af cellernes arveanlæg (mutagen/genotoksisk aktivitet). For disse stoffer anses der ikke at være et nedre eksponeringsniveau uden øget risiko for skadevirkninger. For sådanne stoffer anvendes ikke usikkerhedsfaktorer ved beregning af TDI. I stedet beregnes TDI ved hjælp af en matematisk modelberegning, hvor man på forhånd definerer et risikoniveau man vil acceptere, og beregner så hvilken udsættelse, som giver denne risiko.

Ved vurdering af kræftfremkaldende effekt er det således af stor betydning, om det kræftfremkaldende stof falder ind under den ene eller den anden kategori. Der findes stoffer, som både er genotoksiske – dvs. kræftfremkaldende uden tærskel for effekt, og kræftfremkaldende ved andre mekanismer, hvor der anses at være en tærskel. Ved vurdering af denne type stoffer, kræver det en ekspertvurdering at afgøre, om de data der ligger til grund, bedst giver grundlag for at vurdere stoffet som tilhørende den ene eller anden kategori. Der har således i EU været arbejdet på at udvikle

nærmere vejledning i hvordan disse typer stoffer kan vurderes[14].

5.2 Vurdering af kræftfremkaldende effekt

EU, WHO IARC (International Agency for Research on Cancer) og US EPA anvender en række forholdsvis sammenlignelige procedurer/ kriterier ved vurdering af, om et stof skal i kategorien "kræftfremkaldende". Stofferne indplaceres i forskellige underkategorier alt efter dokumentationens omfang, og under hensyntagen til om dokumentationen stammer fra humandata eller dyredata.

Yderligere indgår der i vurderingerne også stillingtagen til virkningsmekanismer, fx om stoffer virker gennem en mutagen/ genotoksisk mekanisme. I EU´s klassificeringssystem for kemiske stoffer er der også kriterier for, hvornår et stof skal kategoriseres som "mutagent".

Principperne for inddeling af kræftfremkaldende stoffer i kategorier er mere udførligt beskrevet i Miljøprojekt Nr. 974 (2005) afsnit 3.6.

Ved vurdering af et stofs kræftfremkaldende effekt i forbindelse med risikokarakterisering og beregning af kvalitetskriterier tages der så vidt muligt udgangspunkt i de ovennævnte vurderinger, idet der suppleres med opdateret viden fra litteraturen. Det vurderes om der er dokumentation for at den kræftfremkaldende effekt er en følge af genotoksisk virkning, og om stoffet derfor skal betragtes som værende uden en nedre tærskel for effekt.

Hvis data taler for, at stoffet virker gennem en ikke-genotoksisk mekanisme, anses stoffet for at besidde en tærskelværdi for skadelig effekt. For en række stoffer vil data og viden vedrørende virkningsmekanisme være meget mangelfuld, og det kan være vanskeligt at sondre, om stoffet skal betragtes som værende enten med eller uden tærskelværdi. Sådanne tvivlstilfælde kan bedst håndteres ved at betragte

[14] European Food Safety Authority (2005). Opinion on a Harmonised Approach for Risk Assessment of Compounds Which are both Genotoxic and Carcinogenic.

stoffet som havende en tærskelværdi, og i den forbindelse anvende en øget usikkerhedsfaktor (UF_{III}) for at tage hensyn til usikkerheden om eksistensen af en tærskelværdi. Der bør således være et vist datagrundlag, der peger hen mod en genotoksisk mekanisme, før stoffet vurderes efter en model uden nedre grænse.

Ved vurderingen af kræftfremkaldende effekt tages stilling til om den er knyttet til bestemte eksponeringsveje, og om disse er relevante i forbindelse med et kvalitetskriterium for stoffet i jord, luft eller drikkevand.

Visse kræftformer kan være forsøgsdyrsspecifikke, og når sådanne kræftfund kan dokumenteres som ikke-relevante i human sammenhæng, tillægges disse fund kun begrænset vægt (se afsnit 3.5).

Ved vurderinger i relation til kvalitetskriterier tages der alene stilling til om stoffet skal betragtes som kræftfremkaldende, og om stoffet ud fra dets virkningsmekanisme skal anses for at have en tærskelværdi eller ej. Der foretages således ikke en mere detaljeret indplacering i forskellige kategorier i forhold til dokumentationens art og omfang, dvs. der inddeles ikke i kategorier for humane og dyreeksperimentelle kræftfremkaldende stoffer.

Et stof, som på baggrund af dokumentationen indplaceres i EU's Carc1 eller Carc 2, i IARC's gruppe 1 eller gruppe 2A/2B, og/eller i US EPA's gruppe A eller B1/B2, vil som udgangspunkt medføre, at stoffet betragtes som kræftfremkaldende, med mindre der er nyere undersøgelser eller velunderbyggede informationer og fortolkninger af data, der taler imod en sådan vurdering.

Tilsvarende gælder stoffer, som er opført på Arbejdstilsynets liste over stoffer, som anses for at være kræftfremkaldende. Det skal dog understreges, at stoffer, som hverken er vurderet af EU, IARC og/eller US EPA eller er indplaceret i en lavere kategori (EU Carc3, IARC gruppe 3 og/eller US EPA gruppe C/D), godt kan blive betragtet som kræftfremkaldende i relation til fastsættelse af kvalitetskriterier for kemikalier i jord, luft og drikkevand, hvis der er velunderbyggede data, der taler for det. Det kan for eksempel være i

tilfælde af, at der er publiceret nye undersøgelser siden en eventuel vurdering er foretaget af EU, IARC og/eller US EPA, eller der kan være stor kemisk strukturlighed med andre kendte kræftfremkaldende stoffer. I sidstnævnte tilfælde kan QSAR-modellering indgå som supplerende støtte for vurderingen.

5.3 Beregning af TDI for kræftfremkaldende stoffer uden tærskelværdi

5.3.1 Tolerabelt risikoniveau

TDI-værdien for kræftfremkaldende stoffer uden tærskelværdi fastsættes til en værdi, der repræsenterer et accepteret risikoniveau for udvikling af kræft. Denne værdi har traditionelt været fastsat til en 10^{-6} livstidsrisiko, og vil også med udsendelse af denne vejledning være det ønskede risikoniveau.

Konkret betyder dette, at man med udgangspunkt i humandata eller dyreforsøg, hvor den kræftfremkaldende effekt er påvist, ved hjælp af matematisk modellering estimerer dosis-respons kurvens forløb så langt ned i lav-eksponeringsområdet, at en eksponering svarende til en 10^{-6} livstidsrisiko kan beregnes. TDI er således den daglige gennemsnitseksponering, der ud fra *teoretiske beregninger* svarer til en forøget risiko for cancer på 1 ud af en million mennesker, som er udsat for stoffet gennem en hel livstid.

5.3.2 Metode til beregning af TDI og livstidsrisiko

Der er udviklet forskellige metoder til beregning af, hvor stor en risiko for udvikling af svulster en given eksponering for et genotoksisk kræftfremkaldende stof udgør. For alle metoder gælder, at der anvendes en eller anden form for matematisk ekstrapolation fra dosisniveauer med de kendte eksperimentelle værdier for forekomsten af svulster til de som regel meget lavere dosisniveauer, der svarer til en forekomst hos 1 ud af en million. Metoderne beskriver relationen mellem den daglige eksponering (udtrykt som dosis

eller koncentration) og den sammenhørende sandsynlighed for udvikling af svulster.

Beskrivelse af forskellige metoder og sammenligning mellem disse fremgår af Miljøprojekt Nr. 974 (2005) afsnit 5.3 og 5.4.

Den foretrukne metode til fastsættelse af TDI er T25-metoden, som også anvendes i EU-regi. Den baserer sig ligesom one-hit metoden på lineær ekstrapolation med udgangspunkt i laveste dosisniveau, hvor der optræder signifikant forøget antal svulster[15].

Metoden er en simplificeret lineær metode med udgangspunkt i en beregnet T25-dosis. T25-dosis defineres i denne sammenhæng som den kroniske eksponering (enhed: mg/kg legemsvægt per dag eller mg/m^3), som vil give 25% af forsøgsdyrene svulster i et specifikt væv, efter korrektion for den spontane hyppighed, indenfor den standardiserede levetid for den pågældende dyreart. Med udgangspunkt i denne T25-dosis foretages lineær ekstrapolation ved simpel forholdsregning ned til en dosis, der svarer til et tolerabelt risikoniveau.

Metoden og dens konkrete anvendelse i forhold til et tolerabelt 10^{-6} livstidsrisikoniveau er nøjere beskrevet i bilag 1.

5.3.3 Anvendelse af risikoestimater angivet i litteraturen

I en række tilfælde vil eksponeringsniveauet svarende til en 10^{-6} livstidsrisiko (eller "unit-risk"-estimater) på forhånd være beregnet af ekspertgrupper under fx WHO eller US EPA. For især nyere vurderinger kan det være relevant at anvende disse værdier som ligeværdigt udgangspunkt på

[15] Miljøstyrelsen har tidligere som administrativ praksis anvendt one-hit metoden. Anvendelse af T25-metoden vil i forhold til den medføre lidt højere værdier (skønsmæssigt en faktor 2-4). Denne forskel skal dog ses i et 10^{-6} perspektiv, og må anses som meget beskeden, og i praksis uden betydning i forbindelse med det tilstræbte beskyttelsesniveau.

linie med T25-metoden ved beregning af TDI og kvalitetskriterier.

Argumenterne for at vælge et risikoestimat frem for et andet kan dels være rent faglige (fx bero på konkrete virkningsmekanismer) og dels være mere pragmatiske, idet man også bør vurdere nødvendigheden af at foretage selvstændige beregninger af et risikoniveau. For eksempel kan opgaven være meget omfattende for stoffer med stor datarigdom (fx PAH-stoffer), hvor en selvstændig vurdering vil kræve ulige mange resurser og inddragelse af høj faglig ekspertise på området for at kunne leve op til kvaliteten af en international vurdering.

Derfor vil man i konkrete tilfælde kunne basere kvalitetskriterier for kræftfremkaldende stoffer på andre metoder end T25 metoden.

Referencer

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

European Food Safety Authority (2005). Opinion on a Harmonised Approach for Risk Assessment of Compounds Which are both Genotoxic and Carcinogenic.

6 Beregning af kvalitetskriterier for kemikalier

Dette kapitel beskriver hvorledes kvalitetskriterierne for kemikalier i jord, luft og drikkevand kan beregnes med udgangspunkt i førnævnte TDI-værdier.
Første led i beregningen er en vurdering af, om hele TDI-værdien eller kun en brøkdel heraf tildeles til beregning af det konkrete kvalitetskriterium. Dette kan dels være begrundet i eksponeringsmæssige overvejelser - den såkaldte allokering, hvor der tages hensyn til evt. andre kilder, der har betydning for eksponeringen. Eller det kan være begrundet i øvrige forhold, der kan være afgørende for, om der anvendes en reduktionsfaktor, så kun en mindre brøkdel af TDI-værdien anvendes ved beregning af kvalitetskriteriet.

Kapitlet angiver hvilke værdier for medieeksponering der anvendes ved beregning af kvalitetskriterierne. En mere uddybet forklaring på valget af disse værdier er givet i bilag 2.

6.1 Generel metode for beregning af et kvalitetskriterium

Et sundhedsmæssigt baseret kvalitetskriterium for et stof i jord, luft eller drikkevand beregnes ud fra den tolerable daglige indtagelse, ved at dividere TDI-værdien med den daglige eksponering for det relevante medie WHO/IPCS[16,17]. Det vil sige, at selve beregningen af kvalitetskriteriet ud fra TDI i princippet er ens for de tre medier, jord, luft og drikkevand

[16] WHO/IPCS (1994). Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. Environmental Health Criteria no. 170. International Programme on Chemical Safety.
[17] WHO/IPCS (1999). Principels for the assessment of risks to human health from exposure to chemicals. Environmental Health Criteria no. 210. International Programme on Chemical Safety.

og for alle stoffer, uanset om der foreligger tærskelværdi for den kritiske effekt eller ej.

Følgende generelle beregningsmetode kan opstilles:

$$KK_{l,j,v} = \frac{TDI \times V \times f}{E_{l,j,v}}$$

hvor

$KK_{l,j,v}$: kvalitetskriterium for jord, luft eller drikkevand
TDI: angives i mg/ kg legemsvægt/ d
V: legemsvægt i kg
f: allokeringsfaktor, brøkdel af TDI som ud fra eksponeringsfordeling anvendes til eksponering fra jord, luft eller drikkevand
$E_{l,j,v}$: daglig udsættelse/ forbrug af luft (m^3/d), jord (kg/d), eller drikkevand (liter/d).
(Hvis $E_{l,j,v}$ angives i (m^3/kg lgv/ d), (kg/kg lgv/d), eller (liter/kg lgv/d) udgår V i ovenstående formel).

6.1.2 Anvendelse af TDI

For stoffer hvor der ikke anses at være en tærskelværdi for effekt, dvs. de genotoksiske kræftfremkaldende stoffer, anvendes TDI-værdien (sv.t til en 10^{-6} livstidsrisikodosis) direkte til beregning af kvalitetskriteriet.
For andre stoffer, hvor der anses at være en tærskelværdi, indgår efterfølgende overvejelser vedrørende anvendelse af en allokeringsfaktor eller reduktionsfaktor.

Allokering

Allokeringsfaktoren "*f*" angiver den brøkdel af TDI som tildeles udsættelsen via det enkelte medie. Ofte tildeles mindre end 100% af et kemisk stof til beregning af et kvalitetskriterium, da der for en række kemiske stoffer vil være udsættelse gennem andre medier end det enkelte medie jord, luft og vand. I en række tilfælde vil bidrag gennem forurening af fødevarer eller gennem påvirkning fra indeklimaet udgøre hovedkilden for en persons udsættelse, og vil dermed "lægge beslag på" en betydelig del af TDI-værdien, hvorfor der kun tildeles en mindre andel til kvalitetskriteriet.

I de tilfælde hvor bidrag fra andre medier ikke forventes, fastsættes kvalitetskriterierne som udgangspunkt ved, at hele TDI allokeres til det pågældende medie. Når det vurderes, at der foreligger øvrige betydende kilder allokeres som udgangspunkt 10% af TDI til det pågældende medie, med mindre der er konkret viden om øvrige kilders bidrag, der siger noget andet. For visse stoffer, hvor langt de største bidrag kommer fra øvrige kilder, kan der således allokeres helt ned til kun 1% af TDI (dette er for eksempel gjort for visse plastblødgørere i forbindelse med et kvalitetskriterium for drikkevand).

Reduktionsfaktor

I særlige tilfælde kan andre forhold end de rent eksponeringsmæssige betragtninger medføre, at der anvendes en reduktionsfaktor, således at der kun anvendes en vis brøkdel af TDI til beregning af kvalitetskriteriet. En sådan faktor kan efter individuel vurdering komme på tale fx :

- ved særligt kritiske forhold som persistens og bioakkumulering af stoffet,
- i tilfælde, hvor kvalitetskriteriet er udarbejdet ud fra et enkelt stofs effekter, men hvor stoffet repræsenterer en hel stofgruppe, og hvor eksponeringen typisk vil være karakteriseret ved en blandingseksponering med denne stofgruppe,
- i tilfælde hvor der samtidig kan optræde eksponering fra flere stoffer, og hvor der er formodning om, at der ved de relevante niveauer vil kunne optræde kombinationseffekter (fx samvirkende effekter) fra denne blandingseksponering.

6.1.3 Eksponeringsbetragtninger

Målet for eksponeringen i nævneren i brøken til kvalitetskriterie-beregningen baseres på standardbetragtninger for daglig udsættelse.

I afsnit 6 i Miljøprojekt Nr. 974 (2005) er der foretaget en opdateret gennemgang af viden om befolkningens udsættelse for jord, luft og vand, samt angivet hvilke standardbe-

tragtninger der anvendes inden for WHO, US EPA og i EU.

Ud fra hensigten om specifikt at tage hensyn til børns eksponering er der foretaget en revision af den hidtidige praksis. Grundlaget for den reviderede praksis er anført i bilag 2.

6.2 Beregning af luftkvalitetskriteriet

Følgende fremgangsmåde anvendes til beregning af luftkvalitetskriteriet (KK_{luft}), når TDI for de(n) kritiske effekt(er) er fra studier hvor stoffet er givet via munden og angivet i enheden mg/kg legemsvægt/dag:

$$KK_{luft} = \frac{TDI \times f}{E_{luft}} \quad \text{hvor}$$

TDI: tolerabel daglig indtagelse (mg/ kg lgv/d)
f: brøkdel af TDI, der allokeres til udeluften
E_{luft}: eksponering luft, standardværdi for dagligt indåndingsvolumen: 0,5 m^3/kg lgv/d for 1-5 årige børn.

Følgende fremgangsmåde anvendes, når (nul)effektniveauer NO(A)EL eller LO(A)EL) for de(n) kritiske effekt(er) er fra studier hvor stoffet er inhaleret og angivet som en koncentration i enheden mg/m^3:

$$KK_{luft} = TK \times f$$

f: allokeringsfaktor
TK: tolerabel koncentration

$$\text{hvor} \quad TK = \frac{NO(A)EC \text{ eller } LO(A)EC}{UF_I \times UF_{II} \times UF_{III}}$$

For de fleste typer af systemiske effekter anses det at være den samlede dosis og ikke stoffets koncentration i luften, der er af betydning for udvikling af disse effekter. Ved fastsæt-

telse af luftkvalitetskriteriet i disse tilfælde foretages en omregning af det fastlagte (nul)effektniveau til et gennemsnitligt døgnniveau (kontinuert eksponering) ud fra de i studiets aktuelle eksponeringsbetingelser. Det vil sige, at der kompenseres for, at eksponeringen ikke har foregået i alle døgnets timer over en fuld uge. Hvis eksponeringen for eksempel er foretaget 6 timer per dag i 5 dage per uge, korrigeres der med en faktor 6/24 til kontinuert eksponering gennem et helt døgn og en faktor 5/7 til kontinuert eksponering gennem hele ugen.

For visse lokale effekter (effekter, der optræder lokalt i luftvejene samt direkte effekter på hud og øjne) anses det sædvanligvis at være stoffets koncentration i luften og ikke den samlede dosis som sådan, der er af betydning for udvikling af disse effekter. For sådanne stoffer kan omregning til en kontinuert eksponering sædvanligvis udelades fra en konkret vurdering.

Lugt

Nogle kemiske stoffer har en meget kraftig lugt, og hensyntagen til lugt ved fastsættelse af luftkvalitetskriteriet vil for mange stoffer (fx en række organiske opløsningsmidler) medføre lavere luftkvalitetskriterium end det sundhedsbaserede luftkvalitetskriterium, idet luftkvalitetskriteriet fastsættes til 1/3 af 50 %-lugtgrænsen.

Lugtgrænsen for et kemisk stof er generelt defineret som den koncentration i luften, hvor 50 % af et lugtpanel (kontrolleret laboratorieforsøg med bestemmelse af lugtgrænse med frivillige forsøgspersoner) kan registrere lugten.

Som et luftkvalitetskriterium vurderes en sådan 50 % lugtgrænse at kunne medføre gener hos en ikke uvæsentligt del af befolkningen.

I en analyse af en række laboratoriedata, hvor lugtgrænsen er blevet bestemt for kemiske stoffer, viser det sig, at dosis-respons sammenhængen generelt er ret stejl for et lugtpanel (DK-Teknik 2001). For personer, med intakt lugtesans (svarende til personer, der indgår i et lugtpanel), er der ge-

nerelt en forholdsvis lille spredning m.h.t. hvornår en lugt kan opfattes. Ud fra disse data vurderes, at ved et niveau på 1/3 af lugtgrænsen vil maksimalt 1-5% af befolkningen under optimale betingelser kunne fornemme lugt.

Lugtgrænser, der er angivet i litteraturen, kan variere voldsomt (flere størrelsesordner) Dette skyldes, at metoder til bestemmelse af lugtgrænser kan variere meget, alt efter hvor og hvornår de er blevet foretaget, samt at der kan være foretaget undersøgelser af forskellige stofkvaliteter, hvor stofferne ikke er entydigt definerede.

Medmindre lugtgrænsen er bestemt ud fra nye og meget velbeskrevne metoder, hvor kvalitet og pålidelighed af undersøgelsen kan vurderes, anbefales det at fastsætte luftkvalitetskriterier på baggrund af lugt fra bestemmelse af lugtgrænse foretaget af et akkrediteret laboratorium.

6.3 Beregning af jordkvalitetskriteriet

Følgende fremgangsmåde anvendes ved beregning af jordkvalitetskriteriet, når de(n) kritiske effekt(er) er en følge af gentagen udsættelse:

$$KK_{jord} = \frac{TDI \times V \times f}{E_{I,jord} \text{ (eller } E_{H,jord})} \quad \text{hvor}$$

f: procentdel af TDI, der allokeres til indtagelse af jord
V: legemsvægt, 1-3 årigt barn: 13 kg
$E_{I,jord}$: daglig eksponering (indtagelse) for jord, standardværdi:

1) 0,0002 kg/d (sv.t. 95-percentilgrænsen) i tilfælde hvor hele TDI-værdien eller hovedparten af denne anvendes til beregning af kvalitetskriteriet
2) 0,0001 kg/d (sv.t. medianudsættelse) i tilfælde hvor TDI er en 10^{-6} livstidsrisikodosis for et kræftfremkaldende stof, eller i tilfælde, hvor der anvendes en mindre del af TDI til jordkvalitetskriteriet

$E_{H,jord}$: daglig eksponering (hudkontakt) for jord, standardværdi: 0,001 kg/d for barn.

$E_{H,jord}$ anvendes i forbindelse med særligt hudgennemtrængelige stoffer, hvor systemisk bidrag fra hudoptag summeres med det orale bidrag. $E_{H,jord}$ anvendes separat hvis den kritiske effekt er relateret til den direkte hudpåvirkning af forureningskomponenten.

Følgende fremgangsmåde anvendes ved beregning af jordkvalitetskriteriet, når den kritiske effekt er akut toksicitet:

$$KK_{jord} = \frac{TD \times V}{E_{I,jord} \text{ (eller } E_{H,jord})} \quad \text{hvor}$$

TD: tolerabel enkeltdosis

$$\text{hvor} \quad TD = \frac{NO(A)EL_{akut} \text{ eller } LO(A)EL_{akut}}{UF_I \times UF_{II} \times UF_{III}}$$

V: legemsvægt, 1-3 årigt barn: 13 kg
E_I: maksimum enkeltindtag af jord (0,010 kg)
E_H: maksimal hudkontakt med jord (0,010 kg) (Anvendes for særligt hudgennemtrængende stoffer).

Ved beregning bør data for optagelse fra mave-tarmkanalen (biotilgængelighed[18]) så vidt muligt inkluderes, idet de forurenende stoffer i visse tilfælde kan binde sig kraftigt til jordpartikler og derved medføre en reduceret biotilgængelighed. Hvis data ikke foreligger, regnes med samme biotilgængelighed, som i de forsøg, der ligger til grund for TDI-beregningen (dette kan typisk være forsøg, hvor teststoffet er opblandet i foderet eller i drikkevandet).

[18] "Biotilgængelighed" refererer i denne sammenhæng til det engelske "bioaccessibility". Bioaccessibility beskriver den pool af stoffet, som jorden kan frigive, og den indeholder således også let bundne former.

Lugt, udseende

Ud over rent sundhedsmæssige aspekter tages der ved fastsættelse af jordkvalitetskriteriet hensyn til, at jorden ikke må lugte eller syne forurenet. Det vil sige, at jorden ikke ved inspektion må afgive lugt fra forureningen eller se forurenet ud (klumper af stof(fer) eller misfarvning. Der foreligger ikke nøjere retningslinier for en sådan subjektiv vurdering. Med hensyn til lugt i forbindelse med afdampning henvises til Miljøprojekt Nr. 974 (2005) afsnit 7.4.3, der beskriver anvendelsen af luftkvalitetskriteriet i forbindelse med afdampning af forurenende stoffer fra jord.

6.4 Beregning af drikkevandskvalitetskriteriet

Følgende fremgangsmåde anvendes ved beregning af drikkevandskvalitetskriteriet:

$$KK_{drikkevand} = \frac{TDI \times f}{E_{drikkevand}} \quad \text{hvor}$$

TDI: tolerabelt dagligt indtagelse (mg/ kg lgv/d)
f: er den procentdel af TDI, der allokeres til indtagelse af drikkevand
$E_{drikkevand}$: daglig eksponering for drikkevand, standardværdi:

1) 0,08 liter/ kg lgv/d (sv.t. 95-percentilen) for 1-10 årige børn. Anvendes i forbindelse med akutvirkende stoffer eller når hovedparten af TDI-værdien benyttes til beregning af drikkevandskvalitetskriteriet.
2) 0,03 liter/ kg lgv/d (sv.t. medianværdi for 1-10 årige børn) i tilfælde hvor TDI er en 10^{-6} livstidsrisikodosis for et kræftfremkaldende stof, eller i tilfælde hvor kun en mindre andel af TDI-værdien benyttes til beregning af drikkevandskvalitetskriteriet.

Kemiske stoffer, der er særligt hudoptagelige, vil kunne optages i en ikke uvæsentlig mængde i forbindelse med badning, og stoffer, der let fordamper (for eksempel mange opløsningsmidler), kan især ved brusebadning indåndes som dampe/aerosoler. Omfanget af disse eksponeringsformer via brusebadning og karbadning afhænger af, hvor letoptageligt stoffet er gennem huden, og i hvor stor udstrækning stoffet frigives ved fordampning fra vandet. Der bør derfor i konkrete tilfælde (stoffer med høj hudgennemtrængelighed og stoffer med høj flygtighed fra vandfasen), tages højde for disse bidrag ved fastsættelse af drikkevandskvalitetskriteriet, således at den samlede optagelse via drikkevand og badevand ikke overskrider den del af TDI, som er allokeret til drikkevand.

Der er ikke opstillet konkrete modeller for beregningen af sådanne bidrag, hvorfor bidragene må vurderes fra sag til sag under inddragelse af de data og de vurderinger, der er foretaget for det konkrete stof.

Lugt, smag, udseende

Drikkevandet må ikke lugte, smage eller syne forurenet og smag, lugt og udseende af drikkevandet har en væsentlig betydning, også selv om det ikke udgør en sundhedsfare i relation til indtagelse af drikkevand.

Det vil i nogle tilfælde være lugt, smag, eller udseende, og ikke stoffets sundhedsmæssige effekter, der er bestemmende for værdien af drikkevandskvalitetskriteriet.

Lugt- og smagsgrænser, der findes opgivet i litteraturen, kan variere voldsomt selvom de forskellige undersøgelser er udført med samme stof. Dette skyldes, at metoder kan variere meget alt efter hvor, og hvornår de er blevet foretaget.

Medmindre lugt- og smagsgrænsen er bestemt ud fra nye og meget velbeskrevne metoder, hvor kvalitet og pålidelighed af undersøgelsen kan vurderes, anbefales det, at fastsættelse af drikkevandskvalitetskriterier på baggrund af lugt og smag foretages ud fra bestemmelse af lugt- og smagsgrænse foretaget af et akkrediteret laboratorium.

Udgangspunktet for fastsættelse af et lugt- og smagsbaseret kvalitetskriterium bør være et nul-effekt-niveau (NOEL) i testpanelet, dvs. den koncentration hvor testpanelet som helhed ikke kan lugte eller smage stoffet.

Ofte angives lugt- og smagsgrænser i vand på tilsvarende måde som lugtgrænse i luft, dvs. det niveau, hvor 50 % af et testpanelet kan lugte/smage stoffet. Angives således lugtgrænsen i den tilgængelige litteratur som 50 %-grænsen ganges denne værdi med 1/3 (som beskrevet under luftkvalitetskriteriet), idet anvendelse af en sådan faktor skønnes at sikre, at kun en mindre andel af befolkningen vil kunne fornemme stoffet ved dette niveau.

Referencer

DK-Teknik (2001). Vurdering af lugttærskelværdier. Rapport udarbejdet for Miljøstyrelsen.

Miljøstyrelsen (2005). Miljøprojekt Nr. 974. Principper for sundhedsmæssig vurdering af kemiske stoffer med henblik på fastsættelse af kvalitetskriterier for luft, jord og vand.

Bilag 1

Kvantitativ vurdering ved benyttelse af T25 ekstrapolationsmetoden.

Anbefaling af T25-metoden

Der er udviklet forskellige metoder til beregning af, hvor stor en risiko for udvikling af svulster en given eksponering for et genotoksisk kræftfremkaldende stof udgør. Ens for dem alle er at der anvendes en form for statistisk ekstrapolation fra dosisniveauer med kendte eksperimentelle værdier til de som regel meget lavere dosisniveauer, der oftest er relevante i relation til den generelle befolknings eksponering for kemiske stoffer i miljøet. Metoderne beskriver relationen mellem den administrerede daglige dosis (eller koncentration) og den resulterende hyppighed af svulster.

Som anført i afsnit 5.3.2. anbefales det at anvende T25-ekstrapolering som beskrevet i en baggrundsrapport udarbejdet af en arbejdsgruppe i forbindelse med EU´s risikovurderingsarbejde (EU-Commission 1999).

T25-metoden er blevet evalueret ved at sammenligne resultater opnået ved denne metode med resultater opnået ved anvendelse af US EPA´s LMS-metode (Linearised Multistage Model) og ved LED_{10} metoden, hvor der foretages lineær ekstrapolation ned i lavdosisområdet ud fra en benchmark-dosis på 10 % effektniveau (se afsnittene 5.3.2-4 i Miljøprojekt Nr. 974 (2005)). Ved gennemgang af resultaterne for lavdosis-estimaterne vurderede man at de er sammenlignelige.

Ved sammenligning af one-hit metoden og T25-metoden vurderes de at medføre sammenlignelige værdier, idet udgangspunktet for begge vurderingerne i princippet er at anvende laveste dosis med signifikant respons, og herfra

foretage lineær ekstrapolation ned i lavdosisområdet. I T25-metoden divideres med en skaleringsfaktor fra dyr til menneske opløftet i ¼ (sv.t. skalering i forhold til stofskiftet), mens tilsvarende skaleringsfaktor i one-hit metoden er opløftet til 1/3 (sv.t. skalering i forhold til overfladeareal). Dette forhold alene vil maksimalt bevirke en forskel på de to metoder på ca. en faktor 2.

Anvendelse af T25-metoden

T25 defineres som den kroniske dosis (enhed: mg/kg legemsvægt per dag), som vil give 25 % af forsøgsdyrene svulster i et specifikt væv, efter korrektion for den spontane hyppighed, inden for den standardiserede levetid af den pågældende art.

T25 beregnes med udgangspunkt i et langtidscancerstudie, hvor den laveste dosis, der giver en signifikant forøgelse af forsøgsdyr med svulster i et specifikt væv, som udgangspunkt anvendes ved beregningen af T25. Forekomsten af ondartede og godartede svulster sammenlægges, når de godartede svulster må mistænkes for at kunne udvikles til ondartede. Hvis der er en højere hyppighed ved en højere dosis, der giver en lavere T25, anvendes sidstnævnte, med mindre der er særlige begrundelser for ikke at tage udgangspunkt i denne. Hvis der er flere data- sæt, beregnes T25 for det mest relevante datasæt. Hvis forskellige datasæt giver T25-værdier, som ligger inden for et relativt snævert interval, anvendes gennemsnittet af disse T25-værdier. Hvis sidstnævnte procedure ikke anvendes, skal rationalet for anvendelse af en anden procedure begrundes nøje.

Beregning af T25 foretages ved at gange dosis D (mg/kg lgv/dag), hvor signifikant forøget antal svulster forekommer med faktoren 0,25/p, hvor p er den aktuelle hyppighed af svulster: $T25 = D \times 0{,}25/p$.

Ud fra denne T25 foretages lineær ekstrapolation ned i lavdosisområdet, idet dosis svarende til et givent risikoniveau beregnes ved simpel forholdsregning. En gennemsnitlig daglig dosis svarende til en øget livstidsrisiko på 10^{-6} kan således beregnes på følgende måde:

$$Dosis_{(10\text{-}6\ livstidsrisiko)} = 10^{-6} / 0{,}25 \times T25\text{-dosis}$$

Imidlertid indgår der i beregningerne også dosiskorrektion med baggrund i en skaleringsfaktor mellem dyr og mennesker, og en faktor som angiver andelen af dyrenes levetid, hvor eksponeringen har fundet sted.

Konkret kan TDI svarende til en 10^{-6} livstidsrisiko beregnes ud fra følgende formel, når T25 er beregnet ud fra forsøg med oral eller dermal eksponering hos dyr:

$$TDI = \frac{I_t \times [L_e / L]^2 \times [(T25 \times l_e) / L_e]}{0{,}25 \times [W_h / W_a]^{0,25}}$$

I_t: Den tolerable livstidsrisiko (10^{-6}).
L_e: Den aktuelle levetid for dyrene.
L: Den teoretiske gennemsnitslængde af levetiden for dyrene.
T25: Beregnet daglig dosis (mg/kg legemsvægt per dag), der medfører en 25% forøget forekomst af tumorer hos forsøgsdyrene.
l_e: Eksponeringstid.
$[W_h / W_a]^{0,25}$: Dosiskorrektion på basis af stofskifte
hvor
W_h: Menneskets vægt i kg (sættes oftest til 70 kg).
W_a: Gennemsnitsvægt af det pågældende forsøgsdyr (kg).

L, L_e l_e:
Hvis eksponeringsvarigheden er kortere end standardlevetiden for den pågældende art, eller hvis studiet afsluttes inden standardlevetiden, foretages dosiskorrektion, som beskrevet i den anførte formel. Standardlevetiden for mus, rotter og hamstre sættes med mindre andet er angivet for den specifikke stamme til 24 måneder.

Dosis (T25)
Når konkrete data ikke specifikt angiver dyrenes legemsvægt, og dosis i mg/kg lgv/d i forbindelse med dosering gennem foder eller drikkevand, anvendes følgende værdier i beregningen:

Dyreart	Legemsvægt (kg)	Indtagelse af foder (g/kg/dag)	Indtagelse af drikkevand (ml/kg/dag)
Rotte	0,10 (ung) 0,40 (ældre)	100 50	 75
Mus	0,020	150	-
Marsvin	0,75	40	-
Kanin	2,0	30	-
Hund	10,0	25	-
Abe	5,0	50	-

$(W_h/W_a)^{0,25}$

Er stoffet givet oralt eller dermalt , foretages der en dosiskorrektion for forskelle i kropsstørrelse mellem dyr og mennesker ved at omregne T25 til den tilsvarende humane dosis i mg/kg lgv./dag ved allometrisk skalering på basis af stofskiftet (afsnit 4.4.1.2). Denne skalering opnås ved at dividere T25-dosis med faktoren $(W_h/W_a)^{0,25}$

Ved beregning ud fra inhalationsundersøgelse beregnes den tolerable indåndingskoncentration TC ved anvendelse af formlen:

$$TC = \frac{I_t \times [L_e / L]^2 \times [(T25 \times l_e) / L_e]}{0{,}25}$$

I_t: Den tolerable livstidsrisiko (10^{-6}).
L_e: Den aktuelle levetid for dyrene.
L: Den teoretiske gennemsnitslængde af levetiden for dyrene.
T25: Beregnet daglig dosis (mg/m³ i indåndingsluften), der medfører en 25% forøget forekomst af tumorer hos forsøgsdyrene.
l_e: Eksponeringstid.

Ved indsættelse af T25 anvendes koncentration i indåndingsluften og der foretages sædvanligvis omregning til en gennemsnitligt indåndingskoncentration , hvis forsøget ikke er udført med kontinuerlig eksponering. Dette gøres ved at korrigere for antal timer pr. dag og antal dage pr. uge hvor eksponeringen har fundet sted.

Referencer

EU-Commission (1999). Guidelines for quantitative risk characterisation of non-threshold carcinogens in the framework of Existing Chemicals following Council Regulation (EEC)

793/93. Commission working Group on the Technical Meetings for Risk Assessment for Existing Substances. Document NO_NL/01/99_Rev.1

Bilag 2

Baggrund for anvendte eksponeringsværdier ved beregning af kvalitetskriterier

I denne vejledning ændres praksis med hensyn til de eksponeringsværdier, der anvendes ved beregning af kvalitetskriterierne for kemikalier.

Tidligere har man i forbindelse med beregning af jordkvalitetskriterier som udgangspunkt antaget, at et barn dagligt indtager 0,2 g jord/d (0,0002 kg/d), mens man ved beregning af luftkvalitetskriterier har anvendt et scenarie, hvor en voksen person dagligt indånder 20 m^3 luft. Ved beregning af kvalitetskriterier for kemikalier i drikkevand har udgangspunktet været et dagligt indtag på 2 liter vand for en voksen.

Børn
Beskyttelse af børn er de senere år i stigende grad kommet i fokus, og det har i den forbindelse været debateret i hvilken udstrækning kvalitetskriterier beregnet ud fra voksnes udsættelse i tilstrækkeligt omfang beskytter børn, da børn i forhold til deres legemsvægt generelt indtager/ indånder en større mængde drikkevand/ luft.

I miljøprojekt nr. 589 "Children and the unborn child – exposure and susceptibility to chemical subtances-" fra 2001, blev der foretaget en detaljeret gennemgang af børns særlige følsomhed og udsættelsesmønster i forbindelse med miljøforureninger og kemiske stoffer. I miljøprojektet konkluderes, at der ved fremtidig fastsættelse af kvalitetskriterier for kemikalier bør tages udgangspunkt i eksponeringsværdier for børn, for at opnå at børn er fuldt omfattet af det beskyttelsesniveau, kvalitetskriteriet repræsenterer. Denne vejledning indarbejder således ønsket om, at principperne for fastsættelse af kvalitetskriterier tager hensyn til børns særlige udsættelse.

Børns udsættelse vil således være udgangspunktet for de fremtidige standardværdier for eksponering i forbindelse med jord, luft og drikkevand.

Standardværdien for børns legemsvægt vil i fremtiden være 13 kg.

Særligt udsatte undergrupper
Almindelig biologisk variation og forskellige former for adfærd vil betyde, at nogle grupper vil være mere udsatte end andre for en given påvirkning via miljøet. Der er således også med denne vejledning taget stilling til, i hvilken udstrækning kvalitetskriterier/grænseværdier for kemikalier skal tage hensyn til de mere udsatte grupper i befolkningen.

Når kvalitetskriterier for kemikalier beregnes ved at anvende eksponeringsværdier, der svarer til befolkningsvægtede gennemsnitsværdier eller medianværdier, betyder dette, at ca. halvdelen af den befolkning, som kvalitetskriteriet skal søge at beskytte, vil kunne blive udsat for *større* eksponering end udgangspunktet for beregningen (fx en TDI-værdi). Hvis sigtet med et kvalitetskriterium er, at størstedelen af befolkningen skal være omfattet at det ønskede beskyttelsesniveau, vil det således være nødvendigt at anvende en øvre fraktilværdi for befolkningens udsættelse.

Det skal nævnes, at de standardværdier der hidtil har været anvendt for børn (jord) og voksne (luft og drikkevand) jf. beskrivelsen af eksponeringsværdier, svarer til sådanne øvre percentilgrænser.

Kvalitetskriterier for kemikalier skal fortsat sigte mod at beskytte flertallet af befolkningen, dvs. også de mere udsatte undergrupper. I de tilfælde hvor befolkningsfordelingen af eksponeringerne kendes, vil valg af øvre eksponeringsværdier typisk kunne foretages ved at tage udgangspunkt i værdier svarende til 90- eller 95-percentilerne.

For at sikre særligt udsatte grupper anvendes følgende principper :

a) I situationer, hvor man ved beregning af kvalitetskriteriet har tildelt hele TDI-værdien, hovedparten eller der er eksakt

viden om størrelsen af udsættelsen via det aktuelle medie, vil det være nødvendigt at anvende øvre percentilgrænser for eksponering. Herved sikres at kun en mindre andel af befolkningen vil blive eksponeret over TDI-niveau.

b) *I andre tilfælde, hvor der anvendes en reduceret TDI-værdi som følge af anvendelse af en allokeringsfaktor (evt. en særlig reduktionsfaktor), anvendes derimod medianværdier for udsættelse med mediet. Dette forhold begrundes med, at anvendelse af en allokeringsfaktor eller reduktionsfaktor sædvanligvs betyder anvendelse af runde værdier (10 % eller 1% af TDI), som ofte vil være på "den sikre side". I sådanne tilfælde, hvor der i forvejen er indbygget en øget grad af sikkerhed, vil det ikke være påkrævet også at anvende en øvre percentilværdi for eksponering. Anvendelse af en medianværdi vurderes her, at kunne opfylde målet om at beskytte størstedelen af befolkningen.*

c) *For kræftremkaldende stoffer uden tærskelværdi for effekt foretages som tidligere nævnt ingen allokering af TDI. I disse tilfælde anvendes medianværdier for eksponering ved beregning af kvalitetskriteriet, idet TDI-værdien repræsenterer en daglig dosis for en befolkningsvægtet gennemsnitlig livstidsrisikoforøgelse (en ekstrarisiko på 1 ud af 1 million udsatte over livstid), hvorfor en gennemsnits-/medianværdi for befolkningens udsættelse må være udgangspunktet.*

Luft, daglig standardeksponering

I forbindelse med valg af standardeksponering for luft tages der udgangspunkt i afsnit 6.1.1 i Miljøprojekt Nr. 974 (2005). I dette afsnit vurderes US EPA´s eksponeringsvurderinger at udgøre det bedste grundlag. I EU´s risikovurderingprogram for kemiske stoffer henvises ligeledes til de amerikanske værdier.

Uddrag af tabel 6.1.1

Alder	Legemsvægt (kg)	V_R – gennemsnit (m^3/dag)	V_R – inaktiv[a] (m^3/dag)	V_R - aktiv (m^3/dag)
Børn:				
Under 1 år	7.6	4.5	2.35	6.35
1 – 2 år	13	6.8	4.16	9.15

3 – 5 år	18	8.3	4.98	10.96
6 – 8 år	26	10	5.95	13.09

V_R = Respirations Volumen som henholdsvis gennemsnit, inaktivitet og aktivitet.

Værdierne er alders- og kønsopdelte, men angivelserne omfatter ikke spredningen (fordelingen) i de enkelte aldersgrupper. Det er således ikke ud fra de forliggende data muligt at aflæse en 90- eller 95 percentilværdi for eksponeringen af de forskellige aldersgrupper.

Som ovenfor nævnt vil der ved fastsættelsen af luftkvalitetskriterier blive taget hensyn til eksponeringen af børn.

Fra tabellen ses, at børn under 1 år i gennemsnit over et døgn indånder 4,5 m^3 svarende til 0,59 m^3/kg lgv/dag, mens børn i aldersgruppen 1-2 år og 3-5 år indånder henholdsvis 6,8 m^3 og 8,3 m^3 (svarende til henholdsvis 0,52 og 0,46 m^3/kg lgv/dag). Især de 1-5 årige må anses at være udeaktive.

På denne baggrund vil der ved beregning af luftkvalitetskriterier fremover blive anvendt en standardværdi for 1-5 årige børn på 0,5 m^3 luft/ kg lgv.

Til sammenligning kan nævnes at luftkvalitetskriteriet hidtil er blevet beregnet ud fra et dagligt indåndingsvolumen på 0,3 m^3/ kg lgv., idet udgangspunktet her var en voksen person (70 kg), der dagligt indåndede 20 m^3 luft.

Jord, daglig standardeksponering

Miljøprojekt Nr. 974 (2005) sammenfatter i afsnit 6.1.2 den seneste viden med hensyn til børns udsættelse for jord samt anfører forskellige organisationers vurdering.

Ud fra en sammenfattende vurdering kan følgende eksponeringsværdier for børn opstilles:

Eksponeringsvej	Eksponeringsværdi
Oralt, maksimum enkeltindtag	10 g

Oralt, dagligt gennemsnit	0,1 g/d
Oralt, 95-percentil	0,2 g/d
Hudkontakt, dagligt gennemsnit	1 g/d
Hudkontakt, maksimum	10 g/d

For børns indtag gennem munden fastsættes jordkvalitetskriteriet for akut toksiske stoffer ud fra et enkeltindtag på 10 g jord.

I tilfælde, hvor hele TDI eller hovedparten af TDI anvendes til beregning af jordkvalitetskriteriet, tages der specifikt hensyn til særligt udsatte børn, idet der ved beregning af jordkvalitetskriteriet tages udgangspunkt i 95-percentilen for udsættelse dvs. 0,2 g jord/d for herved at minimere risikoen for at overskride TDI-værdien.

I andre tilfælde, hvor der kun anvendes en mindre del af TDI-værdien til jordkvalitetskriteriet, anvendes ved beregningen af jordkvalitetskriteriet 0,1 g jord/d som et gennemsitligt standardindtag.

For kræftfremkaldende stoffer, hvor TDI svarer til en gennemsnitlig 10^{-6} livstidsrisikodosis blandt børn, anvendes 0,1 g jord/dag som udgangspunkt for beregning af jordkvalitetskriteriet.

Drikkevand, daglig standardeksponering

I Miljøprojekt Nr. 974 (2005) i afsnit 6.1.3 gennemgås viden om forskellige aldersgruppers indtagelse af drikkevand, idet indtagelse både opgives for forskellige aldersgrupper og fordelingen inden for disse (gennemsnitsværdier og 90-/95 percentiler). Beskrivelse i baggrundsrapporten omfatter vurdering og anbefalingerne fra US EPA´s Exposure Factors Handbook.

Baggrundsrapporten henviser i forbindelse med børns indtag af drikkevand til tabel 6.1.3 D, der angiver US EPA´s anbefalede værdier:

Alder	Gennemsnit (mean)	50 Percentil	90 Percentil	95 percentil

Under 1 år	0,30 liter/dag 44 ml/kg/dag	0,24 liter/dag 35 ml/kg/dag	0,65 liter/dag 102 ml/kg/dag	0,76 liter/dag 127 ml/kg/dag
Under 3 år	0,61 liter/dag		1,5 liter/dag	
3 – 5 år	0,87 liter/dag		1,5 liter/dag	
1 – 10 år	0,74 liter/dag 35 ml/kg/dag	0,66 liter/dag 31 ml/kg/dag	1,3 liter/dag 64 ml/kg/dag	1,5 liter/dag 79,4 ml/kg/dag

For spædbørn angives medianindtaget at være 35 ml/ kg lgv/d mens 95-percentilen angives at være på 127 ml/ kg lgv/d. For børn i aldersgruppen 1-10 år anføres tilsvarende en medianværdi på 31 ml/kg lgv./d og en 95-percentil på 79,4 ml/kg lgv./d.

Som standardværdi vurderes det mest relevant at anvende værdien for 1-10 årige børn, idet evt. forskelle i vaner m.h.t. amning i USA og Danmark kan have stor indflydelse på drikkevandsindtagelse for spædbørn. Endelig dækker TDI-begrebet alene moderens direkte eksponering og derved barnets indirekte udsættelse gennem modermælken, og TDI er således ikke formelt set beregnet til at dække spædbarnets direkte udsættelse. Gennemsnitligt er indtagene dog direkte sammenlignelige.

Ved beregning af drikkevandskvalitetskriteriet, hvor der ved en given forureningskomponent er foretaget en allokering på 100 % eller hovedparten af TDI til drikkevandet, eller i situationer hvor den kritiske effekt er en akut toksisk effekt, anvendes 95-percentilværdien for 1-10 årige børns indtag, dvs. et dagligt indtag på 0,08 liter/ kg lgv/d. I disse situationer, vil drikkevandskvalitetskriteriet således også omfatte spædbørns direkte udsættelse, idet dette også afspejler indtaget for børn under 1 år, som har en høj direkte udsættelse.

Ved beregning i andre situationer, hvor der er foretaget en allokering på en mindre del af TDI til drikkevandskvalitetskriteriet (eller der er anvendt en reduktionsfaktor) anvendes medianværdien for 1-10-åriges forbrug af drikkevand sv.t. 0,03 liter/ kg lgv/d. Allokeringen vil også betyde at spædbørns direkte udsættelse er omfattet af kvalitetskriteriet.

For kræftfremkaldende stoffer, hvor TDI svarer til en gennemsnitlig 10^{-6} livstidsrisikodosis blandt børn, anvendes

0,03 liter/ kg lgv/d som udgangspunkt for beregning af drikkevandskvalitetskriteriet.

Bilag 3

Anvendte forkortelser

BMD – BenchMark Dosis
D – dag
ED – Eksponerings Dosis
EEC – European Economic Community
EU – Europæiske Union
GLP – Good Laboratory Practice
IARC – International Agency for Research on Cancer
IPCS – International Programme on Chemical Safety
KK – kvalitetskriterie
LED – Linear Ekstrapolations Dosis
Lgv – legemsvægt
LMS – Linear Multistage Model
LOAEL – Lowest Observed Adverse Effect Level
LOEL – Lowest Observed Effect Level
NOAEL – No Observed Adverse Effect Level
NOEL – No Observed Effect Level
OECD – Organisation for Economic Cooperation and Development
PAH – Poly Aromatiske Hydrocarbon
QSAR – Quantitative Structure Activity Relationships
TDI – Tolerabel Daglig Indtag (Tolerabel Daglig Eksponering, el. Tolerabel Daglig Dosis)
TK – Tolerabel Koncentration
UF – Usikkerheds Faktor
US EPA – United States Environmental Protection Agency
V_R – indåndingsvolumen
WHO – World Health Organisation
W – Kropsvægt

Generelt

Et helt grundlæggende princip i risikovurdering af miljøtoksiner er adskillelse af risikovurdering og risikohåndtering.

Et grundlæggende princip er endvidere at Risikovurderingen skal foretages foretages stricte på videnskabeligt grundlag, uden indflydelse af fra politiske eller økonomiske interesser. På EU-niveau er dette bagrunden for oprettelsen af de Europæiske argenturer, herunder EFSA og EEA.

EFSA og EEA foretager vurderinger uden indflydelse fra de politiske lag i EU, og der er strenge krav til de eksterne eksperter argenturerne anvender for at sikre mod inhabilitet, i form af politiske eller økonomiske interesser. Således indgår eksempelvis ingen eksperter med relationer til industri-interessenter.

Når EU kommisionen støtter sig til vurdering fra en lille NGO (ICNIRP), frem for vurderinger foretaget af EEA, bryder de herved med den normale praksis i EU. Endvidere er størstedelen af medlemmerne i ICNIRP fysikere, og derfor ikke fageksperter i forhold til biologiske effekter. Endelig er der ikke en tilsvarende sikkerhed for inhabilitet, som normalt sikres i argenturernes vurderinger.

EEA har blandt andet i rapporten Late lessons fra 2013 anbefalet at forsigtighedsprincippet tages i anvendelse i forhold til Radiofrekvent Elektromagnetisk stråling. Citat (s. 38-39): It is therefore very important that large scale emerging technologies, such as biotechnologies, nanotechnologies and information and communication technologies, apply the precautionary principle based on the experiences and lessons learned from these and other case studies.

Dette dokument beskriver hvordan EFSA mener den videnskabelige evidens bør vurderes:
https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.4971

https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/sp.efsa.2020.EN-1843
(inhabilitet=impartiality)

I forhold til risikovurdering vil der altid være data gaps, og der vil altid være modstridende videnskabelige resultater.
http://www.efsa.europa.eu/en/methodology/guidance

Som det fremgår af EFSAs dokumenter, er det en alvorlig fejl hvis der kræves absolut bevis for effekt, herunder når ICNIRP eksempelvis afviser den meget omfattende forskning som viser non-thermale biologiske effekter, med den begrundelse mekanismen bag ikke fuldstændig forstået.

Som det fremgår af EFSAs dokumenter, er det derfor afgørende at den gruppe forskere der udvælges til at vurdere evidensen er habile, og eventuelle inhabilitet tages meget alvorlig. Dette er en af Agenturernes hovedopgaver.

Nationale vurderinger og myndighedernes håndtering

De nationale myndigheder (i DK SSI, FVST og MST) kan foretage risikovurdering dels på vurderinger fra de Europæiske Argenturer, men kan også tage risikovurderinger foretaget af nationale eksperter i betragtning. De nationale risikovurderinger skal igen foretages af forskere, som ikke har økonomiske eller politiske interesser, og der vil normalt være tale om eksperter på danske universiteter. På Fødevareområdet foretages sådanne risikovurderinger af Fødevareinstituttet DTU, mens Miljøstyrelsen får vurderinger fra flere af universiteterne.

Ligesom i EU, blev risikovurdering og risikohåndtering adskilt i Danmark ved udgangen af 2006, hvor sektorforskningsinstitutionerne (under ministerierne) blev adskilt fra myndighederne og langt ind under universiteterne. Forskerne har siden ikke været underlagt de myndigheder de rådgiver (med undtagelse af SSI).

https://www.eea.europa.eu/about-us

EEA

The European Environment Agency (EEA) is an agency of the European Union, whose task is to provide sound, independent information on the environment. The EEA aims to support sustainable development by helping to achieve significant and measurable improvement in Europe's environment, through the provision of timely, targeted, relevant and reliable information to policymaking agents and the public.

The European environment information and observation network (Eionet) is a partnership network of the EEA and its member and cooperating countries. Through Eionet, the EEA brings together environmental information from individual countries concentrating on the delivery of timely, nationally validated, high-quality data.

This knowledge is made widely available through the EEA website and forms the basis of both thematic and integrated environmental assessments. This information serves to support environmental management processes, environmental policymaking and assessment, as well as citizen participation.

Rapport:

Late lessons from early warnings: science, precaution, innovation

https://www.eea.europa.eu/publications/late-lessons-2

Kilde: https://mst.dk/kemi/kemikalier/graensevaerdier-og-kvalitetskriterier/

Grænseværdier og kvalitetskriterier

Hver dag bliver vi mennesker udsat for et stort antal kemiske stoffer. Det kan for eksempel ske gennem maden vi spiser, luften vi indånder, eller vandet vi drikker. For at beskytte mennesker og miljø mod uacceptable mængder af kemikalier, fastsætter Miljøstyrelsen grænseværdier - i form af de såkaldte kvalitetskriterier - for mange stoffer.

Sundheds-kvalitetskriterier

Sundhedskvalitetskriteriene bliver brugt til at beskytte mennesker mod forurening fra de mange kemikalier, som vi bruger og bliver udsat for i hverdagen. Se bl.a. grænseværdier for luft, jord og vand, datablade samt baggrundsrapporter.

Miljø-kvalitetskriterier for forurenende stoffer i vandmiljøet

Miljøstyrelsen fastsætter kvalitetskriterier for forurenende stoffer i vandmiljøet (søer, vandløb og havet). Kvalitetskriterierne bliver fastsat på baggrund af kemiske stoffers effekter på de dyr og planter, der lever i vandet. Få digital adgang til information om pt. 164 stoffer fordelt på 148 datablade.

https://mst.dk/kemi/kemikalier/graensevaerdier-og-kvalitetskriterier/sundhedskvalitetskriterier/

Sundhedskvalitetskriterier

Vi bruger mange kemikalier i vores hverdag. Derfor er det nødvendigt at beskytte mennesker og miljø mod forurening fra disse. Til det formål bliver der fastsat grænseværdier - de såkaldte kvalitetskriterier - for, hvilke koncentrationer af kemikalier, der må være i jord, luft, spildevand og drikkevand.

Om grænseværdier

Grænseværdier bliver brugt til at beskytte mennesker og miljø mod forurening fra de mange kemikalier, som vi bruger og bliver udsat for i hverdagen.

Grænseværdier for luft

Grænseværdier for kemikalier i luft bliver brugt til at begrænse og forebygge luftforurening fra virksomheder, energiproduktion og affaldsforbrænding.

Grænseværdier for jord

Grænseværdier for kemikalier i jord bliver brugt, når man skal vurdere forurenet jord, rydde op på forurenede grunde, og når man skal rådgive personer, der bor på lettere forurenede grunde.

Grænseværdier for vand

Grænseværdier for kemikalier i grundvand og drikkevand bliver fastsat, så vores drikkevand er sundhedsmæssigt forsvarligt at drikke, ser rent ud og er uden farve, smag eller lugt.

Datablade for stoffer med luftkvalitetskriterier og B-værdier
Her finder du en liste over stoffer, hvor der i forbindelse med fastsættelsen af kvalitetskriterier for luft og deraf følgende B-værdier er udarbejdet et datablad over stoffets sundhedseffekter mv.

Baggrundsrapporter (kriteriedokumenter) for stoffer med luftkvalitetskriterier
Her finder du en liste over stoffer, hvor der i forbindelse med fastsættelsen af kvalitetskriterier for luft er udarbejdet en baggrundsrapport.

Datablade for stoffer med jord- og drikkevandskvalitetskriterier
Her finder du en liste over stoffer, hvor der i forbindelse med fastsættelsen af kvalitetskriterier for jord og/eller drikkevand er udarbejdet et datablad over stoffets sundhedseffekter mv.

Baggrundsrapporter (kriteriedokumenter) for stoffer med jord- eller drikkevandskvalitetskriterier
Her finder du en liste over stoffer, hvor der i forbindelse med fastsættelsen af kvalitetskriterier for jord eller drikkevand er udarbejdet en baggrundsrapport.

https://mst.dk/kemi/kemikalier/graensevaerdier-og-kvalitetskriterier/sundhedskvalitetskriterier/baggrundsrapporter-for-stoffer-med-jord-eller-drikkevandskvalitetskriterier/

https://www2.mst.dk/Udgiv/publikationer/2006/87-7052-182-4/pdf/87-7052-182-4.pdf

Kvalitetskriterier for miljøfarlige forurenende stoffer i vandmiljøet

Miljøstyrelsen fastsætter kvalitetskriterier for forurenende stoffer i vandmiljøet (søer, vandløb og havet). Kvalitetskriterierne bliver fastsat på baggrund af kemiske stoffers effekter på de dyr og planter, der lever i vandet.

Miljøkvalitetskriterierne udgør den højeste koncentration af et forurenende stof, hvor myndighederne vurderer, at det ikke giver skader på vandmiljøet. Kvalitetskriterierne for vandmiljøet er baggrund for de lovmæssigt fastsatte miljøkvalitetskrav, som ligger til grund for at begrænse spildevandsudledninger til vandmiljøet.

De lovfastsatte miljøkvalitetskrav fremgår af bekendtgørelse nr. 1625 af 19. december 2017 om fastlæggelse af miljømål for vandløb, søer, overgangsvande, kystvande og grundvand, se den på retsinformations hjemmeside.

Nedenfor findes en liste over stoffer, hvor der i forbindelse med fastsættelsen af kvalitetskriterier for vand og/eller sediment og biota, er udarbejdet et datablad over stoffets miljøeffekter mv.

Databladene fremstår med varierende layout, forord og logo grundet tidspunktet for udarbejdelsen, og alle indeholder relevante informationer om kemikaliets fysisk-kemiske egenskaber, giftighed og fastsættelse af miljøkvalitetskriterier.

I databladene henvises til det vejledningsmateriale, som ligger til grund for udarbejdelsen. Det drejer sig om relevant vejledning fra Miljøstyrelsen samt EU-kommissionens vejledning ”Guidance document No. 27 Technical Guidance for Deriving Environmetal Quality Standards”, som har gennemgået opdateringer, hvilket afspejles i databladene i forhold til miljøkvalitetskriteriernes udarbejdelse, samt for hvilke matricer (vand, sediment og biota), der er fastsat miljøkvalitetskriterier for.

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Late lessons from early warnings: science, precaution, innovation

Summary

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European Environment Agency

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Contents

A full version of the report can be found at:
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Preface

An investment in knowledge pays the best interest
— Benjamin Franklin '*The Way to Wealth*' (1758).

There is something profoundly wrong with the way we are living today. There are corrosive pathologies of inequality all around us — be they access to a safe environment, healthcare, education or clean water. These are reinforced by short-term political actions and a socially divisive language based on the adulation of wealth. A progressive response will require not only greater knowledge about the state of the planet and its resources, but also an awareness that many aspects will remain unknown. We will need a more ethical form of public decision-making based on a language in which our moral instincts and concerns can be better expressed. These are the overall aims of Volume 2 of *Late lessons from early warnings*.

Volume 1 of *Late lessons from early warnings* was published at a time when the world was experiencing an economic slowdown, China had joined the World Trade Organization and western Europe was still a 15-member Union. Global grain production had declined for the third time in four years due mainly to droughts in North America and Australia, and the world saw major recalls of contaminated meat, foot and mouth disease and bovine spongiform encephalopathy (mad cow disease). Global temperatures continued to climb and many bird populations were in decline, but the United States of America had rejected the Kyoto Protocol. We were seeing ourselves through the lens of the first human genome sequence, yet we were trying to manage chemicals known to be harmful to humans and ecosystems, through international conventions and treaties such as the Basel Convention to deal with toxic waste dumping in the developing world; the OSPAR/HELCOM Conventions to reduce the discharges, emissions and the loss of hazardous substances into the sea and the Montreal Protocol, to phase out ozone-depleting substances. The destruction of the World Trade Center had just happened.

Since then, we have witnessed a period of extraordinary hubris. Most visibly, the financial profligacy of the first decade of the century led inexorably to the crises of 2007–2009 whereby the major components of the international financial system were weakened to the extreme by indebtedness, mispriced products, lax monetary policies and mis-engineered protection against risks and uncertainty. The world experienced more not less volatility. Political systems became silted up by vested interests and a determination by citizens to protect assets accumulated in easier times, and beneath it all lay a deeper environmental crisis epitomised by climate change and biodiversity loss.

There was also a collapse of trust, not only in financial institutions but in big companies, as they abandoned staff, pensions and health care schemes. Recent evidence from social psychology has shown that despite rising levels of education and innovation in products and services, people trust only those they know and not strangers. As Stephen Green said in *Good value: reflections on money, morality, and an uncertain world* in 2009:

> 'There has been a massive breakdown of trust: trust in the financial system, trust in bankers, trust in business and business leaders, trust in politicians, trust in the media, trust in the whole process of globalisation — all have been severely damaged, in rich countries and poor countries alike'.

The scientific elites have also been slowly losing public support. This is in part because of the growing number of instances of misplaced certainty about the absence of harm, which has delayed preventive actions to reduce risks to human health, despite evidence to the contrary.

Suddenly, our problems have grown into what Charles W. Churchman in 1967 termed *wicked problems* — difficult or impossible to solve because of incomplete, contradictory and changing requirements, difficult to recognize, resistant to resolution because of the complexity of their interdependencies and needing to be tackled not by one but via many forms of social power. Solving

them requires a new combination of hierarchical power, solidarity and individualism.

What could this mean, for example, for the 100 thousand chemicals currently in commercial use?

To begin with we have more conventions and treaties in place than a decade ago: the 2004 Rotterdam Convention on the Prior Informed Consent (PIC) Procedure covering international trade of 24 pesticides, four severely hazardous pesticide formulations and 11 industrial chemicals; the 2004 Stockholm Convention on Persistent Organic Pollutants to protect human health and the environment from substances which are highly toxic, persistent, bio-accumulative and move long distances in the environment, such as DDT, PCBs, various industrial chemicals, and a set of unintentional chemical by-products such as dioxin. But these conventions only address the top-down hierarchical approach to power.

At the same time Europe has put in place legislation to achieve a global regulatory influence including the EU Cosmetic Directive banning the use of chemicals known or strongly suspected of being carcinogens, reproductive toxins, or mutagens causing cancer, mutation or birth defects; the EU Restriction of Hazardous Substances Directive, which restricts the use of hazardous materials in the manufacture of various types of electronic and electrical equipment including lead, mercury, cadmium, hexavalent chromium, the flame retardents polybrominated biphenyls and polybrominated diphenyl ethers, and which encourages the substitution to safe/or safer alternatives in the electric and electronic equipment industry; the closely linked 2006 EU Waste Electrical and Electronic Equipment Directive for collection, recycling and recovery of electrical goods; the 2006 Strategic Approach to International Chemicals Management (SAICM); and the 2007 EU Registration, Evaluation and Authorisation of Chemicals, widely known as REACH, to assign greater responsibility to industry to manage the risks from chemicals and to provide safety information on substances. The effects of these regulatory tools are described in different chapters, but once again point to the main economic actors rather than communities or individuals.

One thing that has become clearer over the past decade is that certain chemical substances are highly stable in nature and can have long-lasting and wide ranging effects before being broken down into a harmless form. The risk of a stable compound is that it can be bio-accumulated in fatty tissues at concentrations many times higher than in the surrounding environment. Predators, such as polar bears, fish and seals, are known to bio-magnify certain chemicals in even higher concentrations with devastating consequences for both humans and ecosystems. So exposure to toxic chemicals and certain foodstuffs are at risk of causing harm, especially to vulnerable groups such as foetuses in the womb or during childhood when the endocrine system is being actively built. Even with small dose exposures, the consequences can in some instances be devastating with problems ranging from cancer, serious impacts on human development, chronic diseases and learning disabilities. Here the power to act could be more properly set by well-informed individuals and communities.

The relationship between knowledge and power lies at the heart of Volume 2. In many chapters, the implicit links between the sources of scientific knowledge about pollutants, changes in the environment and new technologies, and strong vested interests, both economic and paradigmatic, are exposed. A number of authors also explore in greater depth, the short-sightedness of regulatory science and its role in the identification, evaluation and governance of natural resources, physical and chemical hazards. By creating a better understanding of these normally invisible aspects, it is hoped that this volume will enable communities and people to become more effective stakeholders and participants in the governance of innovation and economic activities in relation to the associated risks to humans and the planet.

Much of what we are able to learn from the histories of past environmental and public health mistakes is also directly applicable to the better regulation and governance of global institutions and financial and economic risks. Robin G. Collingwood argued in his *Autobiography* (1939), that:

> 'History can offer something altogether different from [scientific] rules, namely insight. The true function of insight is to inform people about the present…we study history in order to see more clearly into the situation in which we are called upon to act… the plane on which, ultimately, all problems arise is the plane of 'real' life: that to which they are referred for their solution is history.'

In this volume, we go further. Whilst still drawing lessons from such widely accepted tragedies as leaded petrol, mercury poisoning in Japan's

Minamata Bay and older pesticides which sterilised many men who used it, we have ventured into the uncertainties of potential yet contested harm, from genetically modified products; nanotechnologies; chemicals such as Bisphenol A; new pesticides and mobile phones. There is also an examination of the 80 or so potential 'false positives' where there had been indications of harm but where it was subsequently claimed that there were in fact no risks to prevent: these cases too can provide information that can help to improve future decision-making about innovation and emerging technologies.

A major part of effective decision-making lies in the way issues are framed. In the case of climate change, the first order question is whether it is worth worrying about at all. US Vice President Al Gore chose to make the question a matter of choice between believers and sceptics. However, problems arose when the public was asked to make a scientific decision when too few people had the qualifications to make any kind of reasoned judgement. They were in fact asked to make a false choice. Instead the question should have been framed around which areas should people and governments make decisions and which should be delegated to experts.

In the end there are few certain and enduring truths in the ecological and biological sciences, nor in the economics, psychologies, sociologies and politics that we use to govern them. One, however, comes from the work of Elinor Ostrom, a late and widely missed colleague, who showed from her work on managing fisheries and ecosystems that complex problems can be solved if communication is transparent and open, visions are shared, trust is high and communities are activated to work from the bottom-up as well as from the top down.

As we navigate the Anthropocene, the epoch named in recognition of our impact on the planet, we will need to encourage more people to become involved in solving the wicked problems of our times. Whether through gathering local information or becoming more aware of the many uncertainties and unpredictabilities in our surroundings, the power structures of knowledge will need to change. And if we are to respond more responsibly to the early warning signals of change, we will need to re-design our style of governance to one which reflects a future defined by the local and specific rather than only the global and the average. We hope that Volume 2 of *Late lessons from early warnings* with its many lessons and insights can help us all meet such a challenge.

Professor Jacqueline McGlade,
Executive Director

1 Introduction

Why further late lessons from early warnings?

The 2013 *Late lessons from early warnings* report is the second of its type produced by the European Environment Agency (EEA) in collaboration with a broad range of external authors and peer reviewers.

Volume 1 of *Late lessons from early warnings: the precautionary principle 1896–2000* published in 2001, looked at the history of a selection of occupational, public health and environmental hazards and asked whether we could have been better at taking action early enough to prevent harm. Twelve key lessons for better decision-making were drawn from cases where public policy was formulated against a background of scientific uncertainty and 'surprises' — and where clear evidence of hazards to people and the environment was often ignored (see box on page 11).

The 14 case studies and 12 key lessons from the 2001 report remain highly pertinent today, and underline four main reasons for a second report. The first relates to expanding the late lessons approach to consider long-known, important additional issues with broad societal implications such as lead in petrol, mercury, environmental tobacco smoke and DDT, as well as issues from which lessons have emerged more recently such as the effects of the contraceptive pill on feminisation of fish and the impacts of insecticides on honeybees.

The second concerns filling an acknowledged gap in the 2001 report, by analysing the issue of false positives where government regulation was undertaken based on precaution but later turned out to be unnecessary. Most of the cases examined in the *Late lessons from early warnings* reports are 'false negatives' — instances where early warnings existed but no preventive actions were taken.

The third reason is to address the rapid emergence of new society-wide challenges such as radiation from mobile phones, genetically-modified products, nanotechnologies and invasive alien species as well as if, how and where precautionary actions can play a role.

The final reason relates to how precautionary approaches can help manage the fast-changing, multiple, systemic challenges the world faces today, what new insights can be drawn in this context and how these can underpin opportunities for sustainable innovations and, supported by information technologies, greater public participation in their selection.

Overall approach

As for Volume 1, the approach in Volume 2 has been to include a wide range of relevant case studies produced by external authors along with chapters written by members of the report's editorial team (see acknowledgements section for details). The relevant topics for case study treatment were selected on the basis of advice from the editor, in collaboration with the editorial team and an advisory board, members of the EEA Scientific Committee and the Collegium Ramazzini ([1]).

The chapters in Volume 2 are grouped into five parts: A. Lessons from health hazards; B. Emerging lessons from ecosystems; C. Emerging issues; D. Costs, justice and innovation; and E. Implications for science and governance.

The chapters have been written by authors who, to varying degrees, have had substantial involvement in the subject area being addressed. Indeed they would not have been approached if

([1]) The Collegium Ramazzini is an independent, international academy founded in 1982 by Irving J. Selikoff, Cesare Maltoni and other eminent scientists. Its mission is to advance the study of occupational and environmental health issues and to be a bridge between the world of scientific discovery and the social and political centers, which must act on the discoveries of science to protect public health.

they had not already extensively studied the case that they were asked to write about. All of them, as respected experts in their fields and in line with their professional scientific training, were expected to be as objective as possible in answering the questions put to them by EEA. To support this, and to develop consistency between chapters, the authors were provided with seven structuring questions to be followed when building their chapter.

The case studies have been peer-reviewed by recognised experts in the respective fields who gave of their time freely and provided their feedback within a set of editorial guidelines provided by the EEA.

Scope

The report has been designed, structured and written in order to, inter alia, help politicians, policymakers and the public to:

i understand better the ways in which **scientific knowledge** is financed, created, evaluated, ignored, used and misused in taking timely and precautionary decisions about how to reduce harms, whilst stimulating benign innovations and generating useful employment;

ii learn from some **very expensive 'mistakes' in the past** so as to help societies make fewer mistakes now, and in the future, especially with some of the relatively new, largely unknown, yet already widespread technologies like nanotechnology and mobile phones;

iii be aware of less visible, important factors such as the skewed ways in which the **costs of actions and inactions** for hazardous technologies have been estimated, and the role that **some businesses** have played in ignoring early warnings and in manufacturing doubt about the science supporting such warnings;

iv consider how the law, or administrative arrangements, could be better used to deliver **justice, to those people (and ecosystems) that have been, or could be, harmed** by poorly designed, or badly deployed, innovations;

v explore how best to **engage the public** in helping to make **strategic choices over innovations**, and their technological and social pathways, as well as their involvement in **ecosystems management** and in long term monitoring through **citizen science**.

Part A of the report commences with an analysis of 'false positives' showing that these are few and far between as compared to false negatives and that carefully designed precautionary actions can stimulate innovation, even if the risk turns out not to be real or as serious as initially feared. The remaining nine chapters address false negatives — lead in petrol, perchlorethylene contaminated water, Minamata disease, occupational beryllium disease, environmental tobacco smoke, vinyl chloride, dibromochloropropane (DBCP), Bisphenol A and dichlorodiphenyltrichlorethane (DDT) — from which three common themes emerge: there was more than sufficient evidence for much earlier action; slow and sometimes obstructive behaviour by businesses whose products endangered workers, the public and the environment; and the value of independent scientific research and risk assessments.

Part B focuses on emerging lessons from the degradation of natural systems and their wider implications for society — booster biocides, the pill and the feminisation of fish, climate change, floods, insecticides and honeybees as well as ecosystem resilience more broadly. It considers, like its predecessor, the issues of scientific evidence as the basis for action/inaction, the multiple, often complex factors and feedback loops in play, many of which are not fully understood, as well as the interfaces between science, policy and society and how all actors can move together towards necessary actions in the context of heightened systemic risks, and substantial unknowns.

Part C analyses some newly emerging and large-scale products, technologies and trends, which potentially offer many benefits but also potentially much harm to people and ecosystems and thereby ultimately economic development. Cases addressed include the Chernobyl and Fukushima nuclear accidents; genetically modified agricultural crops and agroecology; the growing threat of invasive alien species; mobile phones and the risk of brain tumours; and nanotechnologies. There is often little science, and very little direct hindsight, to assist in the management of these emerging technologies but the lessons from the historical case studies need to be applied if hazards are to be avoided.

The evidence from the chapters in Part C is that, by and large, societies are not making the most use of the costly lessons that can be gleaned from their histories. A key question is how this can be improved given the many reasons identified from the case studies why taking actions have been delayed including: the novel

and challenging nature of the issues themselves; poorly or inconsistently evaluated information; strong opposition by the corporate and scientific establishments of the day; and the tendency by the decision making institutions, practices and cultures to favour the status quo and the short term perspective. This section also illustrates the value of bottom up as well as top down approaches to innovations in ensuring that the directions of technological pathways, the equitable distributions of benefits, costs and knowledge ownership, and the diversity of locally sensitive technological options are relevant to the food, energy and ecosystems crises.

The historical chapters illustrate numerous harms which for the most part have been caused by irresponsible corporations. This fact, coupled with shortcomings in how decisions are made by governments on when to act on early warnings, and in the law when it comes to compensating victims of harm, are analysed in three chapters in Part D of the report. Each chapter analyses the reasons behind prevailing practice and then goes on to offer insights, for example, on how cost calculation methods can be improved; on how insurance schemes could be used to compensate future victims of harm; and on the reasons why businesses frequently ignore early warnings.

The cases in Parts A–D form the basis for considering in Part E the governance implications for science, public policy and public engagement, and how current practices could be improved to enable society to maximise the benefits of innovations while minimising harms. The main insights are that science could be more relevant for precautionary decision-making; that the wider use of the precautionary principle can avert harm and stimulate innovation; and that the late lessons of history and precautionary approaches are highly pertinent to today's multiple and inter-connected crises — such as those arising from finance, economics, the use of ecosystems, climate change, and the use and supply of energy and food.

Finally, many of the historical and recent case studies illustrate the value of engaging the public in broadening the knowledge base and stimulating robust innovations.

Twelve late lessons

Based on the case studies of Volume 1 of *Late lessons from early warnings* (EEA, 2001), twelve key lessons for better decision-making were drawn:

1	Acknowledge and respond to ignorance, as well as uncertainty and risk, in technology appraisal and public policymaking
2	Provide adequate long-term environmental and health monitoring and research into early warnings
3	Identify and work to reduce 'blind spots' and gaps in scientific knowledge
4	Identify and reduce interdisciplinary obstacles to learning
5	Ensure that real world conditions are adequately accounted for in regulatory appraisal
6	Systematically scrutinise the claimed justifications and benefits alongside the potential risks
7	Evaluate a range of alternative options for meeting needs alongside the option under appraisal, and promote more robust, diverse and adaptable technologies so as to minimise the costs of surprises and maximise the benefits of innovation
8	Ensure use of 'lay' and local knowledge, as well as relevant specialist expertise in the appraisal
9	Take full account of the assumptions and values of different social groups
10	Maintain the regulatory independence of interested parties while retaining an inclusive approach to information and opinion gathering
11	Identify and reduce institutional obstacles to learning and action
12	Avoid 'paralysis by analysis' by acting to reduce potential harm when there are reasonable grounds for concern

Source: EEA, 2001, *Late lessons from early warnings: the precautionary principle 1986–2000,* Environmental issues report No 22, European Environment Agency.

2 The precautionary principle and false alarms — lessons learned

Steffen Foss Hansen and Joel A. Tickner

Most of the cases examined in the *Late lessons from early warnings* reports are 'false negatives' — instances where early warnings existed but no preventive actions were taken. In debates surrounding the precautionary principle it is often claimed that widespread application of the principle will lead to a large number of regulatory false positives — over-regulation of minor risks and regulation of non-existent risks, often due to unwarranted public 'fears'. Understanding and learning from past false positives as well as false negatives is essential for improving decision-making about public health and the environment.

This chapter reviews incidents of 'false positives', where government regulation was undertaken based on precaution but later turned out to be unnecessary. In total 88 cases were identified to be alleged false positives, however, following a detailed analysis most of them turned out to be either real risks, or cases where 'the jury is still out', or unregulated alarms, or risk-risk trade-offs, rather than false positives.

The analysis revealed four regulatory false positives: US swine flu, saccharin, food irradiation, and Southern leaf corn blight. Numerous important lessons can be learned from each, although there are few parallels between them in terms of when and why each risk was falsely believed to be real. This is a lesson in itself: each risk is unique, as is the science and politics behind it and hence a flexible approach is therefore needed, adapted to the nature of the problem. The costs of the false positives identified were mainly economic, although the actions taken to address swine flu in 1976 did lead to some unintended deaths and human suffering, and diverted resources from other potentially serious health risks. Determining the net costs of mistaken regulatory action, however, requires a complete assessment of the impacts of the regulation, including the costs and benefits of using alternative technologies and approaches.

Overall, the analysis shows that fear of false positives is misplaced and should not be a rationale for avoiding precautionary actions where warranted. False positives are few and far between as compared to false negatives and carefully designed precautionary actions can stimulate innovation, even if the risk turns out not to be real or as serious as initially feared. There is a need for new approaches to characterising and preventing complex risks that move debate from the 'problem' sphere to the 'solutions' sphere. By learning from the lessons in this chapter, more effective preventive decisions can be made in the future.

The scarcity of genuine false positives compared to the large number of 'mistaken false positives' could partly be the result of a deliberate strategy in risk communication. Several references and leaked documents have shown that some regulated parties have consciously recruited reputable scientists, media experts and politicians to call on if their products are linked to a possible hazard. Manufacturing doubt, disregarding scientific evidence of risks and claiming over-regulation appear to be a deliberate strategy for some industry groups and think tanks to undermine precautionary decision-making.

3 Lead in petrol 'makes the mind give way'

Herbert Needleman and David Gee

This chapter addresses the widespread use of lead in petrol. It focuses on the period 1925–2005, when leaded petrol was first widely marketed in the US and then spread to the rest of the world before being gradually phased out from the 1970s. In Europe, the Aarhus Protocol (www.unece.org/env/pp/treatytext.html) initiated the phase-out of leaded petrol in the period 1998–2005.

The neurotoxic effects of lead were recognised as far back as Roman times. And in 1925, at the 'one day trial' of leaded petrol in the US, many experts warned of the likely health impacts of adding lead to petrol. Yet, despite the availability of an equally effective alcohol additive which was assessed by experts to be cleaner, the leaded route to fuel efficiency was chosen in the US and then exported to the rest of the world.

For several decades after the introduction of leaded petrol, virtually no independent research was carried out and the main source of information was industry and industry-sponsored researchers. Not until the 1960s and 1970s did independent scientists from outside this group show, for example, that body burdens of lead arising from human activities were not 'normal', as industry claimed, but were hundreds of times higher than before the industrial revolution and were therefore likely to be harmful.

At its peak in the mid-1970s, leaded petrol released about 200 000 tonnes of lead into the atmosphere annually in both the US and Europe. Following the subsequent phase-out, blood lead levels in children (the most sensitive group exposed) quickly fell, in line with the decrease in air concentrations. The lessons nevertheless remain relevant globally today. Although nearly all countries worldwide had phased out leaded petrol by 2012, lead concentrations in soils and sediments remain high. Meanwhile, electronic wastes containing lead and other contaminants also cause elevated blood lead levels.

Supplementary panel texts focus on the events leading up to the US choice of leaded petrol as the primary fuel source in 1925 and more recent accounts of EU policymaking on lead in petrol and the road to phase-outs in Germany and the United Kingdom.

4 Too much to swallow: PCE contamination of mains water

David Ozonoff

PCE (perchlorethylene, also known as 'perc' or tetrachloroethylene), was used in the production of plastic linings for drinking water distribution pipes in the late 1960s and 1970s. This new and relatively untested type of distribution pipe was used in over 700 miles of New England's water distribution systems. Not until 1976 was it discovered that PCE had been leaching into the water from the pipe lining, causing widespread contamination of water supplies that still today require continuous remediation.

Before the pipes were put into production there was a substantial amount of scientific information available about the potential hazards of PCE. This did not include current concerns about PCE's carcinogenicity, teratogenicity and other health consequences of relatively low-level exposure upper most among today's concerns, but many early warnings suggested the need for caution in introducing PCE-based mains pipe linings.

PCE had been used to treat hookworm and data on side effects were in the literature, while later a variety of occupational users were studied, including aircraft workers, small companies in countries where biological monitoring was required, and dry-cleaning firms. Several environmental studies were also conducted to see if drinking water contaminated with PCE or its close relative, TCE (trichloroethylene), was associated with cancer. Results were mixed and the chemical industry consistently denied that PCE was a human carcinogen.

This case study explores the early (pre 1970) history researching the toxicity of the chemical. It also focuses on the failure of one manufacturer, Johns-Manville Corporation, to recognise the warning signals about using a suspected toxic substance. It examines why a new product was deployed without thought to the public health consequences and why evidence of the potential hazard was ignored.

The science has not been hidden. It has been ineffective in guiding and catalysing action. Whether the problem is a failed duty of care or a lack of clarity about what evidence will trigger action, the contemporary argument over how to interpret the scientific evidence is irresolvable within science itself. There are no overarching criteria from the philosophy of science that can dictate a solution.

This chapter also includes two supplementary texts. A panel that analyses the differences between the conclusions of risk assessments based on the same data, focusing in particular on assessments of PCE and TCE. A further panel describes the opportunities to switch to wet-cleaning technologies to reduce the current use of PCE in dry cleaning.

5 Minamata disease: a challenge for democracy and justice

Takashi Yorifuji, Toshihide Tsuda and Masazumi Harada

Minamata disease, which can induce lethal or severely debilitating mental and physical effects, was caused by methylmercury-contaminated effluent released into Minamata Bay by Chisso, Japan's largest chemical manufacturer. It resulted in widespread suffering among those who unknowingly ate the contaminated fish. This chapter documents the story in three phases.

The disease first came to prominence in the 1950s. It was officially identified in 1956 and attributed to factory effluent but the government took no action to stop contamination or prohibit fish consumption. Chisso knew it was discharging methylmercury and could have known that it was the likely active factor but it chose not to collaborate and actively hindered research. The government concurred, prioritising industrial growth over public health. In 1968 Chisso stopped using the process that caused methylmercury pollution and the Japanese government then conceded that methylmercury was the etiologic agent of Minamata disease.

The second part of the story addresses the discovery that methylmercury is transferred across the placenta to affect the development of unborn children, resulting in serious mental and physical problems in later life. Experts missed this at first because of a medical consensus that such transfer across the placenta was impossible.

The third phase focuses on the battle for compensation. Initially, Chisso gave token 'sympathy money' under very limited criteria. In 1971 the Japanese government adopted a more generous approach but after claims and costs soared a more restrictive definition was introduced in 1977, justified by controversial 'expert opinions'. Legal victories for the victims subsequently made the government's position untenable and a political solution was reached in 1995–1996. In 2003, the 'expert opinions' were shown to be flawed and the Supreme Court declared the definition invalid in 2004.

In September 2011 there were 2 273 officially recognised patients. Still, the continuing failure to investigate which areas and communities were affected means that the financial settlement's geographic and temporal scope is still not properly determined. Alongside deep-seated issues with respect to transparency in decision-making and information sharing, this indicates that Japan still faces a fundamental democratic deficit in its handling of manmade disasters.

This chapter is followed by three short updates on the effects of mercury poisoning since Minamata; on attempts to contain it, including the 2009 global agreement to phase mercury out of economic activity; and on the need for better information about contaminant exposures to enable policymakers to make informed choices that balance the benefits of fish consumption against the assumed adverse effects of low-level methylmercury exposures.

6 Beryllium's 'public relations problem'

David Michaels and Celeste Monforton

Scores of workers employed in nuclear weapons production have been diagnosed with chronic beryllium disease (CBD), a progressive and irreversible inflammatory lung disease. This chapter presents a history of knowledge and public policy about preventing beryllium-related disease, focusing primarily on the United States beryllium industry's role in shaping US regulatory policy.

Over several decades increasingly compelling evidence accumulated that CBD was associated with beryllium exposure at levels below the existing regulatory standard. The beryllium industry had a strong financial incentive to challenge the data and decided to be proactive in shaping interpretation of scientific literature on beryllium's health effects. It hired public relations and 'product defence' consulting firms to refute evidence that the standard was inadequate. When the scientific evidence became so great that it was no longer credible to deny that workers developed CBD at permitted exposure levels, the beryllium industry responded with a new rationale to delay promulgation of a new, more protective exposure limit.

This case study underscores the importance of considering the hazards from toxic materials throughout the entire product life cycle. While primary producers of beryllium products may be able to control exposures in their own facilities, it is unlikely that many secondary users and recyclers have the expertise, resources and knowledge necessary to prevent beryllium disease in exposed workers and residents in nearby communities.

The primary lessons of this chapter are widely applicable to many environmental health controversies. In particular, it illustrates the practice of 'manufacturing uncertainty' — a strategy used by some polluters and manufacturers of hazardous products to prevent or delay regulation or victim compensation.

This chapter is followed by an analysis of the rationale for corporate behaviour in the regulation of beryllium. It is argued that the availability of occasional and limited opportunities for companies to change course without suffering onerous consequences would encourage them to rethink their position and create an obligation on shareholders to take the responsible course. Although this may be perceived as letting them 'get away with it', the end result may be better public policy and corporate responsibility.

7 Tobacco industry manipulation of research

Lisa A. Bero

This chapter differs in some ways from the others in Volume 2 of *Late lessons from early warnings*. The history of 'second hand', 'passive' or 'environmental tobacco smoke' (ETS), to which non-smokers are exposed overlaps with the history of active smoking. Those affected include the partners and children of smokers, and the bartenders and other workers who have to work in smoky environments.

The focus in this chapter is on the strategies used by the tobacco industry to deny, downplay, distort and dismiss the growing evidence that, like active smoking, ETS causes lung cancer and other effects in non-smokers. It does not address the history of scientific knowledge about tobacco and how it was used or not used to reduce lung cancer and other harmful effects of tobacco smoke. There is much literature on this and a table at the end of the chapter summarises the main dates in the evolution of knowledge in this area.

The chapter concentrates on the 'argumentation' that was used to accept, or reject, the growing scientific evidence of harm. Who generated and financed the science used to refute data on adverse health effects? What were the motivations? What kind of science and information, tools and assumptions were used to refute data on the adverse health of tobacco?

The release of millions of internal tobacco industry documents due to law suits in the US has given insights into the inner workings of the tobacco industry and revealed their previously hidden involvement in manipulating research. However, this insight is not available for most corporate sectors. The chapter discusses the possibilities of 'full disclosure' of funding sources and special interests in research and risk assessment in order to secure independence and prevent bias towards particular viewpoints.

While smoking bans are now being introduced in more and more countries, other industries are drawing inspiration from tobacco company strategies, seeking to maintain doubt about harm in order to keep hazardous products in the marketplace.

The chapter also includes a summary of the tobacco industry's role in shaping risk assessment in the US and Europe to serve its own interests.

8 Vinyl chloride: a saga of secrecy

Morando Soffritti, Jennifer Beth Sass, Barry Castleman and David Gee

This chapter is about how early warnings in the 1950s and 1960s concerning the short-term harm of vinyl chloride (VC) to the skin and bones of workers, and to the livers of laboratory animals, were initially hidden from other workers and regulators. This was despite some early misgivings by company experts whose advice was initially ignored by their employers. This pattern was repeated when the later, more devastating news of a rare liver cancer in workers was revealed by long-term animal studies and by an attentive and concerned company physician.

Unlike many other histories, however, this story features a very prompt response from the global chemical industry to the publication of the liver cancer evidence, a response that included funding cancer testing and later compliance with a large reduction in the permissible exposure limits. The case also provides early evidence of reproductive effects of vinyl chloride monomer (VCM).

Other features of this story presage the later and common responses of the corporate world to heightened public awareness and pressure from non-governmental organisations (NGOs) and trade unions, including greatly exaggerated estimates of the likely costs of complying with tighter pollution controls; a frequent mismatch between the position of the trade association and that of many, more progressive companies within the association; but also some relatively quick corporate responses to public, NGO and regulatory pressure.

The chapter also features two legal aspects, which, though more common in the US, are also valuable for Europeans. First, the potentially positive role that judicial review of regulatory proposals can play in providing a societal judgement about the behaviour of corporations. This can embrace not just moral judgements but also judgements about the state of the science and what society should do with it.

Second, the role that document discovery in legal compensation cases can play in revealing the real and until then secret activities of corporations. Any proposals to promote justice for victims of environmental and health harms via no fault administrative arrangements need to be accompanied by other measures to extract information about corporate behaviour.

The chapter is followed by a panel analysing the value of animal testing for identifying carcinogens.

9 The pesticide DBCP and male infertility

Eula Bingham and Celeste Monforton

Dibromochloropropane (DBCP) is a pesticide used against nematodes (roundworms or threadworms) that damage pineapples, bananas and other tropical fruits. It was introduced into US agriculture in 1955 and approved for use as a fumigant in 1964. By 1961 laboratory experiments had shown that it made the testicles of rodents shrink and significantly reduced the quantity and quality of sperm. Nonetheless, the compound was widely marketed and became a commercial success.

In 1977, workers at a production plant became worried that they were unable to father children. An emergency study by a US government agency discovered that in many cases the workers were suffering from deficient or absent sperm. While controls were improved at US facilities, the product continued to be marketed and sprayed in Latin America, the Philippines, some African countries, and elsewhere.

By the 1990s, tens of thousands of plantation workers in these countries had allegedly suffered adverse reproductive effects from DBCP use.

The story continues today with contentious legal claims for compensation, contamination of drinking water and industry attempts to prevent a Swedish documentary on the issue from being screened.

This chapter looks at the knowledge available about the hazards and the actions taken, or not taken, to avert them. The DBCP story is significant as it is the first clear example of reproductive damage to workers who manufactured and used a synthetic chemical. This is one of many examples supporting the growing concerns about increasing rates of reproductive and developmental disease, and about the endocrine disrupting chemicals that seem to be playing a role in these disorders.

Protecting production workers, users, consumers and the environment from chemicals that may damage reproduction demands closer integration of scientific disciplines, as well as government action. The lessons of DBCP may help in ensuring timely protection from harm, based on precautionary approaches to scientific evidence.

10 Bisphenol A: contested science, divergent safety evaluations

Andreas Gies and Ana M. Soto

Bisphenol A (BPA) is currently one of the world's best-selling chemicals and primarily used to make polycarbonate plastics. It is widely used in common products such as baby bottles, household electronics, medical devices and coatings on food containers. BPA is known to mimic the female hormone oestrogen and has been found to leach from the materials where it is used.

Studies have suggested that even exposure to low doses of BPA may cause endocrine disrupting effects. As with other hormones, it appears that an organism is most sensitive during development but that effects are often not observed until much later in the lifecycle. This means that at the time when the effects become detectable, the chemical exposure has vanished. This makes it extremely difficult to link exposure to effects in humans.

This chapter maps some of the findings in studies of rodents and humans. It also discusses the challenges of evaluating scientific findings in a field where industry-sponsored studies and independent scientific research seem to deviate strongly. The authors offer suggestions for ways to uncouple financial interests from scientific research and testing.

A widely used and dispersed industrial chemical like Bisphenol A is a controversial example of an endocrine disrupting substance that has implications for policymakers. Different approaches to risk assessment for BPA by US and European authorities are presented. It throws light on the ways in which similar evidence is evaluated differently in different risk assessments and presents challenges for applying the precautionary principle.

The intense discussion and scientific work on BPA have slowly contributed to a process of improving test strategies. While traditional toxicology has relied on a monotonic increasing dose-response relationship as evidence that the effect is caused by the test agent, studies on BPA and other endocrine disruptor chemicals (EDCs) have demonstrated the limitations of this approach and adjustments have been made in some cases.

It has also been widely accepted that effects cannot be predicted by simply thinking of BPA as a weak oestrogen and extrapolating from what is observed for more potent endogenous oestrogens. This lesson is particularly evident in the intense pharmaceutical interest in selective oestrogen response modifiers (SERMs).

The chapter is followed by a panel analysing the value of animal testing for identifying carcinogens.

11 DDT: fifty years since *Silent Spring*

Henk Bouwman, Riana Bornman, Henk van den Berg and Henrik Kylin

'There was a strange stillness. The birds for example — where had they gone? Many people spoke about them, puzzled and disturbed. The feeding stations in the backyards were deserted. The few birds seen anywhere were moribund: they trembled violently and could not fly. It was a spring without voices ... only silence lay over the fields and woods and marsh.'

The book *Silent Spring* by Rachel Carson is mainly about the impacts of chemicals (in particular dichlorodiphenyltrichlorethane also known as DDT) on the environment and human health. Indeed, the close association between humans and birds remains very apt. Representing the only two warm-blooded groups of life on Earth, mammals and birds share the same environments and threats.

Carson's claim that she lived in 'an era dominated by industry, in which the right to make a dollar at whatever cost is seldom challenged' still resonates strongly with the problems that societies face all over the world. One chapter heading, 'The obligation to endure', derived from the French biologist and philosopher Jean Rostand's famous observation that, 'the obligation to endure gives us the right to know'. United States President John F. Kennedy responded to the challenge posed by Carson by investigating DDT, leading to its complete ban in the US. The ban was followed by a range of institutions and regulations concerned with environmental issues in the US and elsewhere, driven by public demand for knowledge and protection.

DDT was the primary tool used in the first global malaria eradication programme during the 1950s and 1960s. The insecticide is sprayed on the inner walls and ceilings of houses. Malaria has been successfully eliminated from many regions but remains endemic in large parts of the world. DDT remains one of the 12 insecticides — and the only organochlorine compound — currently recommended by the World Health Organization (WHO), and under the Stockholm Convention on Persistent Organic Pollutants, countries may continue to use DDT. Global annual use of DDT for disease vector control is estimated at more than 5 000 tonnes.

It is clear that the social conscience awakened by Rachel Carson 50 years ago gave momentum to a groundswell of actions and interventions that are slowly but steadily making inroads at myriad levels. Chapter 17 of her book, 'The other road' reminds the reader of the opportunities that should have been seized much earlier. With more than 10 % of bird species worldwide now threatened in one way or another, it is clear that we missed early warnings or failed to act on them. Will we continue to miss signposts to 'other roads'? Are our obligations to endure met by our rights to know? As Carson said 50 years ago: 'The choice, after all, is ours to make.'

12 Booster biocide antifoulants: is history repeating itself?

Andrew R. G. Price and James W. Readman

Tributyltin (TBT) was widely used as an effective antifouling agent in paints for ships and boats until the European Community restricted its use in 1989 because of its proven harm to the environment and shellfisheries. Thereafter, booster biocides were introduced to enhance the performance of antifouling paints. They were believed to be less damaging to aquatic life than TBT. Subsequently, however, it has been established that booster biocides can also create significant environmental risks.

This chapter outlines the background to booster biocide use, the early warnings about their potential physiological and ecological impacts on non-target species, and the actions taken in response. The science that set some alarm bells ringing is described, along with lessons that could influence the future of an industry still searching for less environmentally invasive solutions.

Booster biocide antifouling agents threaten a variety of habitats — from coral reefs and seagrass beds to open moorings — within the EU and globally. Their primarily herbicidal properties mean that coral zooxanthellae, phytoplankton and periphyton are particularly vulnerable. Compared to TBT, an antifouling agent with a quite specific action, booster biocides have more broad-spectrum impacts. The wider ecological effect of shifting to booster biocides remain poorly understood but of considerable concern because they may affect the base of marine food chains.

From a toxicological viewpoint, booster biocides do not threaten to have endocrine disrupting properties similar to TBTs. At current environmental concentrations, however, some can damage primary producers and some are persistent. While legislation has been introduced to control their use, the rigour of regulations varies between countries. These geographical disparities need to be addressed, and future biocidal products and novel approaches to antifouling should be better appraised.

For policymakers, the challenge is to protect non-target biological communities from selective change resulting from booster biocide use. Persistence, bioaccumulative and toxic (PBT) criteria can be used to evaluate the relative potential impact from the available biocides, and consequently target appropriate legislation. Nevertheless, lateral thinking, aiming to identify novel materials and strategies to address antifouling, could pay dividends in the future.

13 Ethinyl oestradiol in the aquatic environment

Susan Jobling and Richard Owen

Many decades of research have shown that when released to the environment, a group of hormones known as oestrogens, both synthetic and naturally occurring, can have serious impacts on wildlife. This includes the development of intersex characteristics in male fish, which diminishes fertility and fecundity. Although often sublethal, such impacts may be permanent and irreversible.

This chapter describes the scientific evidence and regulatory debates concerning one of these oestrogens, ethinyloestradiol (EE2), an active ingredient in the birth control pill. First developed in 1938, it is released to the aquatic environment via wastewater treatment plants. Although it is now clear that wildlife species are exposed to and impacted by a cocktail of endocrine disrupting chemicals, there is also reasonable scientific certainty that EE2 plays a significant role, and at vanishingly low levels in the environment.

In 2004 the Environment Agency of England and Wales accepted this, judging the evidence sufficient to warrant consideration of risk management. In 2012, nearly 75 years after its synthesis, the European Commission proposed to regulate EE2 as a EU-wide 'priority substance' under the Water Framework Directive (the primary legislation for protecting and conserving European water bodies). This proposal was subsequently amended, delaying any decision on a regulatory 'environmental quality standard' until at least 2016.

This is in part because control of EE2 will come at a significant price. Complying with proposed regulatory limits in the environment means removing very low (part per trillion) levels of EE2 from wastewater effluents at considerable expense.

Is this a price we are willing to pay? Or will the price of precautionary action be simply too high — a pill too bitter to swallow? To what extent is society, which has enjoyed decades of flexible fertility and will also ultimately pay for the control and management of its unintended consequences, involved in this decision? And what could this mean for the many thousands of other pharmaceuticals that ubiquitously infiltrate our environment and which could have sublethal effects on aquatic animals at similarly low levels?

14 Climate change: science and the precautionary principle

Hartmut Grassl and Bert Metz

The first scientifically credible early warning about the possible dangers of climate change due to carbon dioxide (CO_2) emissions from burning fossil fuels came in 1897. While the basic physical principles of global warming are simple, however, the more detailed science of climate change is exceedingly complicated. Even now, more than a hundred years since the first early warning, many important details of climate change cannot be predicted with certainty. It is therefore unsurprising that the science of climate change and questions about the true value of burning fossil fuels have fostered sustained scientific and political controversy.

When the first volume of *Late lessons from early warnings* was drafted there appeared to be too much legitimate controversy about climate change for the issue to be included. A case study could have led to arguments that distracted attention from the valuable and robust lessons from more established issues such as asbestos, polychlorinated biphenyls (PCBs), chlorofluorocarbons (CFCs) and the ozone-hole, X-rays and acid rain. This decision was taken despite the then widespread acceptance that 'the balance of evidence suggests a discernible human influence on global climate' (*Contribution of Working Group I to the Second Assessment Report of the Intergovernmental Panel on Climate Change*, IPCC, 1995).

Over a decade later and after two more reviews by the Intergovernmental Panel on Climate Change (IPCC) of a much greater volume of climate change science it seemed appropriate to include climate change in this volume, despite some continuing controversy. The evidence that human activities are having a dangerous impact on the climate has strengthened since 1995. By 2007, the IPCC was able to conclude with 'very high confidence that the global net effect of human activities since 1750 has been one of warming'. Given the size and irreversibility (on human time scales) of many of the harmful effects of human-induced climate change, there is an urgent need for action to reduce CO_2 emissions and other greenhouse gases. Some contrarian views persist, however, as the authors illustrate.

This chapter summarises the history of growing knowledge about human-induced climate change and of the main actions, or inactions that accompanied it. Like many other chapters, it reflects the lifelong commitment of both authors to trying to understand and mitigate the effects of human-induced climate change. It concludes with some lessons and insights that are relevant to many other environmental and health issues.

Also included is a panel text describing how the IPCC's approach to assessing uncertainty evolved between its first to its fifth assessment reports.

15 Floods: lessons about early warning systems

Zbigniew W. Kundzewicz

Floods are an increasingly acute problem. Intense precipitation has become more frequent and more intense, growing manmade pressure has increased the magnitude of floods that result from any level of precipitation, and flawed decisions about the location of human infrastructure have increased the flood loss potential.

Unlike most other case studies presented in this report, this chapter focuses on flooding as a phenomenon and the requirements for effective early warning systems, rather than addressing a particular event and the lessons that can be learned.

Flooding cannot be wholly prevented. The occurrence of a flood need not be considered a 'failure' and, conversely, minimisation of losses may constitute a 'success'. There are lessons to be learned from every flood and it is important to use them in preparing for the next flood. Once we accept that no flood protection measures can guarantee complete safety, a general change of paradigm is needed to reduce human vulnerability to floods. The attitude of 'living with floods' and accommodating them in planning seems more sustainable than hopelessly striving to eradicate them.

Flood forecasting and warning systems fail because links in the chain perform poorly or fail completely. A single weak point in a system that otherwise contains excellent components may render the overall system performance unsatisfactory. A successful system requires sufficient integration of components and collaboration and coordination between multiple institutions.

The chapter deals primarily with the challenges of fluvial (river) floods. It is complemented by three short supplementary texts. The first highlights the complex, dynamic and diverse ecosystems of river floodplains, which are often degraded during construction of flood defences. Despite their huge economic value, near-natural floodplains are among the most threatened ecosystems globally.

The second discusses uncertainties in anticipating rainfall patterns and intensity, and their relationship to flood levels during extreme flows. Such uncertainties present challenges for scientists and decision-makers alike.

The third addresses the increasing risks of coastal flooding due to factors such as climate change and sea-level rise, and reviews European experience with precautionary action.

16 Seed-dressing systemic insecticides and honeybees

Laura Maxim and Jeroen van der Sluijs

In 1994 French beekeepers began to report alarming signs. During summer, many honeybees did not return to the hives. Honeybees gathered close together in small groups on the ground or hovered, disoriented, in front of the hive and displayed abnormal foraging behaviour. These signs were accompanied by winter losses.

Evidence pointed to Bayer's seed-dressing systemic insecticide Gaucho®, which contains the active substance imidacloprid. This chapter presents the historical evolution of evidence on the risks of Gaucho® to honeybees in sunflower and maize seed-dressing in France, and analyses the actions in response to the accumulating evidence regarding these risks.

The social processes that ultimately lead to application of the precautionary principle for the ban of Gaucho® in sunflower and maize seed-dressing are described, with a focus on the ways in which scientific findings were used by stakeholders and decision-makers to influence policy during the controversy.

Public scientists were in a difficult position in this case. The results of their work were central to a social debate with high economic and political stakes. In certain cases their work was not judged according to its scientific merit but based on whether or not it supported the positions of some stakeholders. This situation tested the ability and courage of researchers to withstand pressure and continue working on imidacloprid.

Other European countries also suspended neonicotinoid seed-dressing insecticides. Evidence of the toxicity of neonicotinoids present in the dust emitted during sowing of coated seeds supported such decisions. Most important, the French case highlighted the major weaknesses of regulatory risk assessment and marketing authorisation of pesticides, and particularly neonicotinoids. These insights were recently confirmed by work by the European Food Safety Authority.

From this case study eight lessons are drawn about governance of controversies related to chemical risks. The study is followed by two additional texts. A first panel presents Bayer Crop Science's comments on the analysis in this chapter. A second contains the authors' response to the Bayer comments.

17 Ecosystems and managing the dynamics of change

Jacqueline McGlade and Sybille van den Hove

A decade after Rachel Carson's *Silent Spring* was published, describing the toxic legacy of the twentieth century, Annie Dillard in her Pulitzer prize winning book *Pilgrim at Tinker Creek*, opened up a different way of looking at the world. It presaged a twenty first century in which the global economy would be based on a more thorough understanding of nature, its functioning and material wealth. Wholly descriptive, yet increasingly relevant, her book captured the very essence of what this chapter is about: that amongst the observations which routinely help to predict the evolution of the natural world are the seeds of surprise — surprise of the unusual and surprise as a portent of future change. Our systemic failure to anticipate such surprises forms the core of this chapter. A series of case studies from fisheries, forests, savannah and aquatic systems are used to underline how early warnings about changes in these natural systems emerged but were not used.

The chapter highlights how the division of knowledge into political, disciplinary and geographic silos has led to the 'recurring nightmares' of short-term interests outcompeting long-term vision; situations where competition replaces co-operation; fragmentation of values and interest; fragmentation of authority and responsibility; and fragmentation of information and knowledge leading to inadequate solutions or even additional problems. In addition, the lack of institutional fit has often confounded the effectiveness of the stewardship of ecosystem services, and led to unexpected surprises, excessive rent seeking and high transaction costs.

Using counterfactual thinking (i.e. the dependence of *whether, when* and *how* one event occurs on *whether, when* and *how* another event occurs and the possible alteration of events), built around the four interconnected concepts of *planetary boundaries, tipping points, panarchy* and *resilience*, the chapter provides an analytical lens through which to explore why many of the warning signals were not seen. The chapter concludes by suggesting why ecosystems are likely to be even more at risk in the future and why we will need to observe and interpret the dynamics of both nature and institutions ever more closely if we are to avoid sudden irreversible ecological changes.

18 Late lessons from Chernobyl, early warnings from Fukushima

Paul Dorfman, Aleksandra Fucic and Stephen Thomas

The nuclear accident at Fukushima in Japan occurred almost exactly 25 years after the Chernobyl nuclear accident in 1986. Analysis of each provides valuable late and early lessons that could prove helpful to decision-makers and the public as plans are made to meet the energy demands of the coming decades while responding to the growing environmental costs of climate change and the need to ensure energy security in a politically unstable world.

This chapter explores some key aspects of the Chernobyl and Fukushima accidents, the radiation releases, their effects and their implications for any construction of new nuclear plants in Europe. There are also lessons to be learned about nuclear construction costs, liabilities, future investments and risk assessment of foreseeable and unexpected events that affect people and the environment.

Since health consequences may start to arise from the Fukushima accident and be documented over the next 5–40 years, a key lesson to be learned concerns the multifactorial nature of the event. In planning future radiation protection, preventive measures and bio-monitoring of exposed populations, it will be of great importance to integrate the available data on both cancer and non-cancer diseases following overexposure to ionising radiation; adopt a complex approach to interpreting data, considering the impacts of age, gender and geographical dispersion of affected individuals; and integrate the evaluation of latency periods between exposure and disease diagnosis development for each cancer type.

Given the degree of uncertainty and complexity attached to even the most tightly framed and rigorous nuclear risk assessment, attempts to weight the magnitude of accident by the expected probability of occurrence have proven problematic, since these essentially theoretical calculations can only be based on sets of pre-conditioning assumptions. This is not an arcane philosophical point but rather a very practical issue with significant implications for the proper management of nuclear risk. With its failure to plan for the cascade of unexpected beyond design-base accidents, the regulatory emphasis on risk-based probabilistic assessment has proven very limited. An urgent reappraisal of this approach and its real-life application seems overdue.

Whatever one's view of the risks and benefits of nuclear energy, it is clear that the possibility of catastrophic accidents and consequent economic liabilities must be factored into the policy and regulatory decision-making process. In the context of current collective knowledge on nuclear risks, planned pan-European liability regimes will need significant re-evaluation.

19 Hungry for innovation: from GM crops to agroecology

David A. Quist, Jack A. Heinemann, Anne I. Myhr, Iulie Aslaksen and Silvio Funtowicz

Innovation's potential to deliver food security and solve other agriculture-related problems is high on the agenda of virtually all nations. This chapter looks at two different examples of food and agricultural innovation: genetically modified (GM) crops and agroecological methods, which illustrate how different innovation strategies affect future agricultural and social options.

GM crops are well suited to high-input monoculture agricultural systems that are highly productive but largely unsustainable in their reliance on external, non-renewable inputs. Intellectual property rights granted for GM crops often close down, rather than open up further innovation potential, and stifle investment into a broader diversity of innovations allowing a greater distribution of their benefits.

Science-based agroecological methods are participatory in nature and designed to fit within the dynamics underpinning the multifunctional role of agriculture in producing food, enhancing biodiversity and ecoystem services, and providing security to communities. They are better suited to agricultural systems that aim to deliver sustainable food security than high external input approaches. They do, however, require a broader range of incentives and supportive frameworks to succeed. Both approaches raise the issue of the governance of innovation within agriculture and more generally within societies.

The chapter explores the consequences of a 'top-down transfer of technology' approach in addressing the needs of poor farmers. Here innovation is often framed in terms of economic growth in a competitive global economy, a focus that may conflict with efforts to reduce or reverse environmental damage caused by existing models of agriculture, or even deter investment into socially responsible innovation.

Another option explored is a 'bottom-up' approach, using and building upon resources already available: local people, their knowledge, needs, aspirations and indigenous natural resources. The bottom-up approach may also involve the public as a key actor in decisions about the design of food systems, particularly as it relates to food quality, health, and social and environmental sustainability.

Options are presented for how best to answer consumer calls for food quality, sustainability and social equity in a wide sense, while responding to health and environmental concerns and securing livelihoods in local small-scale agriculture. If we fail to address the governance of innovation in food, fibre and fuel production now, then current indications are that we will design agriculture to fail.

20 Invasive alien species: a growing but neglected threat?

Sarah Brunel, Eladio Fernández-Galiano, Piero Genovesi, Vernon H. Heywood, Christoph Kueffer and David M. Richardson

Biological invasions are one of the five major causes of biodiversity loss as global human travel and trade have moved, and continue to move, thousands of species between and across continents. Some species of alien origin have a high probability of unrestrained growth which can ultimately lead to environmental damage.

An alien species — animal, plant or microorganism — is one that has been introduced, as a result of human activity, either accidentally or deliberately, to an area it could not have reached on its own. A common definition of the term 'invasive' focuses on its (negative) impact, while other definitions consider only rate of spread and exclude considerations of impact.

Despite the growing amount of legislation being adopted at the global scale, biological invasions continue to grow at a rapid rate, with no indication yet of any saturation effect. Decision-making in this area is very challenging. The overall complexity of the problem, its interdisciplinarity, the scientific uncertainties and the large number of stakeholders that need to be informed and involved, together demand governance actions that are difficult to see emerging at the regional scale (as in the EU), let alone globally.

It is widely agreed that preventing biological invasions or tackling them at a very early stage is the most efficient and cost-effective approach. Harmless species can be confused with harmful invasive species, however, leading to a waste of resources. Even more seriously, harmful invaders can be mistaken for innocuous species — so-called 'invaders in disguise' — and no appropriate action may be taken to counter the threats they pose.

Even with a very good risk assessment system, new outbreaks of invasive alien species could still occur, necessitating a system of rapid early warning and effective eradication response. The decision on where to draw the line on the acceptable environmental risks versus the introduction of new species or new communities that may carry invasive alien species then becomes a value judgement.

There is lively debate within the scientific community regarding the most appropriate strategies for managing invasive alien species. Governments and institutions charged with making decisions have access to considerable knowledge on the topic, but the lack of rules of interactions between multiple parties regularly thwarts effective decision-making.

21 Mobile phones and brain tumour risk: early warnings, early actions?

Lennart Hardell, Michael Carlberg and David Gee

In 2011 the World Health Organization's International Agency for Research on Cancer (IARC) categorised the radiation fields from mobile phones and other devices that emit similar non-ionizing electromagnetic fields (EMFs), as a Group 2B i.e. 'possible' human carcinogen. Nine years earlier IARC gave the same classification to the magnetic fields from overhead electric power lines.

The IARC decision on mobile phones was principally based on two sets of case-control human studies of possible links between mobile phone use and brain tumours: the IARC Interphone study and the Hardell group studies from Sweden. Both provided complementary and generally mutually supportive results. This chapter gives an account of the studies by these two groups — and others coming to different conclusions — as well as reviews and discussions leading up to the IARC decision in 2011. The chapter also describes how different groups have interpreted the authoritative IARC evaluation very differently.

There are by now several meta-analyses and reviews on mobile phones and brain tumours, which describe the challenges of doing epidemiology on this issue, the methodological limitations of the major studies published so far and the difficulties of interpreting their results.

It has been suggested that national incidence data on brain tumours could be used to qualify or disqualify the association between mobile phones and brain tumours observed in the case-control studies. However, in addition to methodological shortcomings, there might be other factors that influence the overall incidence rate such as changes in exposure to other risk factors for brain tumours that are unknown in descriptive studies. Cancer incidence depends on initiation, promotion and progression of the disease. As the mechanism for radiofrequency electromagnetic fields carcinogenesis is unclear, it supports the view that descriptive data on brain tumour incidence is of limited value.

The chapter points to mobile phone industry inertia in considering the various studies and taking the IARC carcinogenic classification into account and a failings from the media in providing the public with robust and consistent information on potential health risks. The IARC carcinogenic classification also appears not to have had any significant impact on governments' perceptions of their responsibilities to protect public health from this widespread source of radiation.

The benefits of mobile telecommunications are many but such benefits need to be accompanied by consideration of the possibility of widespread harms. Precautionary actions now to reduce head exposures would limit the size and seriousness of any brain tumour risk that may exist. Reducing exposures may also help to reduce the other possible harms that are not considered in this case study.

Evidence is increasing that workers with heavy long-term use of wireless phones who develop glioma or acoustic neuroma should be compensated. The first case in the world was established on 12 October 2012. The Italian Supreme Court affirmed a previous ruling that the Insurance Body for Work (INAIL) must grant worker's compensation to a businessman who had used wireless phones for 12 years and developed a neuroma in the brain.

22 Nanotechnology — early lessons from early warnings

Steffen Foss Hansen, Andrew Maynard, Anders Baun, Joel A. Tickner and Diana M. Bowman

Nanotechnology is the latest in a long series of technologies heralded as ushering in a new era of technology-driven prosperity. Current and future applications of nanotechnology are expected to lead to substantial societal and environmental benefits, increasing economic development and employment, generating better materials at lower environmental costs, and offering new ways to diagnose and treat medical conditions. Nevertheless, as new materials based on nanoscale engineering move from the lab to the marketplace, have we learnt the lessons of past 'wonder technologies' or are we destined to repeat past mistakes?

This chapter first introduces nanotechnology, clarifies the terminology of nanomaterials and describes current uses of these unique materials. Some of the early warning signs of possible adverse impacts of some nanomaterials are summarised, along with regulatory responses of some governments. Inspired by the EEA's first volume of *Late lessons from early warnings*, the chapter looks critically at what lessons can already be learned, notwithstanding nanotechnology's immaturity.

Nanotechnology development has occurred in the absence of clear design rules for chemists and materials developers on how to integrate health, safety and environmental concerns into design. The emerging area of 'green nanotechnology' offers promise for the future with its focus on preventive design. To gain traction, however, it is important that research on the sustainability of materials is funded at levels significant enough to identify early warnings, and that regulatory systems provide incentives for safer and sustainable materials.

Political decision-makers have yet to address many of the shortcomings in legislation, research and development, and limitations in risk assessment, management and governance of nanotechnologies and other emerging technologies. As a result, there remains a developmental environment that hinders the adoption of precautionary yet socially and economically responsive strategies in the field of nanotechnology. If left unresolved, this could hamper society's ability to ensure responsible development of nanotechnologies.

23 Understanding and accounting for the costs of inaction

Mikael Skou Andersen and David Owain Clubb

In political decision-making processes, the burden of proof is often distributed such that policymakers only respond to early warning signals from environmental hazards once the costs of inaction have been estimated.

This chapter revisits some key environmental issues for which estimates of costs of inaction have been carefully developed over many years of research. The aim is to consider the methodological challenges involved in producing estimates that are credible and appropriate rather than present specific estimates for these costs.

The case studies also provide insights into how early warning signals might provide a basis for estimating the costs of inaction, when the science base is less consolidated. For example, the case of nitrates in drinking water illustrates that a precautionary approach to the costs of inaction is quite conceivable. The phase-out of ozone-depleting substances, where early-warning scientists successfully alerted the world to the damaging effects of chlorofluorocarbons (CFCs), provides another important case because additional impacts for global warming actually cause the costs of inaction to be considerably higher than initially believed. This is a reminder that figures for the costs of inaction have often been grossly underestimated.

Finally, in the case of air pollution, making use of different estimates for mortality risk avoidance will help decision-makers to see that there are higher- and lower-bound estimates for the costs of inaction. Even if the lower-bound estimates are perhaps too conservative, with a bias towards health effects, they will in many situations encourage more rather than less abatement effort. Reducing emission loads will also tend to bring relief for the intangible assets of biodiversity and nature.

Making the best use of environmental science and modelling helps to make environmental protection and precaution a priority. Producing cost estimates should not be left to economists alone, but should rather be seen as a starting point for a broader discussion, featuring also the relevant expertise in health, ecology, demography, modelling and science. Well researched estimates, based on interdisciplinary collaboration, can strengthen some of those scattered and diffuse interests, which during the ordinary processes of policy-making have difficulty making their voices heard.

24 Protecting early warners and late victims

Carl Cranor

Many *Late lessons from early warnings* chapters provide examples of early warning scientists who were harassed for bringing inconvenient truths about impending harm to the attention of the public and regulators. There is also some evidence that young scientists are being discouraged from entering controversial fields for fear of such harassment. In addition, where warnings have been ignored and damage has ensued, it has often proven difficult in the past to achieve prompt and fair compensation for the victims. Some ideas for reform, building on some current institutional models are explored here.

This chapter first explores the idea of extending whistleblowing laws to help encourage and protect early-warning scientists and others who identify evidence of impending harm. Complementary measures, such as greater involvement of professional societies and the use of recognition awards, as for example in Germany, could also be helpful.

Next, the chapter explores improved mechanisms for compensating victims of pollution and contamination. The chapter on the Minamata Bay disaster provides an extreme example of long delays in getting adequate compensation for the victims of methylmercury poisoning. It was almost fifty years, between 1956 and 2004, before the victims attained equitable levels of compensation and legal recognition of responsibility. Other case studies illustrate similar examples of long delays in receiving adequate compensation.

Options are examined for providing justice to any future victims of those emerging technologies such as nanotechnology, genetically modified crops and mobile phone use, which currently can provide broad public benefits but potentially at a cost to small groups of victims. The potential for widespread exposure and uncertain science could justify 'no-fault' administrative schemes that provide more efficient and equitable redress in situations where the benefit of scientific doubt would be given to victims. The use of anticipatory assurance bonds to help minimise and meet the costs of future environmental damage from large scale technologies is also explored.

A supplementary panel text describes cases of asbestos and mesothelioma, where the senior courts in the United Kingdom have developed innovative ways of dealing with both joint and several liability, and the foreseeability of subsequent asbestos cancers, after the initial recognition of the respiratory disease, asbestosis. Such legal developments in the field of personal injury could illustrate the future direction of long-tail liability in both environmental damage and personal injury.

25 Why did business not react with precaution to early warnings?

Marc Le Menestrel and Julian Rode

In the past, companies have frequently neglected early warning signals about potential hazards for human health or the environment associated with their products or operations. This chapter reviews and analyses relevant interdisciplinary literature and prominent case studies — in particular those documented in both volumes of *Late lessons from early warnings* — and identifies main factors responsible for the disregard of early warning signals.

The chapter shows how economic motives often drive non-precautionary business decisions. In virtually all reviewed cases it was perceived to be profitable for industries to continue using potentially harmful products or operations. However, decisions are also influenced by a complex mix of epistemological, regulatory, cultural and psychological aspects. For instance, characteristics of the research environment and the regulatory context can provide business actors with opportunities to enter into 'political actions' to deny or even suppress early warning signals. Also, business decision-makers face psychological barriers to awareness and acceptance of the conflicts of values and interests entailed by early warning signals. Cultural business context may further contribute to the denial of conflicts of values.

The chapter concludes with a set of reflections on how to support more precautionary business decision making. A prominent policy response to the conflicting interests of business and society is introducing regulations that attempt to steer business rationality towards internalising external effects. Innovative solutions such as assurance bonding should be considered.

There is a need to better understand and expose why business actors do not respond voluntarily to early warning signals with precautionary actions. Blaming business, in particular with hindsight, tends to be common reaction that may not always be constructive. It often misses the complex or even contradictory set of motives and drivers that business actors face.

Public institutions could support progressive business by analysing and publically disclosing the dilemmas and temptations entailed by early warning signals, for example for different industries and for the specific societal and regulatory context of decisions. Rigorous and explicit exposition of the dilemmas will create further incentives for responsible actors to share and communicate their precautionary responses.

An additional reflection centres on the role of political actions of business actors, in particular those actions aimed at suppressing early warning signals. Regulatory efforts that make the political actions of business more transparent can help to sustain a sound balance of power, thereby maintaining our ability to benefit from early warning signals and reducing the likelihood of health and environmental hazards.

26 Science for precautionary decision-making

Philippe Grandjean

The goals of academic researchers may differ from those of regulatory agencies responsible for protecting the environment. Thus, research must take into account issues such as feasibility, merit and institutional agendas, which may lead to inflexibility and inertia.

A large proportion of academic research on environmental hazards therefore seems to focus on a small number of well studied environmental chemicals, such as metals. Research on environmental hazards should therefore to a greater extent consider poorly known problems, especially the potential hazards about which new information is in particular need.

Misinterpretation may occur when results published in scientific journals are expressed in hedged language. For example, a study that fails to document with statistical significance the presence of a hazard is often said to be negative, and the results may be misinterpreted as evidence that a hazard is absent. Such erroneous conclusions are inspired by science traditions, which demand meticulous and repeated examination before a hypothesis can be said to be substantiated.

For prioritising needs for action, research should instead focus on identifying the possible magnitude of potential hazards. Research is always affected by uncertainties and many of them can blur a real association between an environmental hazard and its adverse effects, thereby resulting in an underestimated risk. Environmental health research therefore needs to address the following question: are we sufficiently confident that this exposure to a potential hazard leads to adverse effects serious enough to initiate transparent and democratic procedures to decide on appropriate intervention?

The choice of research topics must consider societal needs for information on poorly known and potentially dangerous risks. The research should be complementary and extend current knowledge, rather than being repetitive for verification purposes, as required by the traditional science paradigm. Research findings should be openly available and reported so that they inform judgments concerning the possible magnitude of suspected environmental hazards, thereby facilitating precautionary and timely decision-making.

27 More or less precaution?

David Gee

Despite its presence in a growing body of EU and national legislation and case law, the application of the precautionary principle has been strongly opposed by vested interests who perceive short term economic costs from its use. There is also intellectual resistance from scientists who fail to acknowledge that scientific ignorance and uncertainty, are excessively attached to conventional scientific paradigms, and who wait for very high strengths of evidence before accepting causal links between exposure to stressors and harm.

The chapter focuses on some of the key issues that are relevant to a more common understanding of the precautionary principle and to its wider application. These include different and confusing definitions of the precautionary principle and of related concepts such as prevention, risk, uncertainty, variability and ignorance; common myths about the meaning of the precautionary principle; different approaches to the handling of scientific complexity and uncertainty; and the use of different strengths of evidence for different purposes.

The context for applying the precautionary principle also involves considering the 'knowledge to ignorance' ratio for the agent in focus: the precautionary principle is particularly relevant where the ratio of knowledge to ignorance is low, as with emerging technologies.

A working definition of the precautionary principle is presented that aims to overcome some of the difficulties with other definitions, such as their use of triple negatives; a failure to address the context of use of the precautionary principle; no reference to the need for case specific strengths of evidence to justify precaution; and overly narrow interpretations of the pros and cons of action or inaction.

The chapter also points to the need for greater public engagement in the process of framing and decision-making about both upstream innovations and their downstream hazards, including the specification of the 'high level of protection' required by the EU treaty. A precautionary and participatory framework for risk analysis is proposed, along with some 'criteria for action' to complement criteria for causation.

The capacity to foresee and forestall disasters, especially when such action is opposed by powerful economic and political interests, appears to be limited, as the case studies in *Late lesson from early warnings* illustrate. The chapter argues that with more humility in the face of uncertainty, ignorance and complexity, and wider public engagement, societies could heed the lessons of past experience and use the precautionary principle, to anticipate and minimise many future hazards, whilst stimulating innovation. Such an approach would also encourage more participatory risk analysis; more realistic and transparent systems science; and more socially relevant and diverse innovations designed to meet the needs of people and ecosystems.

28 In conclusion

The first volume of *Late lessons from early warnings* highlighted the difficulties of balancing precaution with technological innovation and ended with a call to action for policymakers. How much progress has been made since then?

First, there is growing evidence that precautionary measures do not stifle innovation, but instead can encourage it, in particular when supported by smart regulation or well-designed tax changes. Not only has the body of knowledge become richer since 2001, but also the number of stakeholders involved in decision-making has become larger and more diverse. There has also been increasing attention to communicating scientific uncertainty, especially in the fields of climate change, food safety, and emerging risks.

However, there has been less progress in other areas: for example, many of the political and scientific 'bureaucratic silos' still remain, despite frequent calls for policy integration and inter-departmental coordination. This has led to the unintended destruction of stocks of natural capital in some parts of the world and in other instances, the global spread of technologies, despite warnings of impending hazards. The result has been widespread damage, with most polluters still not paying the full costs of pollution.

Yet, more encouragingly, new transformative approaches are emerging to manage the systemic and interconnected challenges the world faces e.g. economic/financial, climate/energy, ecosystems/food. These relate, inter alia, to the increasing use of digital communications and networking by consumers, citizens and shareholders to demand and foster increased participation, more social responsibility, greater levels of accountability and higher transparency, especially in determining future pathways for energy and food production. There is a greater understanding of the complexity of the environment, of scientific ignorance and uncertainties, the irreversibility of many harmful impacts and on the broader risks to the long term interests of society if political and financial institutions remain unchanged. Also some corporations are fundamentally embracing sustainable development objectives in their business models and activities.

The case studies across both volumes of *Late lessons from early warnings* cover a diverse range of chemical and technological innovations, and highlight a number of systemic problems. These include a lack of institutional and other mechanisms to respond to early warning signals; a lack of ways to correct market failures either caused by misleading market prices or where costs and risks to society and nature are not properly internalised; and the fact that key decisions on innovation pathways are made by those with vested interests and/or by a limited number of people on behalf of many. The insights and lessons drawn from the case histories certainly provide the seeds for some of the answers. They also provide knowledge for a series of key actions that are outlined below.

Of course, many questions remain. For example: how can the precautionary principle be used further to support decision-making in the face of uncertainties and the inevitable surprises that come from complex systems?; how can societies avoid a lack of 'perfect' knowledge being used as a justification for inaction in the face of 'plausible' evidence of serious harm?; how can conflicting interests be balanced during the phases of development and use?; and how can the benefits of products and technologies be more equitably distributed?

Reduce delays between early warnings and actions

The majority of the case studies in *Late lessons from early warnings* Volumes 1 and 2 illustrate that if the precautionary principle had been applied on the basis of early warnings, justified by 'reasonable grounds for concern' many lives would have been saved and much damage to ecosystems avoided. It is therefore very important that large scale emerging technologies, such as biotechnologies, nanotechnologies and information

and communication technologies, apply the precautionary principle based on the experiences and lessons learned from these and other case studies.

Precautionary actions can be seen to stimulate rather than hinder innovation; they certainly do not lead to excessive false alarms. As the analysis in Volume 2 shows, of 88 cases of claimed 'false positives', where hazards were wrongly regulated as potential risks, only four were genuine false alarms. The frequency and scale of harm from the mainly 'false negative' case studies indicate that shifting public policy towards avoiding harm, even at the cost of some false alarms, would seem to be worthwhile, given the asymmetrical costs of being wrong in terms of acting or not acting based on credible early warnings.

However, the speed and scale of today's technological innovations can inhibit timely action. This is often because by the time clear evidence of harm has been established, the technology has been modified, thereby allowing claims of safety to be subsequently re-asserted. Even where the technological change has been marginal, the large, often global, scale of investment can lead to widespread technological lock-in, which is then difficult and expensive to alter.

These features of current technological innovation strengthen the case for taking early warning signals more seriously and acting on lower strengths of evidence than those normally used to reach 'scientific causality'. Most of the historical case studies show that by the time such strong evidence of causality becomes available, the harm to people and ecosystems has become more diverse and widespread than when first identified, and may even have been caused by much lower exposures than those initially considered dangerous.

The case studies have also shown that there are many barriers to precautionary action, including: the short-term nature of most political and financial horizons; the existence of technological monopolies; the conservative nature of the sciences involved, including the separate 'silos' within which they operate; the power of some stakeholders; and the cultural and institutional circumstances of public policymaking that often favour the status quo.

Acknowledge complexity when dealing with multiple effects and thresholds

Increasing scientific knowledge has shown that the causal links between stressors and harm are more complex than was previously thought and this has practical consequences for minimising harm. Much of the harm described in Volumes 1 and 2, such as cancers or species decline, is caused by several co-causal factors acting either independently or together. For example, the reduction of intelligence in children can be linked to lead in petrol, mercury and polychlorinated biphenyls (PCBs) as well as to socio-economic factors; bee colony collapse can be linked to viruses, climate change and nicotinoid pesticides; and climate change itself is caused by many complex and inter-linked chemical and physical processes.

In some cases, such as foetal or fish exposures, it is the timing of the exposure to a stressor that causes the harm, not necessarily the amount; the harm may also be caused or exacerbated by other stressors acting in a particular timed sequence. In other cases, such as radiation and some chemicals such as bisphenol A (BPA), low exposures can be more harmful than high exposures; and in others, such as asbestos with tobacco, and some endocrine disrupting substances, the harmful effects of mixtures can be greater than from each separate stressor. There are also varying susceptibilities to the same stressors in different people, species and ecosystems, depending on pre-existing stress levels, genetics and epigenetics. This variation can lead to differences in thresholds or tipping point exposures, above which harm becomes apparent in some exposed groups or ecosystems but not others. Indeed there are some harmful effects which occur only at the level of the system, such as a bee colony, which cannot be predicted from analysing a single part of the system, such as an individual bee.

Our increased knowledge of complex biological and ecological systems has also revealed that certain harmful substances, such as polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichlorethane (DDT) can move around the world via a range of biogeochemical and physical processes and then accumulate in organisms and ecosystems many thousands of kilometres away.

The practical implications of these observations are threefold. First, it is very difficult to establish very strong evidence that a single substance or stressor 'causes' harm to justify timely actions to avoid harm; in many cases only reasonable evidence of co-causality will be available. Second, a lack of consistency between research results is not a strong reason for dismissing possible causal links: inconsistency is to be expected from complexity. Third, while reducing harmful exposure to one co-causal factor may not necessarily lead to a large reduction in the overall harm caused by many other

factors, in some cases the removal of just one link in the chain of multi-causality could reduce much harm.

A more holistic and multi-disciplinary systems science is needed to analyse and manage the causal complexity of the systems in which we live.

Rethink and enrich environment and health research

Environment and health research overly focuses on well-known rather than unknown hazards at the expense of emerging issues and their potential impacts. For example the ten most well-known substances, such as lead and mercury, account for about half of all articles on chemical substances published in the main environmental journals over the last decade. Over the past decade, public research funding in the European Union on nanotechnology, biotechnology as well as Information and Communications Technology (ICT) is heavily biased towards product development with about 1 % being spent on their potential hazards. A more equal division of funding between known and emerging issues, and between products and their hazards, would enrich science and help avoid future harm to people and ecosystems and to the long term economic success of those technologies.

Funding more holistic systems science would also help achieve a greater integration among the different branches of science and counteract problems such as: peer review predominantly within and not across disciplines; short-term interests outcompeting long-term vision; competition replacing cooperation because of conflicts of interest; contradictions amongst paradigms; fragmentation of values and authority; as well as fragmentation of information and knowledge. These can all lead to inferior solutions and provide increased opportunities for those with vested interests to manufacture doubt.

Scientific methods can also be improved. For example, much higher strengths of evidence are required overall before causality is accepted, compared to the evidence being used to assert safety. The assertion that there is *no evidence of harm* is then often assumed to be *evidence of no harm*, even though the relevant research is missing. Historically there has been an over-reliance on the statistical significance of point estimates compared to confidence limits based on multiple sampling. There has also been a bias towards using models that grossly simplify reality rather than using long-term observations and trend data of biological and ecological systems. These approaches have sometimes led to the production of false positives. More importantly the governance of scientific ignorance and unknown unknowns has been neglected.

Finally, many case studies highlight the problems faced by early warning scientists who have been harassed for their pioneering work, including bans on speaking out or publishing, loss of funding, legal or other threats, and demotion. One obvious conclusion is that scientists in these situations should receive better protection either via an extension of 'whistle blowing' and discrimination laws, or by independent acknowledgement of the value of their work.

Improve the quality and value of risk assessments

The majority of the case studies in *Late lessons from early warnings* indicate that risk assessment approaches need to better embrace the realities of causal and systems complexity (rather than use a narrow conception of 'risk') with the inevitable features of ignorance, indeterminacy and contingency. In a number of case studies, for example BPA, where low doses are more harmful than high doses, or tributyltin antifoulants (TBT) and synthetic oestrogen diethylstilboestrol (DES) where the timing of the dose is what makes it harmful, simplistic assumptions are inadequate. Variability in exposures and varying susceptibilities in populations and species exposed also need to be more realistically factored into risk assessments.

This is equally true for technological risk assessments. As the Fukushima Investigation Committee concluded in 2011:

> '…the accidents present us with crucial lessons on how we should be prepared for 'incidents beyond assumptions'. With its failure to plan for the cascade effects beyond design–base accidents 'the regulatory emphasis on risk based probabilistic risk assessment has proven very limited'.

In other words, narrow risk assessment approaches are now outstripped by the realities which they cannot address, recognise and communicate. Too often this contributes to the effective denial of those risks that do not fit the risk assessment frame. It is therefore urgent that risk assessment practices

be transformed to make them broader-based, more inclusive, transparent and accountable. There should also be more communication on the diversity of scientific views, especially on emerging issues where ignorance and uncertainties are high and genuine differences of scientific interpretations are likely, desirable, and defensible. In this sense, recognising the pedigree of knowledge, i.e. the consistency of views amongst peers and the level of convergence coming from different branches of research, is essential for effective decision making and action to support the wellbeing of people and the environment.

The case studies show that evaluations of evidence in risk assessments can be improved by including a wide range of stakeholders when framing the risks and options agenda; broadening the scope and membership of evaluation committees; increasing the transparency of committee approaches and methods, particularly in identifying uncertainties and ignorance; and ensuring their independence from undue influence through using appropriate funding sources and applying robust policies on conflicts of interest.

Public confidence would be increased if all the evidence used in risk assessments was made publicly accessible and open to independent verification, including data submitted by industries to authorities.

As experiences from mercury, nuclear accidents, leaded petrol, mobile phones, BPA, and bees show, there can be a significant divergence in the evaluations of the same, or very similar, scientific evidence by different risk assessment committees. In such instances, differences in the choice of paradigm, assumptions, criteria for accepting evidence, weights placed on different types of evidence, and how uncertainties were handled, all need to be explained. Risk assessors and decision makers also need to be aware that complexity and uncertainty have sometimes been misused to shift the focus away from precautionary actions by 'manufacturing doubt' and by waiting for 'sound science' approaches that were originally developed by the tobacco industry to delay action.

Foster cooperation between business, government and citizens

Policy formulation should start from a broad concept of technological innovation to include non-technological, social, institutional, organisational and behavioural innovation. In this framework, governments have at least three roles: providing direction by putting in place smart regulations and consistent market signals; ensuring that the distributional consequences of innovations are balanced between risks and rewards across society, fostering a diversity of innovations so that the wider interests of society; and take precedence over narrower interests.

Numerous case studies show that decisions to act without precaution often come from businesses. There are, however, several impediments to businesses acting in a precautionary manner, including a focus on short-term economic value for shareholders alongside psychological factors that lead to a so-called 'ethical blindness' or a 'self-serving bias' whereby people largely interpret ambiguous situations in their own interests. Governments and businesses could collaborate more with citizens on publicly disclosing the potential value conflicts entailed in acting on early warning signals. A culture of transparency can in turn promote positive business attitudes and innovations.

Involving the public can also help in choosing between those innovation pathways to the future; on prioritising relevant public research; on providing data and information in support of monitoring and early warnings; improving risk assessments; on striking appropriate trade-offs between innovations and plausible health and environmental harms; and, making decisions about risk-risk trade-offs.

Correcting market failures using the polluter pays and prevention principles

When evidence of initial harm emerges, the costs should be internalised retroactively into the prices of polluting products, via taxes and charges, in line with the polluter pays principle and emerging practice across the world. The revenues could then be devoted partly to stimulating research into less hazardous alternatives, and partly to reform tax systems by reducing taxes and charges on 'societal goods' like employment.

The pollution taxes/charges would rise or fall in line with new scientific knowledge about increasing/decreasing harm, and this would help to level the playing field for less-polluting alternative products. Tax shifts from employment to pollution and the inefficient use of resources can bring multiple benefits such as increased employment, a stimulus to innovation, a more stable tax base in the light of expected demographic changes, and a more efficient tax collection system.

More broadly, firms and governments need to extend their economic accounting systems to incorporate the full impacts of their activities on people's health and on ecosystems. Governments need to anticipate this in their policies, by providing the right blend of fiscal instruments to both protect the public and ensure that firms internalise the true costs of potential harm.

A number of case studies also demonstrate the long time lags between evidence of harm and the additional injustice and time of forcing victims to pursue their cases through civil compensation claims. Prompt and anticipatory no-fault compensation schemes and assurance bonds, could be set up and financed in advance of potential harm by the industries that are producing novel and large-scale technologies, thereby helping to offset any potential market failure. Such schemes can also be designed to increase the incentives for innovating companies to carry out more *a priori* research into the identification and elimination of hazards.

Governance of innovation and innovation in governance

The *Late lessons from early warnings* reports demonstrate the complexities of developing not only the right kind of science and knowledge but also handling the interactions between the many actors and institutions involved — governments, policymakers, businesses, entrepreneurs, scientists, civil society representatives, citizens and the media.

Alongside many other analyses produced across the world today, the reports also stress the need to act to transform our ways of thinking and of doing, and urgently so in the face of unprecedented global changes, challenges and opportunities. Many lessons have been learnt, yet have not been acted upon. Any calls for action will need to reflect on today's global socio-economic setting and support, among other things, the drive to:

- rebalance the prioritisation of economic and financial capital over social, human and natural capitals through the broader application of the policy principles of precaution, prevention and polluter-pays, and environmental accounting;

- broaden the nature of evidence and public engagement in choices about key innovation pathways by directing scientific efforts more towards dealing with complex, systemic challenges and unknowns and complementing this with professional, lay, local and traditional knowledge; and,

- build greater adaptability and resilience in governance systems to deal with multiple systemic threats and surprises, through strengthening institutional structures and deploying information technologies in support of the concept of responsible information and dialogues.

The governance of innovation will remain at the level of good intentions unless it is translated into innovations in science practices, institutional arrangements and public engagements as well as transformations in prevailing business attitudes, practice and influence. These are the tasks that lie ahead.

In memory of Masazumi Harada, 1935–2012

Masazumi Harada, a physician involved for many years in the study of the mercury poisoning Minamata disease, died in June 2012 of acute myelocytic leukemia at his home in Kumamoto City. He was 77.

Harada conducted medical examinations on the disease's sufferers for the first time in the summer of 1961 in Minamata city in Kumamoto Prefecture while he was a student at Kumamoto University's graduate school.

Shocked by their miserable lives, Harada devoted himself to the study of the disease from that time. Harada published a thesis on congenital Minamata disease in 1964. The work had a significant impact as it disproved the conventional belief at the time that the placenta does not pass poisons. He received an award from the Japanese Society of Psychiatry and Neurology for the thesis in 1965.

He then established the Open Research Center for Minamata Studies at the university in 2005, becoming the center's head. He continued to lead the disease's research from non-medical perspectives as well. Harada visited Brazil, China and native Indian communities in Canada to discover those suspected of suffering from the disease.

Author of many books, Harada wrote 'Minamata Byo' (Minamata Disease), which raised awareness on the issue around the world.

Dr. Masazumi Harada first came to Asubpeeschoseewagong (Grassy Narrows) and Wabaseemoong (White Dog) First Nations in Canada in the early 1970s. Harada's death comes at the end of River Run 2012, five days of actions by members and supporters of Grassy Narrows in Toronto, who are seeking to have Minamata disease recognized in Canada and Ontario. Harada's final report for the Grassy Narrows community was released on 4 June 2012 after 30 years of research, showing mercury deposited in the river by the Dryden paper mill in the 1970s is impacting those who were not yet born when the dumping ceased.

In memory of Poul Harremoës, 1934–2003

Poul Harremoës was a key player in environmental issues in Denmark and internationally for more than 30 years until his death, at 69, in 2003. In that time, those who worked closely with him benefited from a continuous, almost daily flow of excellent ideas for new research projects.

He was a member of the Danish Pollution Council, which prepared the first framework national law on environmental protection and advised on the establishment of a Ministry of Environment from 1971. He was a key participant in numerous settings, including the first Scientific Committee of the European Environment Agency from 1995.

He had a civil engineering degree from the Technical University of Denmark. He specialised early on in geo-technics and constructed dams on the Faroe Islands. While teaching geo-technics he wrote a textbook that was used for more than 40 years. However, he was able to quickly change his research direction and develop new areas of excellence. So, for example, he got a grant to study at Berkeley, California, from where he received a M.Sc. degree in environmental engineering.

In 1972, he became professor in environmental engineering at the Technical University of Denmark where he originally worked with wastewater discharge to the sea and the biological processes of wastewater treatment. He became a world leading scientist in the theories of biofilms for removal of organics and nitrogen from wastewater before turning to sewer design and modelling. In 2000, Poul was awarded the *Heineken prize for Environmental Sciences* for his contributions to the theory of biofilm kinetics in relation to biological waste water treatment and for his successful organisation of the international scientific community in water pollution research and control.

As a result of his work with sewers and storm water he went into the area of risk analysis and the role of the precautionary principle. In a short time he became an international expert in this field and was highly demanded for lectures in all parts of the world. A key outcome of his interest was his contributions as chairman of the editorial team for the first volume of *Late lessons from early warnings* published in 2001.

European Environment Agency

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Scientific Committee on Health, Environmental and Emerging Risks

SCHEER

Statement on emerging health and environmental issues (2018)

The SCHEER adopted this statement by written procedure on 20 December 2018.

ABSTRACT

The purpose of this SCHEER statement is to draw the EU Commission Services' attention to emerging issues in the non-food area that SCHEER members have identified as having the potential to impact human health and /or on the environment in the future. The Secretariat will use this list when discussing potential new mandates with relevant Commission services.

Keywords: SCHEER, emerging issues, emerging risks, newly identified health risks, health, environment, impacts

Opinion to be cited as:

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All declarations by Working Group members are available at the following webpage: https://ec.europa.eu/health/scientific_committees/experts/declarations/scheer_en

About the Scientific Committees (2016-2021)

Two independent non-food Scientific Committees provide the Commission with the scientific advice it needs when preparing policy and proposals relating to consumer safety, public health and the environment. The Committees also draw the Commission's attention to the new or emerging problems which may pose an actual or potential threat.

The Scientific Committee on Consumer Safety (SCCS) and the Scientific Committee on Health, Environmental and Emerging Risks (SCHEER) review and evaluate relevant scientific data and assess potential risks. Each committee includes top independent scientists from all over the world who are committed to working in the public interest.

In the formulation of its policies and proposals, the Commission also relies on other Union bodies, such as the European Food Safety Authority (EFSA), the European Medicines Agency (EMA), the European Centre for Disease prevention and Control (ECDC) and the European Chemicals Agency (ECHA).

SCHEER

This Committee, on request of Commission services, provides Opinions on questions concerning health, environmental and emerging risks. The Committee addresses questions on:

- health and environmental risks related to pollutants in the environmental media and other biological and physical factors in relation to air quality, water, waste and soils.

- complex or multidisciplinary issues requiring a comprehensive assessment of risks to consumer safety or public health, for example antimicrobial resistance, nanotechnologies, medical devices and physical hazards such as noise and electromagnetic fields.

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The Opinions of the Scientific Committees present the views of the independent scientists who are members of the committees. They do not necessarily reflect the views of the European Commission. The Opinions are published by the European Commission in their original language only.

http://ec.europa.eu/health/scientific_committees/policy/index_en.htm

TABLE OF CONTENTS

1. INTRODUCTION

The primary purpose of this position statement is to draw the attention of the EU Commission Services to emerging issues in the non-food area that have been identified by the SCHEER members as having the potential to significantly impact human health and /or on the environment in the future.

Identifying emerging issues early on may greatly help for ensure a high level of public safety and environmental protection. However, the data available to correctly identify emerging issues and their impacts is inevitably likely to be very limited. It is therefore important that each issue that is identified is regularly reviewed. The SCHEER aims, therefore, to regularly review any relevant new developments and to produce an updated position statement twice during the Scientific Committee term (this term is from 2016-2021). The Committee can also submit an urgent issue to the Commission at any time. In considering emerging issues, the SCHEER would like to work closely with other EU scientific advisory committees whose mandates also include looking at emerging issues.

The SCHEER recognised the need to establish a very flexible framework to aid the correct identification of emerging issues and their potential impacts (see document 'Emerging Issues and the Role of the SCHEER, Position Paper').

SCHEER members have been asked during plenary meetings, in dedicated 'brainstorming sessions', to identify emerging/relevant issues that they think should be flagged for the Commssion Services.

The criteria used to identify an emerging issue were as follows:

- Novelty of the stressor or process
- Scale of possible impacts on man and /or the environment
- Severity of impacts for particular organisms (priority for life threatening)
- Urgency i.e. the temporal nature of the likely changes (priority for rapid increases)
- Not investigated in depth recently by a reputable scientific body
- Anticipated to be increasingly important over time

To aid this, a standardised format has been used and issues have been placed in particular categories. It is acknowledged that further consideration of some of the issues that have been identified should be led by other scientific committees.

2. FORMAT FOR DESCRIBING AN EMERGING ISSUE

A common format was proposed to describe emerging issues. This was in the format of a table in which the committee members have been asked to fill in the following:

- The topic proposed
- The author (name of SCHEER submitter)

- Sources (one or more selected items from the ones mentioned under point 1 between 1-12)
- Causative factors (one or more selected items from the ones mentioned under point 2 between a and h)
- Preliminary ranking of the hazard ((*,1, 2 or 3 where *=uncertain and 3 is high for uniqueness, soundness, severity, spatial scale, urgency, and interactions, respectively)
- Preliminary estimation of importance (*,1, 2 or 3 where *=uncertain and 3 is high)
- Description / background

1) Sources

Risks associated with:

1) Buildings and infrastructure

2) Energy and electronic communications

3) Disease evolution e.g. due to pathogen changes

4) Industrial and related activities

5) Waste processing and utilisation

6) Use of natural resources

7) Transport and storage

8) Human behaviour (socio-economic, lifestyle, perception)

9) Medical developments (technology, pharmaceuticals)

10) Environmental change

11) Product use/misuse

12) Agriculture and food

13) New materials

2) Causes / Contributing factors:

a) Technical advances opening up the prospect of new products and/or processes and/or raising concerns about waste treatment safety

b) A consequence of changes in the natural environment

c) Changes resulting from alterations in price, supply of materials and commodities

d) Changes due to alterations in legislation or public welfare measures

e) Other socio-cultural or demographic elements

f) Outcomes of research

g) Large scale illegal activities

h) Public/political concern

3. NEXT STEPS

The list will be used by the Secretariat when discussing potential new mandates with relevant Commission services.

4. ISSUES

4.1. Personal communication and listening devices

Topic	**Personal communication and listening devices**
Initiator(s)	Theodoros Samaras
Sources **Causative factors** **(see section 2 of this document)**	8, 11 a, c, e
Hazard (Rank features as 1,2,3 or *) **-uniqueness** **-soundness** **-severity** **-scale** **-urgency** **-interactions**	 1 1 * 3 1 2
Parallels with past emerging issues. Potential interactions with other stressors)	It has already been established that driving while using a portable device presents a quantifiable risk for traffic accidents. In addition, texting has been linked in the past to orthopaedic problems of hand and arm joints, as well as to more serious musculoskeletal injuries. More information: Fares *et al.* (2017). The use of personal digital devices adds to the exposure to screen light (this has been dealt with in previous Opinions about artificial light and LED exposure). Moreover, it exacerbates the problems resulting from environmental noise (also assessed in the past), especially by using high volume levels on earphones.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	1
Background including reliability of data, a key reference if possible any other reasons for concern.	There are mainly two issues associated with personal communication and listening devices, which mostly concern young people since they constitute the main users of such devices and start to use them at an ever earlier age. The first one has to do with pedestrian safety. Distraction due to texting, listening to music or using multimedia apps can compromise cognitive and audiovisual awareness and may pose a risk for the safety of

	pedestrians (e.g. at street crossings). The second issue has to do with nearsightedness (myopia). The increased use of personal digital devices has been mentioned as a risk factor for this trend (especially in the under-40 age group). For more information: **References** 1. Schwebel D.C. *et al.* (2012). 2. Schabrun S.M. *et al.* (2014). 3. Holden B.A. *et al.* (2016).

4.2 Virtual reality

Topic	Virtual reality
Initiator(s)	Ana Proykova
Sources **Causative factors (see section 2 of this document)**	8, 11 a,e,f Virtual reality (VR) and augmented reality (AR) are gaining momentum as promising new technologies. They can potentially expand the field of human knowledge by changing how people learn, work, play and entertain themselves. High-tech VR and AR headsets are popping up everywhere – from the expensive ones from Samsung, Google and Facebook, to the generic cardboard headsets for the lower-end market.
Hazard **(Rank features as 1,2,3 or *)** **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	Health risks: anxiety, nausea, eye strain, radiation exposure. VR can have neurological effects because of its eerily realistic simulated motion. Virtual Reality (VR) sickness can cause intense discomfort, shorten the duration of a VR experience, and create an aversion to further use of VR. 3 * * 3 3 *
Parallels with past emerging issues. Potential interactions with other stressors)	Parallel with Army Aviation - Simulator sickness Environmental Social Stress
Preliminary Estimation of importance (*, 1, 2 or 3 where *=uncertain and 3 is high)	3 Considering the broad usage of Virtual Reality (from games to hospital treatment of anxiety) The hazard and risks are under investigation.
Background including reliability of data, a key reference if possible any other reasons for concern.	**References** 1. Fernandes A.S., Feiner S.K. (2016). 2. Pallavicini F. *et al.* (2013). 3. Veling W. *et al*.. (2016). 4. Jáuregui-Renaud K. (2015). 5. Cobb S.V.G., Nichols S.C., Wilson, J.R. (1995).

4.3 E-cigarette and chronic diseases

Topic	**E-cigarettes and chronic diseases**
Initiator(s)	Demosthenes Panagiotakos
Sources **Causative factors** **(see section 2 of this document)**	9 E-cigarettes, in their modern form, were introduced in the early 2000s as a means for smoking cessation. The e-cigarette liquid contains several chemicals, like nicotine, propylene glycol, glycerin, flavourings and others. Current research suggests that the e-cigarette aerosol contains substances that could be considered as harmful, including flavouring chemicals, metals (like lead), and other cancer-causing chemicals. There is no consistent evidence regarding the effectiveness of e-cigarettes in helping people to quit smoking. Moreover, there is a tendency in people to start vaping (e-cigarettes), instead of smoking. Compared with "regular" cigarettes, e-cigarettes may be less harmful in terms of smoking-related chronic diseases, but regarding their use compared to no smoking, the health effects are not well understood or appreciated. Moreover, taking into account that e-cigarette use is increasingly prevalent and fashionable, especially among adolescents and younger people, it can be regarded as an emerging public health issue.
Hazard **(Rank features as 1,2,3 or *)** **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 1 * 3 3 (due to the increasing frequency of use) 3 (due to the interactions with other lifestyle determinants and psychological stressors)
Parallels with past emerging issues. Potential interactions with other stressors.	The e-cigarette has been described as a possible form of harm reduction. To date, there has been no consistent evidence that e-cigarette can significantly reduce smoking in the population, and there have not been enough studies done on the effect of (active or passive) e-cigarette exposure compared to non-smoking on human health. Moreover, cigarette smoking, in general, is known to interact with a variety of unfavourable lifestyle behaviours, like unhealthy dietary habits and physical inactivity, as well as the presence of chronic stress, leading to increased risk for cardiovascular disease, COPD and types of cancer. Taking into account that the aforementioned synergistic factors are now increasing at alarming rates and the fact that data about the effects of e-cigarette on human health are not well understood yet, the study of e-cigarette on human health is considered more important as ever before, in terms of public health prevention.
Preliminary Estimation of	3

importance (*,1,2 or 3 where *=uncertain and 3 is high)	
Background including reliability of data, a key reference if possible any other reasons for concern.	E-cigarettes have been patented since the 1960s, however, they have been available as a product from the early 2000s. The use of e-cigarettes has risen exponentially over the past 10 years; however, it is difficult to estimate its use at population level. E-cigarettes are now available in Europe and in the majority of countries in the Western world, but with significant differences in use between countries. It is notable that the use of the e-cigarette tends to be a habit by a considerable proportion of young adults and adolescents in many European countries. The health risks of e-cigarettes are uncertain. There are studies suggesting that e-cigarettes may cause, similar to tobacco cigarettes, harm to the cardiovascular and lung system. Although some of the identified harmful components in e-cigarettes were measured in lower quantities than those in cigarettes, recent studies unveiled that the toxic effects of e-cigarettes should not be understated. There is an overlap between tobacco laws and medical drug policies and e-cigarette legislation in many countries. A European Directive of 2016 set standards for liquids, vaporizers, ingredients and child-proof liquid containers while the US FDA extended its regulatory power to include e-cigarettes. In some countries, new legislations are underway to regulate e-cigarette use. **References** 1. Filippidis F.T., Laverty A.A., Vardavas C.I. (2016). 2. Ioakeimidis N., Vlachopoulos C., Tousoulis D. (2016). 3. Makadia L.D., Roper P.J., Andrews J.O., Tingen M.S. (2017). 4. Cai H., Wang C. (2017). 5. Chun L.F., Moazed F., Calfee C.S., Matthay M.A., Gotts J.E. (2017). 6. Benowitz N.L., Burbank A.D. (2016). 7. Rahman M.A., Hann N., Wilson A., Mnatzaganian G., Worrall-Carter L. (2015). 8. European Commission (2014). MEMO, 26 February 2014.

4.4 Potential effects on wildlife of increases in electromagnetic radiation

Topic	Potential effects on wildlife of increases in electromagnetic radiation
Initiator(s)	Marian Scott
Sources **Causative factors** **(see section 2section 2 of this document)**	2 e "On the horizon, a new generation of even shorter high frequency 5G wavelengths is being proposed to power the Internet of Things (IoT). The IoT promises us convenient and easy lifestyles with a massive 5G interconnected telecommunications network. However, the expansion of broadband with shorter wavelength radiofrequency radiation highlights the concern that health and safety issues remain unknown. Controversy continues with regard to harm from current 2G, 3G and 4G wireless technologies. 5G technologies are far less studied for human or environmental effects" (Russell, 2018).
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 1 * 3 3 3 (due to the interactions with other ecosystems and species)
Parallels with past emerging issues. Potential interactions with other stressors.	This concern is more related to the change to 5G rather than a completely new concern. The effects of electromagnetic radiation have been generally well studied, however low frequency electromagnetic radiation is less well studied, hence the justification for introducing this an emerging issue.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background including reliability of data, a key reference if possible any other reasons for concern.	5G networks will soon be rolled out for mobile phone and smart device users. How exposure to electromagnetic fields could affect humans remains a controversial area, and studies have not yielded clear evidence of the impact on mammals, birds or insects. The lack of clear evidence to inform the development of exposure guidelines to 5G technology leaves open the possibility of unintended biological consequences.

	References 1. https://www.rsm.govt.nz/projects-auctions/current-projects/preparing-for-5g-in-new-zealand/folder-potential-health-effects-of-5g-technology/submissions-relating-to-health-concerns.pdf 2. Aertsa S., Wiart J., Martens L., Joseph W. (2017). 3. Pall M.L. (2018). 4. Di Ciaula A. (2018). 5. Russell C.L. (2018).

4.5 Chemicals in recycled materials, an issue in a circular economy

Topic	Chemicals in recycled materials, an issue in a circular economy
Initiator(s)	Theo Vermeire
Sources **Causative factors** **(see section 2 of this document)**	Many potential sources a, c, g, h
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 1 3 * 3 3 3
Parallels with past emerging issues. Potential interactions with other stressors)	In view of EU-wide strategies toward a circular economy, the issue of hazardous substances in recycled products is getting more and more attention. Risks can arise for the environment, consumers, workers. Over the last decade interest in the circular economy and therefore in recycling has increased considerably. One of the problems of recycling is that the materials may contain substances that pose a risk to man and the environment. So the possible advantages of recycling, such as more energy-efficient and CO_2-efficient production, should be weighed against the potential effects of these substances. Examples of hazardous substances incorporated into potentially recyclable material: the flame retardant hexabromocyclododecane (HBCDD) in extruded polystyrene, the plasticiser DEHP, cadmium and lead in polyvinyl chloride (PVC), heavy metals and PAHs in rubber crump from tyres.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background including reliability of data, a key reference if possible any other reasons for	Examples are: Lead, phthalates, cadmium, organotins, POPs, BDEs, HBCDD, e-waste, hazardous chemicals in rubber crump and toys. **References** 1. EFSA CEF Panel (EFSA Panel on Food Contact Materials,

concern.	Flavourings and Processing Aids) (2015). 2. Grant K., Goldizen F.C., Sly P.D., Brune M.-N., Neira M., van den Berg M., Norman R.E. (2013). 3. Janssen M.P.M. *et al.* (2016). 4. KEMI (2012). 5. Verschoor A.J., Bodar C.W.M., Baumann R.A. (2018).

4.6 Pharmaceuticals (human and veterinary) and illicit drugs in wastewater and surface waters

Topic	**Pharmaceuticals (human and veterinary) and illicit drugs in wastewater and surface waters**
Initiator(s)	Pim de Voogt, Marco Vighi
Sources **Causative factors** **(see section 2 of this document)**	5 Urban waste water. Wastes from pharmaceutical industries and from illegal drugs manufacturing. Animal farm emissions. Agricultural soils treated with WWTP sludge or animal manure. Aquaculture.
Hazard **(Rank features as 1,2,3 or *)**	Pharmaceuticals and illicit drugs loads in wastewater increase as a result of increased use of legal, illicit and counterfeit drugs, aging of the population and also due to fly tipping of waste from illegal drug manufacturing sites (Emke *et al*. 2018). As a result wastewater treatment facilities may become jeopardised. Exposure of aquatic environments (receiving surface waters) because WWTPs don´t (completely) remove residuals (Wang *et al.* 2016; Bijlsma *et al.* 2012). The occurrence of pharmaceuticals in wastewater and surface waters has been object of systematic research since the 1990s (Zuccato *et al*., 2006). Therefore, it might seem as if the issue should not be considered as an emerging risk. However, knowledge on the hazards for aquatic communities is still far from being complete and, in many cases, the possible effects on the aquatic ecosystems are completely unknown. Pharmaceuticals and illicit drugs are, by definition, biologically active compounds. The type of biological effect is highly specific and, in most cases, unwanted for natural populations. At the concentrations likely to occur in surface waters, the possibility of effects that may be studied with tools capable to measure traditional endpoints (e.g. acute or chronic toxicity) or more specific effects (e.g. endocrine disrupting effects) is, in most cases, highly improbable. Nevertheless, other types of direct or indirect effects on the functioning of ecosystems determined by the specific biological activity, are possible and largely unknown. A couple of examples of the most known cases are: • Occurrence and spread of antibiotic resistance (AMR). Determined by the presence of antibiotics in surface water, this may represent a change in ecosystem functioning and a risk for human health (see, for example, Xi *et al*., 2009). • Behavioural changes due to antidepressants. Psychoactive drugs alter the behaviour of aquatic vertebrates, for example reducing the capability to escape from predators, with dramatic changes in

- uniqueness **- soundness** **- severity** **- scale** **- urgency** **- interactions**	ecosystem functioning (see, for example, Brooks, 2014). Other possible effects are fully unknown and difficult to predict. 1 * * 3 3 *
Parallels with past emerging issues. Potential interactions with other stressors)	Interactions among different pharmaceuticals with possibilities of synergisms or antagonisms.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background including reliability of data, a key reference if possible any other reasons for concern.	**References** 1. Bijlsma L., Emke E., Hernández F., de Voogt P. (2012) 2. Brooks B. (2014). 3. Emke E., Vughs D., Kolkman A., de Voogt P (2018). 4. Wang J., Wang S. (2016). 5. Xi C., Zhang Y., Marrs C.F., Ye W., Simon C., Foxman B., Nriagu J. (2009). 6. Zuccato E., Castiglioni S., Fanelli R., Reitano G., Bagnati R., Chiabrando C., Pomati F., Rossetti C., Calamari D. (2006). 7. http://score-cost.eu/emcdda-wastewater-analysis-and-drugs/

4.7 Substance Mobility: a new criterion in chemicals regulation

Topic	**Substance Mobility: a new criterion in chemicals regulation**
Initiator(s)	Pim de Voogt
Sources **Causative factors** **(see section 2 of this document)**	5, 8, 9, 11 d,e Changes in public welfare measures / demographic changes Our society is using increasingly more chemical substances, and among the new emerging pollutants we are finding an increasing number of polar organic compounds. Although the concentration level of total organic contaminants decreases by about 2 orders of magnitude going from WWTP effluents to groundwater used for drinking water production to tapwater. The most polar contaminants in the WWTP effluents remain in the water throughout its passage to groundwater and also withstand traditional drinking water treatment processes. As a result, persistent mobile organic chemicals (PMOCs) may reach drinking water. Examples include trifluoromethanesulfonic acid and its halogenated homologues; 1-naphthalenesulfonic acid; 1,3-di-o-tolylguanidine and GenX (2,3,3,3-Tetrafluoro-2-(heptafluoro-propoxy)propanoic acid; aka FRD-903 or HFPO-DA).
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **-interactions**	PMOC may be of an equivalent level of concern as PBT substances. If emissions of PMOC or very persistent very mobile substances are ongoing and removal during water treatment is incomplete, their environmental concentrations will increase over time as these substances circulate and enrich in the water cycle. 3 3 3 3 3 2
Parallels with past emerging issues. Potential interactions with other stressors)	European legislation on chemical substances (REACH) primarily focuses on substances that do not easily break down and are therefore Persistent (P), accumulate in organisms (Bioaccumulation, B) and have an effect on organisms (Toxicity, T). This PBT legislation pays insufficient attention to the drinking water function of our surface waters and groundwater. After all, there are substances that do not accumulate very much but that are very difficult to remove from water. Due to their great affinity for water such substances are Mobile (M). If substances have PMT properties then they can present an exposure risk for humans via drinking water.

	Potential Interactions: Exposure to (mixtures of) chemicals
Preliminary Estimation of importance (*, 1, 2 or 3 where *=uncertain and 3 is high)	3 -Topic is highly relevant for REACH legislation -German UBA has issued a revised proposal for implementing criteria and an assessment procedure to identify Persistent, Mobile and Toxic (PMT) and very Persistent, very Mobile (vPvM) substances registered under REACH.
Background including reliability of data, a key reference if possible any other reasons for concern.	Whether an organic compound does or does not possess an affinity for water is mainly determined by the polarity of that substance. Polar substances and those that have a permanent charge (ions, such as salts) have an extremely high affinity for water. That high affinity means that polar and charged substances dissolve easily in water and are poorly retained in soils through which the water passes, in riverbanks or by sorptive water treatment processes. Such substances are difficult to remove from water during purification. In other words, the substances move easily with the (moving) water, are transported along with it and can easily reach drinking water: the substance is Mobile (M). Drinking water companies are certainly increasingly confronted with new expenditures necessary to cope with polar substances. **References** 1. Reemtsma T., Berger U., Arp H.P.H., Gallard H., Knepper T.P., Neumann M., Quintana J.B., de Voogt P. (2016). 2. Sjerps R.M.A., Vughs D. van Leerdam J.A., ter Laak T.L., van Wezel A.P. (2016). 3. http://www.ufz.de/promote/ 4. https://www.umweltbundesamt.de/en/publikationen/protecting-the-sources-of-our-drinking-water-from 5. Zahn D., Frömel T., Knepper T.P. (2016). 6. Montes R., Aguire J., Vidal X., Rodil R., Cela R., Quintana J.B. (2017). 7. Versteegh J.F.M., de Voogt P. (2017).

4.8 Drinking water treatment interactions with compounds and potential health effects

Topic	**Drinking water treatment interactions with compounds and potential health effects**
Initiator(s)	Marian Scott
Sources **Causative factors** **(see section 2 of this document)**	5,6,10 a,b New and modified drinking water treatments are being used to deal with removal of chemical (natural and anthropogenic) contaminants in the source waters. It is anticipated that in the light of climate change, there may be further interactions between such natural contaminants, leading to new (or increased) concentrations of by-products with potential for human health concerns.
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 1 2 3 1 *
Parallels with past emerging issues. Potential interactions with other stressors.	Many disinfection methods are being used in the production of drinking water. Among these, the advanced oxidation processes serve the dual purposes of disinfection and removal of chemical contaminants present in source water. Studies have shown that water containing natural organic matter, when treated with UV for disinfection, generated multiple disinfection byproducts (DBPs). [Bond *et al*. Water Res 45 (2011) 4341-54; Richardson *et al*. Mutat. Res. 636 (2007) 178-242; Ceretti *et al*. J Public Health Res. 2016 Dec 9; 5(3), 769], including Nitrogen-containing mutagenic DBPs [Vughs *et al.* Environ Sci Pollut Res 25 (2018) 3951-64]. Climate change is expected to increase surface run off in river catchments, leading to increased amounts and loads, as well as possible changes in characteristics, of natural organic matter in the source waters [Soh *et al.*, The Environmentalist 28 (2007) 158-165]. used for producing drinking water. This means that there is an increased probability that DBPs are being formed in the treatment processes required for disinfection and purification. Climate change effects may result in the need to modify water treatment processes resulting in potentially new interactions, and lead to the development of new treatment methods which could result in the

	formation of novel disinfection products with health effects including mutagenicity.
Preliminary Estimation of importance (*, 1, 2 or 3 where *=uncertain and 3 is high)	2
Background including reliability of data, a key reference if possible any other reasons for concern.	There are a variety of papers and studies being published concerning interactions between water treatment chemicals, organic material and residues in drinking water, exacerbated by climate change and with potential health effects (including neurological disorders). Aluminium has been implicated in the past, but the treatment interactions with chemical contaminants and the possible effects of climate change mean that this topic is gaining renewed interest. Examples of some of the studies are given below. **References** 1. Lalas S., Athanasiadis V., Dourtoglou V.G. (2018). 2. Glassmeyer S.T., Furlong E.T., Kolpin D.W., Batt A.L., Benson R., Boone J.S., Conerly O., Donohue M.J., King D.N., Kostich M.S., Mash H.E., Pfaller S.L., Schenck K.M., Simmons J.E., Varughese E.A., Vesper S.J., Villegas E.N., Wilson V.S. (2017). 3. Benson R., Conerly, O.D. Sander W., Batt A.L., Boone J.S., Furlong E.T., Glassmeyer S.T., Kolping D.W. Mash H.E., Schenck K.M., Simmonsi J.E. (2017). 4. Kessing, L.V., Gerds T. A. Nygård Knudsen N., *et al.* (2017). 5. Post G.B., Gleason J.A., Cooper K.R. (2017). 6. Bond T., Huang J., Templeton M.R., Graham N. (2011). 7. Richardson S.D., Plewa M.J., Wagner E.D., Schoeny R., Demarini D.M. (2007). 8. Ceretti E., Moretti M., Zerbini I., Villarini M., Zani C., Monarca S., Feretti D. (2016). 9. Vughs D., Baken K.A., Kolkman A., Martijn A.J., de Voogt P. (2018). 10. Soh Y., Roddick F., Van Leeuwen J. (2007).

4.9 Per- and polyfluorinated organic substances

Topic	**Per- and polyfluorinated alkyl substances (PFAS)**
Initiator(s)	Pim de Voogt
Sources **Causative factors** **see section 2**	4, 11, 13 c, d, h
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	Because of the persistence of PFAS, these compounds constitute potential risks for humans and the environment. 3 3 3 3 3 *
Parallels with past emerging issues. Potential interactions with other stressors)	Parallels: POPs Very few PFAS have been regulated (PFOS Stockholm convention; PFOS in products (EC); TDIs for PFOS and PFOA (EFSA). A new Opinion on PFOS and PFOA in food, by the EFSA Contam panel, has been finalised in Mar 2018. A Scientific Opinion by EFSA on the risk to human health related to the presence of Perfluoroalkylated substances in food, other than Perfluorooctane sulfonate and Perfluorooctanoic acid, is in the process of completion. PFOA is under review by the Stockholm POP convention. Possible interactions: dietary exposure to (mixtures of) chemicals.
Preliminary Estimation of prioritisation (*, 1, 2 or 3 where *=uncertain and 3 is high)	2-3 As a result of new information becoming available on the toxicity of PFAS, the revision of current guideline values (e.g. TDIs, drinking water guidelines) is urgent and for some members of the PFAS group it is underway. For others essential data are still missing. Hence the prioritisation is high.
Background including reliability of data, a key reference if possible any other reasons for concern.	For several members of the PFAS group PBT properties have been demonstrated. Others appear to be persistent and mobile (PM) and cross natural or technological barriers, thus posing risks to public health because of human exposure. Replacements by industry for the major part appear to rely on fluorine chemistry (e.g. GenX) which inherently leads to similarities in persistence. Many more emerging PFAS have been recently found to occur in surface waters (see e.g. Gebbink *et al*., 2017).

	References 1. Scientific Panel on Contaminants in the Food Chain, Minutes of the 85th Plenary meeting held on 4-6 July 2017, Parma (Italy). https://www.efsa.europa.eu/sites/default/files/event/170704-m.pdf; http://registerofquestions.efsa.europa.eu/roqFrontend/questionLoader?question=EFSA-Q-2015-00526; http://registerofquestions.efsa.europa.eu/roqFrontend/questionDocumentsLoader?question=EFSA-Q-2017-00549 2. Gebbink W.A., van Asseldonk L., van Leeuwen S.P.J. (2017). 3. Blum A., Balan S.A., Scheringer M., Trier X., Goldenman G., Cousins I., Diamond M., Fletcher T., Higgins C., Lindeman A.E., Peaslee G., de Voogt P., Wang Z., Weber R. (2015). 4. Ritscher A. *et al.* (2018).

4.10 New RNA pesticides and gene editing to reduce/eradicate pest populations

Topic	**New RNA pesticides and gene editing to reduce/eradicate pest populations**
Initiator(s)	Marian Scott
Sources **Causative factors** **(see section 2 of this document)**	6,8,10,12 a,f,h With increasing concerns about the negative impact of the use of chemical pesticides (see recent debates concerning glyphosates and neonicotinoids), on both the environment and humans, there are new developments in the use of gene technology (in the broadest sense) to manage pest populations).
Hazard **(Rank features as 1, 2, 3 or *)** **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 1 2 3 3 3
Parallels with past emerging issues. Potential interactions with other stressors)	Effects of various pesticides including examples such as neonicotinoids and glyphosate have been much contested, and any form of gene editing is likely to be met with concerns particularly given the social and political debates concerning GMO.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background including reliability of data, a key reference if possible any other reasons for concern.	Laboratory tests have shown that topical application of RNA could be a new way of controlling plant pests, including viruses and insects, by silencing genes that affect survival and reproduction. It is thought this method could be more publicly acceptable than other forms of genetic modification because its effects will not be passed on to offspring. However, the impact of widespread use of the method as a pesticide on non-target species is not yet known. New gene editing technologies could also be used to control animal populations, including invasive species, within the coming decade. For instance, applications of CRISPR-enabled gene drive technology are

	foreseen for several applications, such as the elimination or suppression of insect vectors and transmitting (plant) diseases. At a cost of more than £3 million a year, New Zealand aims to rid itself of rats, possums and stoats by 2050. These methods raise both ethical and ecological questions, from repercussions on wider ecosystems to the potential for gene traits to spread and wipe-out species in unintended areas. **References** 1. Lundgren J.G., Duan J.J. (2013). 2. Albright *et al*. (2017). 3. Medina R.F. (2018). 4. Royal Society Te Apārangi Gene Editing Panel. (2017). https://royalsociety.org.nz/assets/Uploads/Gene-editing-in-pest-control-technical-paper.pdf

4.11 Do-it-Yourself Synthetic Biology, biohacking

Topic	**Do-it-Yourself Synthetic Biology, biohacking**
Initiator(s)	Theo Vermeire
Sources **Causative factors** **see section 2**	3, 9, 11 a, c, g
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 2 3 3 2 1
Parallels with past emerging issues. Potential interactions with other stressors)	As SynBio advances, its methods, equipment and technologies will be cheaper, simpler and easier to use. The number and complexity of products, new pathways to risk-assessment endpoints, large range of types of products, new actors, including DIY bioengineers (also known as biohackers) and complex alignment of potential future products with agency authorities are likely to change rapidly as biotechnology advances. SynBio will likely foster citizen science, i.e. attracting DIY biologists into a field traditionally reserved for highly trained professionals. The nature of the citizen science community raises concerns that its practitioners will not abide by risk assessment and biosafety practices required by law of the professional SynBio community. The issue is not whether SynBio can be safely practiced; it is a question of whether DIY biologists will practice it safely. Accidental use or misuse of SynBio kits by consumers was identified as a cause of concern in the SCENIHR Opinion on SynBio. There is evidence that this may lead to actual risks, e.g. when a kit is contaminated by pathogenic species or malware is incorporated in DNA-samples.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background	**References**

including reliability of data, a key reference if possible any other reasons for concern.	1. SCHER (Scientific Committee on Health and Environmental Risks), SCENIHR (Scientific Committee on Emerging and Newly Identified Health Risks), SCCS (Scientific Committee on Consumer Safety), Synthetic Biology II - Risk assessment methodologies and safety aspects, Opinion, December, 2014. 2. Epstein M., Vermeire T. (2016). 3. National Academies of Sciences, Engineering, and Medicine (2017). 4. Schmidt M. (2008). 5. http://www.dw.com/en/biohacking-genetic-engineering-from-your-garage/a-42030559?maca=en-rss-en-all-1573-rdf 6. https://groups.google.com/forum/#!topic/diybio/PXeoidiWPYA 7. https://www.wired.com/story/malware-dna-hack/ 8. https://www.youtube.com/watch?v=F9HScPlBFhM 9. https://www.lgl.bayern.de/presse/detailansicht.htm?tid=680089 10. https://www.scientificamerican.com/article/mail-order-crispr-kits-allow-absolutely-anyone-to-hack-dna/?sf188033786=1

4.12 Micro and nano-plastic in the environment

Topic	**Micro and nano-plastic in the environment**
Initiator(s)	Marco Vighi, Qasim Chaudhry (SCCS)
Sources **Causative factors (see section 2 of this document)**	5 Urban and industrial waste water. Agricultural soils treated with WWTP sludge. Macroplastic debris. Tyre debris. a
Hazard (Rank features as 1,2,3 or *)	The increasing environmental pollution by plastic materials has recently come to the attention of authorities around the world. For example, WHO has recently launched a health review after microplastics were found in 90% of bottled water (www.bbc.co.uk/news/science-environment-43389031). The UK has banned the use of plastic microbeads in cosmetic products (https://chemicalwatch.com/62944/uk-microbeads-ban-enters-into-force). The production, use and disposal of plastic materials is now ubiquitous. The largest area of plastic use is for packaging materials and a wide range of other objects. Plastic polymers have not been considered substances of health concern because they are generally inert in nature, and are unlikely to be absorbed in the body due to large molecular sizes. They are nevertheless highly persistent in the environment where they may end up via a variety of disposal/emission routes. Gradual degradation of plastic materials over time is known to result in microplastics - which in theory could also further degrade to nano-plastics. Some forms of microplastics are also used in cosmetic products which makes their direct emission into the aquatic environment possible. The effects of MPs on living organisms may be direct, mainly physical (damages to respiratory systems or digestive tracts) or behavioural (changes in food intake), or indirect, determined by the uptake of chemicals adsorbed on the plastic. The actual relevance of these effects is dependent on the realistic exposure in soil and water environments, which is still largely unknown.
- uniqueness **- soundness** **- severity** **- scale** **- urgency** **- interactions**	3 * * 3 3 *
Parallels with past emerging issues.	This is a relatively new topic and after decades of ignorance has recently come under the focus of attention of researchers, authorities and the

Potential interactions with other stressors)	general public. Possible interactions between MPs and potentially toxic chemicals.
Preliminary Estimation of importance (*, 1, 2 or 3 where *=uncertain and 3 is high)	* Considering the amount of the emissions, the issue could be of high importance. However the available information on actual exposure in the different environmental compartments as well as on the effects on living organisms is still highly controversial. The sampling methods for microplastic as well the analytical methods are not standardised and different procedures, providing different results, are used in monitoring studies. Therefore, measured data in the aquatic environment reported in the literature are often difficult to compare. As for the effect assessment, microplastic (arbitrarily defined as bigger than 1 μm) should not cross cell membranes and, if ingested, should remain in the digestive system, producing only physical effects. On the contrary, nanoplastics can probably enter the cells and, possibly, interact with cell metabolism. However, to date, these are just hypotheses. There is some evidence in the literature of the cellular uptake of nanoplastic but the threshold below which this may occur is unknown, as well as the type of biological effects. These uncertainties point to the need for a better assessment of hazard and risk. It is opinion of the SCHEER that the standardisation of methods for assessing exposure, as well as the development of methods for assessing the different behaviour in living organisms of micro and nano plastics, represent urgent priorities.
Background including reliability of data, a key reference if possible any other reasons for concern.	The occurrence of micro and macro plastic in the environment is recognised as one of the most serious environmental problems at the global level. In particular, microplastics (MPs) in the marine environment have been extensively studied for a long time (GESAMP, 2015), while, in freshwater, the problem is much less known (Breuninger *et al*., 2017). **References** 1. GESAMP (2015). Joint Group of Experts on the Scientific Aspects of Marine Environmental Protection). 2. Breuninger E., Bänsch-Baltruschat B., Brennholt N., Hatzky S., Kochleus C., Reifferscheid G., Koschorreck J. (2017). 3. Wright S.L., Kelly F.J. (2017). 4. Andrady A.L. (2017). 5. Galloway T.S., Cole M., Lewis C. (2017). 6. www.efsa.europa.eu/en/press/news/160623 7. www.independent.co.uk/environment/microplastics-microbeads-health-risks-investigations-uk-government-ban-possibility-a7416271.html

4.13 Nanoparticles released from Building Materials and construction waste to the Environment

Topic	**Nanoparticles released from Building Materials and construction waste to the Environment**
Initiator(s)	Rodica-Mariana Ion
Sources **Causative factors** **(see section 2 of this document)**	1,4,5 a, **Sources:** Nanomaterials are found in construction products, primarily in surface coatings, concrete, window glass, insulation and steel. Not all of them contain nanoparticles. However, some nanomaterials may be hazardous due to the presence of very small particles and the similarities observed between some nanomaterials and asbestos fibres. The involved materials have potential impact on both human health and on the environment. The most used are: TiO_2, SiO_2, ZnO, Ag, CuO, and $CaCO_3$. Also, there are some nanomaterials in cement such as: SiO_2, Al_2O_3, Fe_2O_3, ZrO_2, carbon nanotubes (CNTs) and carbon nanofibers (CNFs). Titanium dioxide particles or antimicrobial silver nanoparticles or even carbon nanotubes (CNTs) provide concrete with self-cleaning properties, or antimicrobials or give it with improved strength and potentially electrical conductivity. **Causative factors:** various nanomaterials used in the building industry and their potential release from paint waste, during the renovation and demolition processes, during recycling, landfilling and incineration technologies. The release of NPs may occur when the coatings are not adequately fixed to the stone or when they are not sufficiently effective to prevent stone degradation and crumbling. Those NPs that end up in the water systems can adversely affect aquatic and marine life and in the soil, essential microbial interactions may be interfered with, affecting functional diversity.
Hazard (Rank features as 1,2,3 or *) **-uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 3 * * 3 3 *
Parallels with past emerging issues. Potential interactions with other stressors	Several building materials exist from the older building technologies still exist and are used because of their proximity, availability and geographical location. Such material includes mud bricks (adobe), stones, cobs and wood to mention a few. These the traditional building materials are stereotypically binary: earth related material and wood related. There is currently lack of regulations requiring labeling or other listings of

	these materials containing nanomaterials The possible release of nanomaterials into the environment by this route must therefore be taken into consideration, as follows: [i] identify the sources and the flows of released nanomaterials in construction waste, [ii] identify the potential exposure pathways of released nanomaterials for humans and other organisms, [iii] identify the release of released nanomaterials to technical compartments and the environment and [iv] identify the hazard they represent for organisms. After gathering all these elements, a characterisation of risk is plausible.
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	3
Background including reliability of data, a key reference if possible any other reasons for concern.	**References** 1. Christine G., Benjamín O.O.M. (2018). 2. Shandilya N., *et al*. (2015). 3. Hincapié I., Caballero-Guzmán A., Nowack B. (2015)., 4. Mitrano D.M., Mehrabi K., Dasilva Y.A.R. Nowack B. (2017). 5. Dulger M., Sakallioglu T., Temizel I., Demirel B., Copty N.K.,. Onay T.T., Uyguner-Demirel C.S., Karanfil T. (2016). 6. Sakallioglu T., Bakirdoven M., Temizel I., Demirel B., Copty N.K., Onay T.T., Uyguner Demirel C.S., Karanfil T. (2016). 7. van Broekhuizen P., van Broekhuizen F., Cornelissen R., Reijnders L. (2011).. 8. Hanus M.J., Harris A.T. (2013).

4.14 Environmental factors and the Human Microbiome

Topic	Environmental factors and the Human Microbiome
Initiator(s)	Teresa Borges
Sources **Causative factors** **(see Section 2 of this document)**	 b and f *Microbiome* refers to "the entire habitat, including the microorganisms (bacteria, archaea, lower and higher eukaryotes and viruses), their genome (i.e., genes), and surrounding environmental conditions" (Marchesi and Ravel 2015). A key aspect of the human microbiome is the variation in its composition and function observed among populations, over the human life span, and between body sites such as the gut, skin, or respiratory microbiome. Research is showing that the human microbiome has a modulating role between environmental factors and the health status (Fallani 2016; Gibson *et al*. 2016; Yassour *et al.* 2016; Chu *et al*. 2017). On the other hand, microbial changes in the human microbiome are being linked to an array of neurological, gastrointestinal, metabolic, oncologic, hepatic, respiratory and auto immune disorders (Lynch and Pedersen 2016). In this sense, it seems sensible to have a better understanding on the relation between human exposure to environmental stressors, changes occurring in the human microbiome and onset of certain health conditions. Additionally, there is sufficient scientific knowledge on toxicity and risk assessment of a great number of environmental chemicals, generated during the last decades under several regulatory frameworks (WHO, IPCS, EPA, EFSA, ECHA) that can contribute substantially to gaining a better understanding of the underlying critical factors.
Hazard (Rank features as 1,2,3 or *) **- uniqueness** **- soundness** **- severity** **- scale** **- urgency** **- interactions**	 2 2 * 2 2 3
Parallels with past emerging issues. Potential interactions with other stressors	The structure and function of the human microbiome in both disease and healthy states have been benefiting from improvements in high-throughput and accuracy of DNA sequencing of the genomes of microbial communities that are associated with human samples, complemented by OMICS analysis (transcriptomes, proteomes, metabolomes and immunomes) and by mechanistic experiments in model systems. Also, as a result of international projects dedicated to the Human Microbioma, gut microbioma has been subjected to intensive research and quality criteria and standardisations have been developed i.e. reference microbiota strains, in housing protocols, human sampling protocols. Presently, gut

	microbes can be transplanted effectively and under experimental controlled conditions into germ-free mice to recapitulate their associated phenotypes (Blanton *et al*., 2016). Also, Schwarzer *et al.* (2016) showed in mice that strains of *Lactobacillus plantarum* in the gut microbiota sustained growth hormone activity via signalling pathways in the liver, thus overcoming growth hormone resistance. This evidence strengthens the correlation between the changes in children's gut microbiota composition and children's growth factors, therefore showing that beneficial gut microbes can potentially be exploited to resolve undernutrition syndromes in children. It is expected that in the near future, metrics and biomarkers will be identified to set more effective measures and health therapies (Dietert and Silbergeld 2015).
Preliminary Estimation of importance (*,1,2 or 3 where *=uncertain and 3 is high)	2
Background including reliability of data, a key reference if possible any other reasons for concern.	**References** 1. Gilbert J.A., Blaser M.J., Caporaso J.G., Jansson J.K., Lynch S.V., Knight R. (2018). 2. Schwarzer M., *et al* (2016). 3. Blanton L.V*. et al.* (2016). 4. Collado M.C. *et al.* (2016) 5. Fallani 2016; Gibson *et al.* 2016; Yassour *et al*. 2016; Chu et al. 2017. 6. Lynch S.V., Pederson O. (2016) 7. Dietert R.R. and Silbergeld E.K. (2015).

5. CONCLUSIONS

The SCHEER identified 14 emerging issues to bring to the attention of the Commission services. The overall prioritisation scores (*, 1,2,3 where *=uncertain and 3 is high) are as follows:

4.1	Personal communication and listening devices	1
4.2	Virtual reality	3
4.3	E-cigarette and chronic diseases	3
4.4	Potential effects on wildlife of increases in electromagnetic radiation	3
4.5	Chemicals in recycled materials, an issue in a circular economy	3
4.6	Pharmaceuticals (human and veterinary) and illicit drugs in wastewater and surface waters	3
4.7	Substance Mobility: a new criterion in chemicals regulation	3
4.8	Drinking water treatment interactions with compounds and potential health effects	2
4.9	Per- and polyfluorinated organic substances	2-3
4.10	New RNA pesticides and gene editing to reduce/eradicate pest populations	3
4.11	Do-it-Yourself Synthetic Biology, biohacking	3
4.12	Micro and nano-plastic in the environment	*
4.13	Nanoparticles released from Building Materials and construction waste to the Environment	3
4.14	Environmental factors and the Human Microbiome	2

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