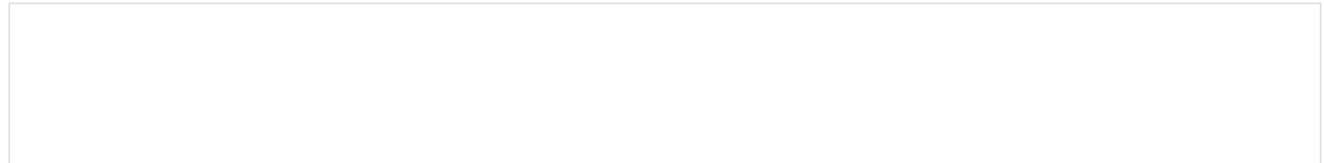


ENVIRONMENT

Are Contaminants Silencing Our Genes?

Some chemicals may leave people vulnerable to diseases like cancer and diabetes, not by mutating genes but by turning them off or on at the wrong time

By Bette Hileman, Environmental Health News on August 3, 2009



Each of us starts life with a particular set of genes, 20,000 to 25,000 of them. Now scientists are amassing a growing body of evidence that pollutants and chemicals might be altering those genes—not by mutating them, but by sending subtle signals that silence them or switch them on at the wrong times.

Last week, several dozen researchers and experts convened by the National Academies tackled this complicated topic, called epigenetics, at a two-day workshop in Washington, D.C. They discussed new findings that suggest chemicals in our environment and in our food can alter genes, leaving people vulnerable to a variety of diseases and disorders, including diabetes, asthma, cancer and obesity. They also considered whether regulatory agencies and industry should start testing the thousands of chemicals in use today for these effects.

“There is little doubt these epigenetic effects are important. The next question is how we test for effects,” said William H. Farland, professor of environmental and radiological health sciences at Colorado State University. “We don’t need to abandon current approaches to chemical testing. When testing chemicals in animals, we may just need to add some new endpoints.”

Exposure to gene-altering substances, particularly in the womb and shortly after birth, “can lead to increased susceptibility to disease,” said Linda S. Birnbaum, who was named director of the National Institute of Environmental Health Sciences and of the National Toxicology Program in December. “The susceptibility persists long after the exposure is gone, even decades later. Glands, organs, and systems can be permanently altered.”

“There is a huge potential impact from these exposures, partly because the changes may be inherited across generations. You may be affected by what your mother and grandmother were exposed to during pregnancy,” Birnbaum said.

What a pregnant mother eats and the chemicals she is exposed to can affect her offspring without causing mutations in the DNA, the experts said. Instead, such exposures can disrupt the way that genes behave, according to both animal and human studies. These changes, in turn, can be passed on to the next generations.

Some environmental chemicals enable methyl groups (carbon atoms with three hydrogen atoms attached) to attack genes, which turns them off or mutes them, at a time when they should be turned on. When genes are turned off, they can't direct the manufacture of proteins that are essential for proper cell function. Chemicals also can uncoil parts of the chromosome, causing genes to be expressed, or turned on, at inappropriate times.

An example is asthmatic children. Wan-Yee Tang, a researcher at the University of Cincinnati, found that children in New York City exposed in the womb to high levels of polycyclic aromatic hydrocarbons (PAHs), common air pollutants from traffic, were much more likely to have asthma than those who were not exposed. By studying cord blood, she found that a particular gene (ACSL3) was methylated in the asthmatic children and unmethylated in the unexposed children, and concluded that the abnormal methylation patterns probably caused the asthma.

The finding could in part explain why worldwide asthma rates have skyrocketed in much of the world, reaching epidemic proportions among children. In the boroughs of New York City with the worst air pollution, about 25 percent of children are asthmatic.

Epigenetic changes also have been observed in children conceived with assisted reproductive technologies, said Richard Meehan of the Medical Research Council in Scotland.

One of the disorders that occurs at a higher rate in these children is Beckwith-Wiedemann syndrome, which is characterized by abdominal wall defects and a higher risk of certain childhood cancers. The culture medium where fertilized eggs are grown for several days before implantation probably causes the syndrome, he said. It appears that all the different media used for the eggs might be problematic because they contain chemicals that stimulate the addition of methyl groups to the cells.

The scientists at the workshop said it's important to understand epigenetics not only to figure out which chemicals might endanger public health, but to find new ways to prevent or treat diseases.

Scientists are just now beginning to figure out normal methylation patterns in the genome so

they can learn what is abnormal, said [Karl T. Kelsey](#), professor of community health and pathology at Brown University in Rhode Island. As a result of this new understanding, epigenetic therapies have been developed for some types of cancers, and some have been successful in clinical trials, he said. Unlike traditional cancer drugs, which kill cells, the new drugs simply change how the cells act.

Research with rats shows that gene-altering chemicals can change animals' brains—in some cases, in a beneficial way.

[Moshe Szyf](#), a pharmacology and therapeutics professor at McGill University Medical School in Montreal, found that rats that received healthy doses of maternal licking as pups grew up to be calmer than pups who had inattentive mothers. The maternal grooming brought about a chemical change in the part of the pup's brain that produces stress hormones, he said.

The rats reared by attentive mothers had different levels of corticoid gene expression and lower levels of stress hormones than those reared by inattentive mothers. Szyf found he could cure the stressed rats by injecting a chemical called TSA into their brains, which reversed the inappropriate methylation caused by inattentive mothering.

This understanding of epigenetics may lead to new medications for treating human problems. By using approaches similar to those used in the rat study, Szyf is hoping to find drugs that will help alleviate human psychiatric conditions.

Szyf also studied the preserved brains of [suicide](#) victims and of people who died suddenly from causes other than suicide. He found that certain genes in the suicide victims were methylated, or turned off. In contrast, those same genes were not methylated in the victims who died by other means. Abnormal methylation patterns could cause depression in some people, he said.

Some compounds, such as nickel, chromium and arsenic, are well-known carcinogens—not because they are toxic to cells but because of their epigenetic effect, said [Max Costa](#), a New York University professor of environmental medicine and pharmacology. They increase DNA methylation, which results in gene silencing and cell transformation and leads to cancer, he explained.

Researchers at the meeting spent a great deal of time discussing whether and how to test chemicals for their ability to cause epigenetic changes.

Most researchers there agreed that compounds need to be tested for epigenetic effects. But practical testing of the 80,000 or so chemicals in commerce would require rapid screens that would prioritize the compounds into high, medium, and low-risk groups. Those at high risk for epigenetic effects could then be subjected to more definitive and expensive tests.

John M. Grealley, associate professor at the Albert Einstein College of Medicine in New York City, pointed out that no single test is ideal for detecting epigenetic effects.

“All of the assays have drawbacks,” he said. For example, one assay requires immediate sample processing so it cannot be used on stored samples.

Nevertheless, many researchers said that testing chemicals for epigenetic changes can begin soon.

“The fact that we don’t know a great deal about this area doesn’t mean it’s daunting,” said George Daston, research fellow at Procter & Gamble. “We just need to build on what we have. Microassays already show how chemical exposures change the gene expression in certain parts of the genome. The fact that we don’t know a lot doesn’t mean we can’t start testing quickly.”

Birnbaum, who formerly was head of experimental toxicology at the U.S. Environmental Protection Agency, said regulators and industry don’t have to start from square one.

“We’re already marching down this road,” said Birnbaum. “The National Toxicology Program is already talking about including some epigenetic studies in the program.”

The most important public health issue that arises from epigenetics, Birnbaum told Environmental Health News, is that the current environment may not be the crucial factor to consider when examining what causes diseases.

“Asking heart attack victims what they ate this year or last may be far less important than what they were exposed to in the womb and shortly after birth,” she said.

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ABOUT THE AUTHOR(S)

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District of Oregon

Portland Division

AHM, by and through
her Guardian *ad litem* and father,
David Mark Morrison, and
David Mark Morrison, individually,

v.

Portland Public Schools,

Defendant.

Civil Action No. 3:11-cv-00739-MO

**Amended Declaration of
Dr. David O. Carpenter, M.D.**

I, Dr. David O. Carpenter, M.D., under penalty of perjury pursuant to 28 U.S.C. § 1746, hereby make the following declaration in support of an injunction against Portland Public Schools' use of WI-FI:

1. I am a public health physician, educated at Harvard Medical School. My current title is Director of the Institute for Health and the Environment at the University at Albany and Professor of Environmental Health Sciences within the School of Public Health. Formerly, I was the Dean of the School of Public Health at the University of Albany and the Director of the Wadsworth Center for Laboratories and Research of the New York State Department of Health.

2. I served as the Executive Secretary to the New York State Powerlines Project in the 1980s, a program of research that showed children living in homes with elevated magnetic fields coming from powerlines suffered from an elevated risk of developing leukemia. After this I became the spokesperson on electromagnetic field (EMF) issues for the state during the time of my employment in the Department of Health. I have published several reviews on the subject and have edited two books.

3. I am a Co-Editor and a Contributing Author of the *BioInitiative: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)*, www.bioinitiative.org. It documents bioeffects, adverse health effects and public health conclusions about impacts of electromagnetic radiation (electromagnetic fields including extremely-low frequency ELF-EMF and radiofrequency /microwave or RF-EMF fields). The public health chapter from this report was subsequently published in a peer-reviewed journal.

4. Additionally, I am a Co-Author of *Setting Prudent Public Health Policy for Electromagnetic Field Exposures*, *Reviews on Environmental Health*, Volume 23, No 2, 2008, attached as Addendum A-2.

5. In addition, in 2009, I was invited to present to the President's Cancer Panel on the subject of powerline and radiofrequency fields and cancer, and have testified on this issue before the United States House of Representatives.

6. In sum, I am a public health physician, professor and former public health school Dean with expertise in electrophysiology, low-frequency electromagnetic fields bioeffects, and

radiofrequency (RF) and microwave (MW) radiation bioeffects.

7. WI-FI deploys pulse-modulated (“PM”) microwave (“MW”) radiation (within the larger RF radiation spectrum) with a carrier frequency that is similar to that used by a microwave oven: about 2.45 GHz. This is the “Agent”. The 2.45 GHz frequency was chosen for the oven because of its wavelength and harmonic resonance with the water molecule, to ensure the most efficient absorption by living tissues and effective heating by way of the agitation of water at the molecular level. The pulse-modulation of a wave with lower frequencies in addition to the high-frequency carrier signal, increases the exposure complexity and in turn the bioeffects in an exposed population.

8. In the context of school development, WI-FI exposes building occupants including children and adults constantly from both computers and infrastructure antennas. Duration may be an even more potent contributing factor to RF/MW radiation bioeffects than exposure levels. Chronic, such as all-day, school exposure, is more likely than short and intermittent exposure, such as cell phone use, to produce harmful health effects, and is likely to do so at lower exposure levels.

9. Persons stationed close to school computers with WI-FI and especially those very near to any WI-FI infrastructure will receive considerably higher exposure than do others.

10. It is generally accepted within the relevant scientific community and has been established beyond any reasonable doubt that adverse human health effects occur at far lower levels of RF/MW radiation exposure than those that cause noticeable heating, particularly where the wavelength approaches body-part size and thus maximizes absorption, where the wavelength has resonance with the water molecule, where there is more complex, modulated wave, where there is chronic exposure duration, and where exposed persons lack the capacity voluntarily to remove themselves from radiation sources.

11. Some effects are shown to occur at several hundred thousand times below the FCC public exposure guidelines, which are set based on the fallacious assumption that there are no adverse health effects at exposures that do not cause easily measureable heating. FCC guidelines

also only apply to 30-minute public exposures; therefore do not even infer safety at durations >30 minutes, such as in a school setting.

12. Exposure to high-frequency RF and MW radiation and also the extreme low frequency (ELF) EM fields that accompany WI-FI exposure have been linked to a variety of adverse health outcomes. Some of the many adverse effects reported to be associated with and/or caused by ELF fields and/or RF/MW radiation include neurologic, endocrine, immune, cardiac, reproductive and other effects, including cancers.

13. Studies of isolated cells have shown that RF/MW exposures may cause changes in cell membrane function, cell communication, metabolism, activation of proto-oncogenes, and can trigger the production of stress proteins at exposure levels below FCC guidelines and also at and less than school WI-FI exposure levels and parameters. Resulting effects in cellular studies include without limitation DNA breaks and chromosome aberrations, cell death including death of brain neurons, increased free radical production, activation of the endogenous opioid system, cell stress and premature aging.

14. Human studies of comparable RF/MW radiation parameters show changes in brain function including memory loss, retarded learning, performance impairment in children, headaches and neurodegenerative conditions, melatonin suppression and sleep disorders, fatigue, hormonal imbalances, immune dysregulation such as allergic and inflammatory responses, cardiac and blood pressure problems, genotoxic effects like miscarriage, cancers such as childhood leukemia, childhood and adult brain tumors, and more.

15. There is consistent evidence for increased incidence of effects in individuals who live near to high-power short-wave, AM, FM and TV transmission towers. This is particularly relevant because, like WI-FI, radio-TV transmission towers give continuous, whole-body radiation, not just radiation to the head, constantly.

16. Since WI-FI transmitters, both infrastructural and on computers, are indoors, where children and teachers may be very close by, and since WI-FI, at 2.45 GHz, deploys a

wavelength, at ~12.2 cm or ~ 4.8 inches, more absorbable by children's and adults' bodies and brains than radio-TV wavelengths, the harmfulness of WI-FI radiation likely exceeds that of radio-TV towers.

17. Like second-hand smoke, EMF and RF/MW radiation involve complex mixtures, where different frequencies, intensities, durations of exposure(s), modulation, waveform and other factors are known to produce variable effects, often more harmful with greater complexity. Decades of scientific study have produced substantial evidence that EMF and RF/MW radiation may be considered neurotoxic, carcinogenic and genotoxic. Sources of fields and radiation, but are not limited to: power lines, navigational radar, cell phones, cordless phones [or Digitally Encoded Cordless Transmission Devices (D.E.C.T.) phones], cell towers, 'smart' meters and their grids or infrastructure, "smart" boards, meters and grids, WiMax and wireless internet (WI-FI).

18. The RF/MW radiation and low-frequency EMF science that currently exists includes tens of thousands of studies dating back to the 1920s. On the basis of this vast body of literature, many public health experts believe, myself included, that it is likely society will face epidemics of neurotoxic effects and degeneration, cancers and genotoxicity in the future, resulting from the extreme and mostly involuntary exposure to RF/MW radiation and EMFs. WI-FI radiation in schools exceeds natural background levels of microwave radiation by trillions of times. Thus, it is important that all of us restrict our use of cell phones, and be as free as possible from exposure to unnatural, background sources of MW radiation, particularly WI-FI.

19. In public health science, it is generally accepted fact that vulnerable subgroups exist within any human population. This is also recognized specifically for RF/MW radiation and fields. These groups include children, pregnant women, the elderly and those with preexisting illnesses and/or impairments. Children are more vulnerable to RF/MW radiation because of the susceptibility of their developing nervous systems. RF/MW penetration is greater relative to head size in children, who have a greater absorption of RF/MW energy in the tissues of the head at WI-FI frequencies.

Such greater absorption results because children's skulls are thinner, their brains smaller, and their brain tissue is more conductive than those of adults, and since it has a higher water content and ion concentrations. The Presidential Cancer Panel found that children 'are at special risk due to their smaller body mass and rapid physical development, both of which magnify their vulnerability to known carcinogens, including radiation.'

http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf

20. FCC public RF/MW radiation exposure guidelines are based on the height, weight and stature of a 6-foot tall man, not children or adults of smaller stature. The guidelines do not take into account the unique susceptibility of growing children to exposures. Since children are growing, their rate of cellular activity and division is more rapid, and they are at more risk for DNA damage and subsequent cancers. Growth and development of the central nervous system is still occurring well into the teenage years, such that the neurological impairments predictable by the extant science may have great impact upon development, cognition, learning, and behavior. Prenatal exposure has been identified as a risk factor for childhood leukemia, and is associated with miscarriage. Children are largely unable to remove themselves from exposures to harmful substances in their environments. Their exposure is involuntary.

21. When WI-FI is in operation in a school, children and their parents have no choice but to allow the school to expose them to trillions of times higher microwave radiation than exists naturally on Earth at the same frequencies. Children and other building users are exposed to as much as 30-40 hours per week of constant, digitally encoded WI-FI signals from each wireless device and infrastructural antenna in a school building. Based upon a review of the Mount Tabor WI-FI Floor Plan, a given child is subject to direct signals from multiple WI-FI transmitters, including rooms full of students and teachers transmitting numerous laptop and other wireless signals. There is a major legal difference between an exposure that an individual chooses to accept and one that is forced upon a person, especially a dependent, who can do nothing about it.

22. WI-FI in the Portland Schools deploys similar PM MW radiation, at 2.45 and 5 GHz, to that of cell and cordless phones and their infrastructure. There is clear and strong evidence that intensive use of cell phones increases incidence of brain cancer, tumors of the auditory nerve, and cancer of the parotid gland, the salivary gland in the cheek by the ear. Cell and cordless phone radiation closely resembles that of WI-FI radiation exposure, except that WI-FI is more hazardous by way of frequency, duration, and the involuntary nature of exposure. While a cell or cordless phone is used only intermittently and primarily voluntarily, a WI-FI radiation microenvironment is constant in duration, with unavoidable radiation exposure even when nearby students are not actively using it. Because WI-FI radiation is essentially the same as, but more hazardous than, that for cell and cordless phones, there is every reason to understand that the health effects will be the same or worse, varying in relation to the total dose of radiation, and intensified by the constancy of duration. There is evidence from Scandinavian studies of cell phone usage that children who use cell phones are about five times more likely to develop brain cancer than if their usage starts as an adult. Thus, it is especially necessary to protect children from pulse-modulated MW radiation such as both cell phones and WI-FI deploy.

23. Based on a high degree of scientific certainty, Portland Public Schools' use of WI-FI is causing and will continue to cause AHM, other students, and school staff and faculty adverse health effects, and should be discontinued immediately. Educating by way of the Internet via cabled systems only decreases MW radiation exposure and is of minimal expense.

24. Having reviewed hundreds, possibly thousands, of studies in RF/MW radiation and ELF fields, published from decades ago to the present, I would provide you the following primary evidence, without limitation. Due to the active suppression of the RF/MW literature, some researchers in public health science are less aware of these studies. However, the forefront experts specializing in these areas, RF/MW radiation and ELF fields, recognize the certainties in this large body of scientific literature, which establishes without limitation that PM MW radiation with chronic duration is quite harmful to humans, particularly children, as well as to animals and plants.

25. It is not surprising that even as of 1990, the US Environmental Protection Agency ("EPA") had determined RF/MW radiation a "probable carcinogen". Now that we have much more confirming study in the interim, the conclusion is yet more certain. And when we focus on MW radiation, particularly pulse-modulated radiation, on long, non-intermittent duration and on more vulnerable subgroups such as children, we see that the cancer outcome is very certain, indeed. Amongst the epidemiologic studies showing cancer outcomes, the following are particularly strong:

- a. Dode AC, Leao M, Tejo FdeAF, gomes ACR, Dode DC, Dode MC, Moreira CW, Condessa VA, Albinatti C and Calaffa WT. Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais State, Brazil. *Sci Total Environ* 409: 3649-3665:2011. This study shows higher rates of cancer in people living close to cell phone towers than for people living further away. Cell phone radiation is similar to but likely not as harmful as 2.45 GHz radiation from WI-FI. The exposure levels in this study are lower than those that Portland school building occupants receive from WI-FI.
- b. Oberfeld G. Environmental Epidemiology Study of Cancer Incidence in the Municipalities of Hausmannstatten & Vasoldsberg (Austria), 2008. This government-commissioned study found significantly increased cancer risk relative to a lower-exposure reference category, 23x higher for breast cancer and 121x higher for brain tumors, with strong exposure-effect relations.
- c. Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, Barca A and Perucci CA. Adult and childhood leukemia near a high-power radiostation in Rome, Italy. *Am J Epidemiol.* 155: 1098-1103: 2002. The authors show that there is a significant elevation of childhood leukemia among residents living near to Vatican Radio, and that the risk declines with distance away from the transmitter. This is RF radiation in frequencies similar to that of WI-FI.

- d. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM and Pack JK. Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am J Epidemiol* 166: 270-279: 2007. Leukemia and brain cancer in children in Korea were investigated in relation to residence within 2 km of AM radio transmitters. There was a significant elevation in rates of leukemia but not of brain cancer. WI-FI radiation is more harmful than AM.
- e. Park SK, Ha M, Im HJ. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. *Int Arch Occup Environ Health*. 2004 Aug;77(6):387-94. This study found higher mortality areas for all cancers and leukemia in some age groups in the area near the AM towers.
- f. Hallberg O. Johansson O. *Med Sci Monit* 2004 Jul;10(7):CR336-40. Malignant melanoma of the skin – not a sunshine story! Increased incidence and mortality from skin melanoma are concluded to result from continuous disturbances of cell repair mechanisms by body-resonant EMFs from FM/TV networks.
- g. Hallberg O. Johansson O. 2005. FM Broadcasting exposure time and malignant melanoma incidence, *Electromagnetic Biology and Medicine* 24;1-8. Age-specific incidence of malignant melanoma of the skin is related to FM broadcasting radiation at whole-body resonant frequencies. This is very relevant to children, since the smaller wavelengths of WI-FI are at resonant frequencies with dimensions of the human head, particularly the child's head.
- h. Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I, Elliot P. Cancer Incidence near radio and television transmitters in Great Britain. I – Sutton-Colfield transmitter, and II. A1 high-power transmitters. *Am J Epidemiol* 1997; 145(1):1-9 and 10-17. In the first study, there was a statistically significant

increase in cancer; in the second, a small but significant increase in adult leukemia.

i. Hocking B, Gordon IR, Grain HL, Harfield GE. Cancer incidence and mortality and proximity to TV towers. *Medical J of Australia*. 1985;165:601-605. At extremely low exposure levels, there was an association between increased childhood leukemia incidence and mortality and proximity to TV towers. TV radiation, in the VHF and UHF bands, is similar to but not as harmful as WI-FI radiation at 2.45 GHz.

j. Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: A nested case-control study. *Am J Epidemiol* 1996; 143:480-6. This study found an association between exposure to ELF and RF/MW radiation and brain tumors.

k. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Sci Total Environ* 1996;180:9-17. This study showed huge increases in leukemia and Non-Hodgkin's lymphomas. Though exposure levels are higher in this study than they would be with school WI-FI, it is possible that certain students or teachers stationed immediately next to the WI-FI infrastructure could receive comparable levels in radiation peaks.

26. Additional studies show neurologic, immune, endocrine, reproductive and cardiac, adverse health effects from low-dose, chronic exposure to RF/MW radiation in humans:

a. Papageorgiou CC, Hountala CD, Maganioti AE, Kyprianou MA, Rabavilas AD, Papadimitriou GN, Capsalis CN. Effects of WI-FI signals on the p300 component of event-related potentials during an auditory listening task. *J Integr Neurosci* 2011 Jun;10(2):189-202. This study concludes that WI-FI exposure may exert gender-related alterations on neural activity.

- b. Altpeter ES, Roosli M et al. Effect of Short-wave magnetic fields on sleep quality and melatonin cycle in humans: The Schwarzenburg shut-down study. *Bioelectromagnetics* 27:142-150, 2006. Sleep quality improved and melatonin excretion increased when the transmitter was shut down.
- c. Abelin T et al. Sleep disturbances in the vicinity of the short-wave broadcast transmitter Schwarzenburg. *Somnologie* 9:203-209, 2005. There is strong evidence of a causal relationship between operation of a short-wave radio transmitter and sleep disturbances in the surrounding population.
- d. Hutter HP et al. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup Environ Med* 2006;63:307-313, 2006. There was a significant relation of some symptoms, especially headaches, to measured power density, as well as effects on wellbeing and performance.
- e. Preece AW, Georgious AG, Duunn EJ, Farrow SC. *Occup Environ Med* 2007 Jun;64(6):402-8. Compared to control village, there were highly significant differences in the reporting of migraine, headache and dizziness military and cell phone antenna systems.
- f. Buchner K, Eger, H. Changes of clinically important neurotransmitters under the influence of modulated RF fields – a long-term study under real-life conditions. *Umwelt-Medizin-Gesellschaft* 24(1):44-57, 2011. There is clear evidence of health-relevant effects, including increase in adrenaline/noradrenaline, subsequent decrease in dopamine from a new MW-emitting base station. During counterregulation, trace amine PEA decreased and remained decreased. Clinically documented increases in sleep problems, cephalgia, vertigo, concentration problems and allergies followed the onset of new microwave transmissions.

- g. Eliyahu I, Luria R, Hareuveny R, Margalioth M, Neiran N and Shani G . Effects of radiofrequency radiation emitted by cellular telephones on the cognitive functions of humans. *Bioelectromagnetics* 27: 119-126: 2006. A total of 36 human subjects were exposed to PM MW and were tested on four distinct cognitive tasks. Exposure to the left side of the brain slows left-hand response time in three of the four tasks.
- h. Barth A, Winker R, Ponocny-Seliger E, Mayrhofer W, Ponocny I, Sauter C and Vana N. *Occup Environ Med* 65: 342-345: 2008. A meta-analysis for neurobehavioural effects due to electromagnetic field exposure emitted by GSM mobile phones. The authors looked at 19 studies of cognitive function in cell phone users, and found in the meta-analysis that there is evidence for a decreased reaction time, altered working memory and increased number of errors in exposed persons.
- i. Augner C, Hacker GW, Oberfeld G, Florian M, Hitzl W, Hutter J and Pauser G. Effects of exposure to base station signals on salivary cortisol, alpha-amylase and immunoglobulin A. *Biomed Environ Sci* 23: 199-207: 2010. This was a human experimental study with exposure to PM MW radiation wherein immune indicators were monitored after five 50-minute sessions. The researchers found dose-dependent changes in cortisol and alpha-amylase.
- j. Avendano C, Mata A, Sanchez Sarimiento CA and Doncel GF. Use of laptop computers connected to internet through WI-FI decreases human sperm motility and increases sperm DNA fragmentation. *Fert Steril*, 2012, In press. In this study human sperm were exposed to WI-FI from a laptop, and were found to show reduced motility after a 4-hour exposure. The results are consistent with other publications (see Agarwal et al., *Fert Steril* 89: 124-128: 2008) that reported that those who use cell phone regularly have reduced sperm count.

k. Baste V, Riise T and Moen BE (2008) *Int J Epidemiol* 23: 369-377: 2008. Radiofrequency electromagnetic fields: male infertility and sex ratio of offspring. This is a study of Norwegian Navy personnel chronically exposed to RF fields on the job. The rates of infertility were related to level of exposure in a dose-dependent fashion.

27. Many toxicologic and other animal studies, of which the following are but a few, support conclusions of cancer, genotoxicity, neurotoxicity and other health outcomes from RF/MW radiation.

a. Sinha R. Chronic non-thermal exposure of modulated 2450 MHz microwave radiation alters thyroid hormones and behavior of male rats. *Int. J. Radiation Biol.* 84:6:505-513, 2008. This study of 2.45 GHz at levels and durations comparable to and less than those of school WI-FI concluded that the radiation was sufficient to alter the levels of thyroid hormone as well as emotional reactivity compared to controls.

b. Nittby H, Grafstrom G, Tian DP, Malmgren L, Brun A, Persson BRR, Salfors LG and Eberhardt J. *Bioelectromagnetics* 29: 219-232: 2008. This study showed cognitive impairment in rats after long-term exposure to PM MW radiation. This study of rats shows that after 2 hours per week for 55 weeks there was impaired memory for objects in exposed as compared to sham animals.

c. Kimmel S et al. Electromagnetic radiation: Influences on honeybees (*Apis mellifera*). A significant difference between non-exposed and fully irradiated bees was the result of the influence of high-frequency PM RF/MW radiation.

d. Panagopoulos DJ et al. Bioeffects of mobile telephony radiation in relation to its intensity or distance from the antenna. *Int. J Radiat Biol*, 86;(5):345-357, 2010. The PM MW radiations at 900 and 1800 MHz decreased the reproductive capacity by cell death induction, with an increased bioactivity “window” at 10

uW/cm², and still evident down to 1 uW/cm².

e. Everaert J, Bauwens D. A possible effect of electromagnetic radiation from mobile phone base stations on the number of breeding house sparrow (*passer domesticus*). *Electromagnetic Biology and Medicine*, 26:63-72, 2007.

Long-term exposure to higher-level low-intensity PM MW radiation negatively affects the abundance or behavior of House Sparrows in the wild.

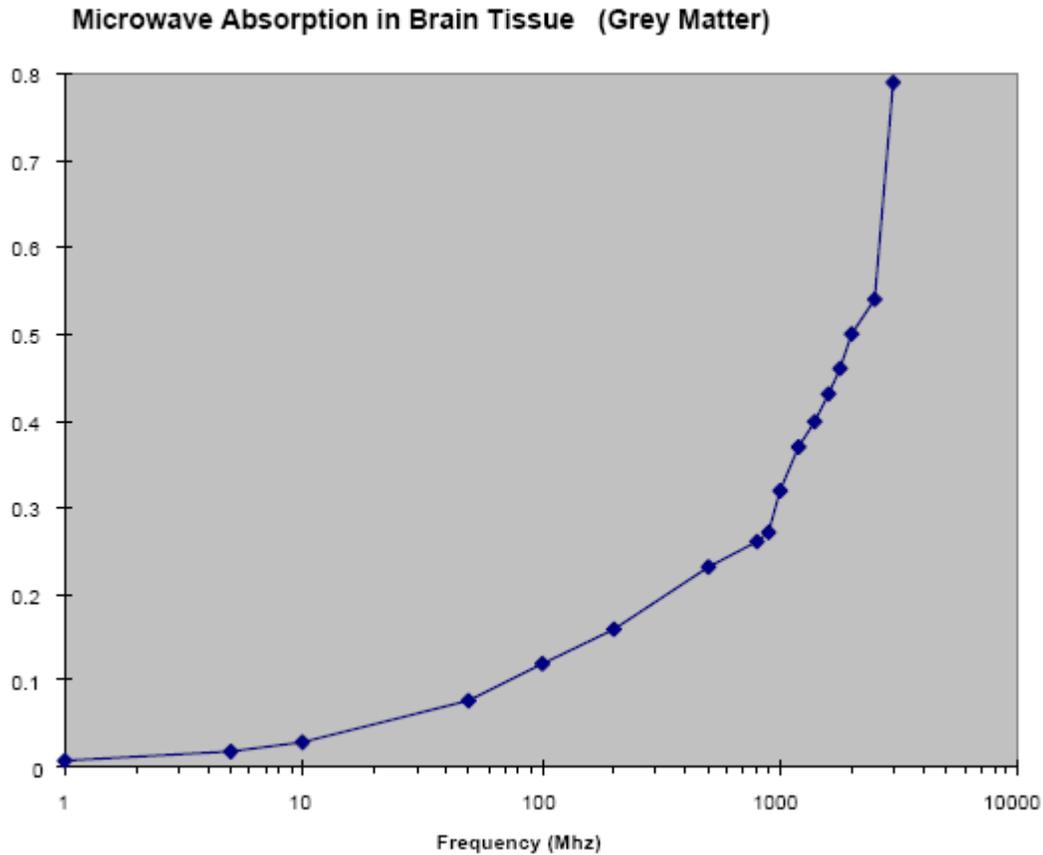
f. Magras I, Xenos T. RF Radiation-Induced Changes in the Prenatal Development of Mice. *Bioelectromagnetics* 18:455-461, 1997. Near almost 100 TV and FM broadcast transmitters, with exposure levels between 0.168 uW/cm² and 1.053 uW/cm², found in the more exposed groups testicular damage and decreasing size of litters to irreversible infertility.

g. Balmori A. Electromagnetic pollution from phone masts. *Effects on wildlife, Pathophysiology* 2009. This large review of wildlife effects concludes, “pulsed telephony microwave radiation can produce effects on nervous, cardiovascular, immune and reproductive systems,” including damage to the nervous system by altering EEG and changes to the blood-brain barrier, disruption of the 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, growth problems, problems in building the nest or impaired fertility, embryonic development, hatching percentage, genetic and developmental problems, problems of locomotion, promotion of tumors and more.

28. Exposure thresholds for harmful effects are lowered in human populations and individuals when duration is increased. Due to the variability of thresholds for harmful effects both in the population and within the individual, there is no exposure power density that is safe. The School's WI-FI deploys arguably the worst possible frequency of 2.45 GHz, that of the

microwave oven, worst because it is most absorbable by the brain and most resonant with the water molecule, such that:

- a. absorption-per-exposure is maximized, dramatically lowering effects thresholds for population and individual effects; and
- b. water molecules in tissues and cells are highly agitated.



Curry, Ph.D., *Wireless LANs in the schoolroom*

29. This above graph, from physicist William Curry PhD's presentation *Wireless LANs in the Schoolroom*, shows how absorption in brain tissue (grey matter) increases exponentially toward the ultra-high frequency (UHF) area of the microwave oven and WI-FI.

30. In the case of the Portland Schools, the additional, unused but still deployed carrier frequency of 5 GHz would likely increase absorption in other, smaller organs, such as the thyroid.

31. The graph also illustrates the problem with the drive of the wireless industry toward ever higher frequencies within the cm microwave band. While nearly all the lower frequency bands have already been allocated by the FCC for specific types of radio transmissions, and transmission of ever more information content on any given channel requires greater bandwidth, each new deployment undermines further the integrity of the population's health. Engineers who design these systems have no training that would qualify them to consider the effects on biologic systems, which is why public health scientists need to be called in to policymaking *prior to* contracting and deployment, not after the fact.

32. The following studies explain the mechanisms of interaction between RF/MW radiation and biologic systems at the cellular level.

- a. The cell membrane recognition process -- which includes signal transduction and 'heat-shock protein' release -- was first discerned by Litovitz and his co-workers at Catholic University of America in the mid-1990s.

Below are a few citations that make the point.

- i. Litovitz, T., C. Montrose, et al. (1994). "Superimposing spatially coherent electromagnetic noise inhibits field induced abnormalities in developing chick embryos." *Bioelectromagnetics* **15**(2): 105-113.
- ii. DiCarlo, A., J. Farrell, et al. (1998). "A simple experiment to study electromagnetic field effects: Protection induced by short term exposures to 60 Hz magnetic fields." *Bioelectromagnetics* **19**(8): 498-500.
- iii. Penafiel, L., T. Litovitz, et al. (1997). "Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929

- cells." *Bioelectromagnetics* **18**(2): 132-141.
- iv. Dicarlo, A. L., Michael T. Hargis, L. Miguel Penafiel, Theodore A. Litovitz, A. (1999). "Short-term magnetic field exposures (60Hz) induce protection against ultraviolet radiation damage." *International journal of radiation biology* **75**(12): 1541-1549.
 - v. Litovitz, T., C. Montrose, et al. (1990). "Amplitude windows and transiently augmented transcription from exposure to electromagnetic fields." *Bioelectromagnetics* **11**(4): 297-312.
 - vi. Litovitz, T., M. Penafiel, et al. (1997). "The role of temporal sensing in bioelectromagnetic effects." *Bioelectromagnetics* **18**(5): 388-395.
 - vii. Litovitz, T., L. Penafiel, et al. (1997). "Role of modulation in the effect of microwaves on ornithine decarboxylase activity in L929 cells." *Bioelectromagnetics* **18**: 132-141.]
 - viii. Litovitz, T., D. Krause, et al. (1993). "The role of coherence time in the effect of microwaves on ornithine decarboxylase activity." *Bioelectromagnetics* **14**(5): 395-403.
- b. Cell membrane reaction is lipid peroxidation.
 - i. Serban, M. and V. Ni (1994). "Lipid peroxidation and change of plasma lipids in acute ischemic stroke." *Romanian journal of internal medicine= Revue roumaine de médecine interne* **32**(1): 51.

- ii. Vileno, B., S. Jeney, et al. (2010). "Evidence of lipid peroxidation and protein phosphorylation in cells upon oxidative stress photo-generated by fullerols." *Biophysical chemistry*.
 - iii. Maaroufi, K., E. Save, et al. (2011). "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." *Neuroscience*.
 - iv. Nelson, S. K., S. K. Bose, et al. (1994). "The toxicity of high-dose superoxide dismutase suggests that superoxide can both initiate and terminate lipid peroxidation in the reperfused heart." *Free Radical Biology and Medicine* **16**(2): 195-200.
 - v. Alvarez, J. G. and B. T. Storey (1989). "Role of glutathione peroxidase in protecting mammalian spermatozoa from loss of motility caused by spontaneous lipid peroxidation." *Gamete research* **23**(1): 77-90.
 - vi. Devasagayam, T., K. Bloor, et al. (2003). "Methods for estimating lipid peroxidation: An analysis of merits and demerits." *Indian journal of biochemistry & biophysics* **40**(5): 300-308.
- c. Free-Radical Damage:
- i. Ozgur, E., G. Güler, et al. (2010). "Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants n-acetyl cysteine and epigallocatechin-gallate." *International journal of radiation biology*(00): 1-11.

- ii. Gutteridge, J. and X. C. Fu (1981). "Enhancement of bleomycin-iron free radical damage to DNA by antioxidants and their inhibition of lipid peroxidation." *FEBS letters* **123**(1): 71.
- d. mRNA:
 - i. Yan, J. G., M. Agresti, et al. (2009). "Qualitative Effect on mRNAs of Injury-Associated Proteins by Cell Phone Like Radiation in Rat Facial Nerves." *Electromagnetic Biology and Medicine* **28**(4): 383-390.
 - ii. Yan, J. G., M. Agresti, et al. (2008). "Upregulation of specific mRNA levels in rat brain after cell phone exposure." *Electromagnetic Biology and Medicine* **27**(2): 147-154.
 - iii. Simbürger, E., A. Stang, et al. (1997). "Expression of connexin43 mRNA in adult rodent brain." *Histochemistry and cell biology* **107**(2): 127-137.
 - iv. Chen, J., H. C. He, et al. (2010). "Effects of Pulsed Electromagnetic Fields on the mRNA Expression of RANK and CAII in Ovariectomized Rat Osteoclast-Like Cell." *Connective Tissue Research* **51**(1): 1-7.
- e. Epigenetic changes.... environmentally induced genetic change:
 - i. Migliore, L. and F. Copped (2009). "Genetics, environmental factors and the emerging role of epigenetics in neurodegenerative diseases." *Mutation Research/Fundamental and Molecular*

Mechanisms of Mutagenesis **667**(1-2): 82-97.

- ii. Currenti, S. (2009). "Understanding and Determining the Etiology of Autism." *Cellular and Molecular Neurobiology* **30**(2): 161-171.
- f. Micronuclei formation:
 - i. Tice, R. R., G. G. Hook, et al. (2002). "Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells." *Bioelectromagnetics*, **23**(2): 113-126.
 - ii. Lerchl, A. (2009). "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)." *Int Arch Occup Environ Health* **82**(2): 275-278.
 - iii. Vijayalaxmi and T. J. Prihoda (2009). "Genetic damage in mammalian somatic cells exposed to extremely low frequency electro-magnetic fields: a meta-analysis of data from 87 publications (1990-2007)." *Int J Radiat Biol* **85**(3): 196-213.
 - iv. Sannino, A., M. Sarti, et al. (2009). "Induction of adaptive response in human blood lymphocytes exposed to radiofrequency radiation." *Radiat Res* **171**(6): 735-742.
- g. DNA repair disruption:
 - i. Brusick, D., R. Albertini, et al. (1998). "Genotoxicity of radiofrequency radiation. DNA/Genetox Expert Panel." *Environ*

- Mol Mutagen* **32**(1): 1-16.
- ii. Belyaev, I. Y., E. Markova, et al. (2009). "Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes." *Bioelectromagnetics* **30**(2): 129-141.
 - iii. Sun, L. X., K. Yao, et al. (2006). "[Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro]." *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* **24**(8): 465-467.
 - h. Immune response suppression:
 - i. Lyle, D. B., P. Schechter, et al. (1983). "Suppression of T-lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields." *Bioelectromagnetics* **4**(3): 281-292.
 - ii. Elekes, E., G. Thuroczy, et al. (1996). "Effect on the immune system of mice exposed chronically to 50 Hz amplitude-modulated 2.45 GHz microwaves." *Bioelectromagnetics* **17**(3): 246-248.
 - iii. DABALA, D., D. SURCEL, et al. (2008). "Oxidative and Immune Response in Experimental Exposure to Electromagnetic Fields." *Electromagnetic field, health and environment: proceedings of EHE'07*: 105.
 - iv. Surcel, D., D. Dabala, et al. (2009). "Free Radicals, Lipid Peroxidation and Immune Response in Experimental Exposure to Electromagnetic Fields." *Epidemiology* **20**(6): S118.

Conclusions

33. To understand the seriousness of this Agent of PM RF/MW radiation in interaction with populations and individuals, we need to consider some basic facts in addition to the many relevant and reliable studies above. For example, where shortwave, AM, FM, TV and cell phone infrastructure frequencies are demonstrated to be harmful, as they consistently are shown to be at low intensities with long duration, then, all other factors being equal, MW radiation at 2.45 GHz will likely be more harmful yet, due to its higher absorption-per-exposure and water molecule resonance. Increasing the constancy and length of exposure toward the maximum of occupational and 24-7 durations will lower the threshold for effects in populations and individuals. Complex radiation microenvironments with pulse-modulated wave and multiple sources, such as are deployed in WI-FI-equipped schools, are more harmful than a single, isolated MW radiation exposure at the same power density and duration. There are only a few of the many studies of RF/MW radiation infrastructure such as base stations that fail to show their studied effect. However, even were the reverse true, i.e., if there existed greater number than those that do show adverse effects, it is the case that positive studies (those that show adverse effects) hold more weight than negative studies (those that show no effect).

34. The FCC-appointed guideline-setting Commission, ASTM-IEEE, in 1991 referred in its conclusions to RF/MW radiation, the Agent, as a ‘Hazard,’ specifically setting a ‘Hazard Threshold.’ It has been discovered that, even amongst the 120 studies chosen by the Committee to prove the validity of its Hazard Threshold, there were 15 studies that concluded adverse effects at levels *lower* than the Hazard Threshold, thus disproving its validity. Three of these studies actually showed adverse effects at less than 10 percent of the Hazard Threshold. Thus the guidelines have no credibility.

35. The large body of scientific literature moreover redundantly proves this Agent to be a hazard. The media-promulgated notion that the relevant scientific studies are inconsistent and inconclusive is false and misleading. Chronic exposure to PM MW radiation harms every individual in a population in some ways, even if these are not always detectable by the individual or consciously attributed to the responsible RF/MW radiation sources. This Agent injures some individuals into a condition in which symptoms will be more easily retriggered with subsequent exposure. And for *a priori* susceptible individuals and those using electronic medical devices, it can respectively exacerbate the extant medical conditions and disrupt medical device operation, even to the point of death. Bassen 1997 discusses the hundreds of excess deaths, even at that time, from wireless communications radiation. See also *Radiofrequency Interference with Medical Devices*, IEEE Engineering in Medicine and Biology Magazine 17(3):111-114(1998), <http://ewh.ieee.org/soc/embs/comar/interfer.htm>.

36. For these reasons, WI-FI must be banned from school deployment.

37. I will receive no compensation for my testimony beyond out-of-pocket expenses.

Dated this 20th day of December, 2011.



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CURRICULUM VITAE

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Schenectady, New York 12303

Positions Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Education: 1959 B.A., Harvard College, Cambridge, MA
1964 M.D., Harvard Medical School, Boston, MA

Positions Held:

- 9/61-6/62 Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
- 7/64-6/65 Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
- 7/65-2/73 Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
- 2/73-3/80 Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
- 3/80-9/85 Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 9/85-1/98 Dean, School of Public Health, University at Albany
- 9/85-Pres. Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
- 9/85-7/98 Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 1/98-1/05 Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
- 2001-Pres. Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
- 2005-Pres. Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York

Editor-in-Chief: Cellular and Molecular Neurobiology, 1981 - 1987

Editorial Advisor: Cellular and Molecular Neurobiology, 1987 - Present

Editorial Boards: Journal of Public Health Management and Practice, 1995 - 2002
International Journal of Occupational Medicine & Environmental Health
1996 – Present

Journal of Alzheimer's Disease– Associate Editor,2007-2009
Reviews in Environmental Health; 2008-present
International Archives of Occupational and Environmental Health; 2009-present.
Journal of Environmental and Public Health, 2009-present.
Environmental Health Perspectives, 2010-present

National and International Committees:

- 1978, 1981 Physiology Study Section (Ad hoc member)
- 1979-1985 NIH International Fellowship Study Section
- 1974-1981 Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
- 1981-1989 Member, USA National Committee for the International Brain Research Organization
- 1985-1986 Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
- 1986-1987 Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
- 1987-1989 Member, Science Advisory Council of the American Paralysis Association
- 1987-1990 Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
- 1985-1993 Committee #79, National Council on Radiation Protection and Measurements
- 1986-1997 Member, Legislative and Education Committees, Association of Schools of Public Health
- 1989-1994 Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
- 1994, 1995 Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
- 1994-1997 Member, Legislative Committee of the Association of Schools of Public Health
- 1997 Member, Executive Committee of the Association of Schools of Public Health
- 1997-2000 National Advisory Environmental Health Sciences Council of the National Institutes of Health
- 1998-Pres. Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
- 2000-Pres. Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
- 2001-2008 United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
- 2002-2003 Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
- 2003-2008 Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
- 2003-Pres. Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
- 2007-2011 Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.

State and Local Committees:

- 1980-1987 Executive Secretary, New York State Power Lines Project
- 1985-1989 Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
- 1986-1989 Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
- 1991-1992 Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
- 1991-1992 Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
- 1991-1992 Member, State Task Force for the Reform of Middle Level Education in NY State
- 1992-1993 Member, State Needs Task Force on Health Care and Education
- 1987-1998 Delegate-at-Large, New York State Public Health Association
- 1991-1995 Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
- 1994 Chair, Council of Deans, University at Albany, SUNY
- 1997-2008. Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
- 2000-Pres. Member, Board of Directors, Healthy Schools Network, Inc.
- 2000-2003 Member, Medical Advisory Board, Hepatitis C Coalition, New York
- 2000-2004 Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
- 2001-2008 Member, Board of Directors, Environmental Advocates of New York
- 2004-2007 Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
- 2005-Pres. Member, Schooling Chefs Curriculum Advisory Board
- 2005-2008 Member, Board of Directors, Citizens Environmental Coalition
- 2006-2009 Member, Board of Directors, Marine Environmental Research Institute
- 2007-2009 Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

- 1959 B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, Ambystoma tigrinum" (Thesis advisor, Professor George Wald)
Elected to Phi Beta Kappa and to Sigma Xi
- 1964 M.D. awarded cum laude for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
- 1964 Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
- 1970 Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
- 1971 Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months

- 1982, 1986 Visiting Professor of Physiology, Department of Physiology, Kyushu
 1987 University, Fukuoka, Japan, for a period of three months each
 1989 Awarded Jacob Javits Neuroscience Investigator Award from the National
 Institute of Neurological and Communicative Diseases and Stroke
 1999 Awarded Homer N. Calver Award from the American Public Health
 Association for studies in environmental health.
 2001 Awarded 2001 Academic Laureate from the University at Albany
 Foundation.
 2010 Awarded the Albion O. Bernstein, M.D. Award in recognition of an
 outstanding contribution to public health and the prevention of disease through
 lifelong research of environmental health hazards and for limitless devotion to
 medical education by the Medical Society of the State of New York.

Federal Grants Held: (Principal Investigator Only)

- 1980-1983 United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs",
 \$76,847 total direct costs.
 1982-1988 National Institute of Health, "Mechanisms of Desensitization at Central Synapses",
 \$464,786 total direct costs.
 1984-1986 Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs@,
 \$330,504 total direct costs.
 1986-1996 National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and
 Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct
 costs.
 1989-1993 National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total
 direct costs
 1990-1995 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O.
 Carpenter, P.I. \$5,783,419 total direct costs.
 1995-2001 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Central/Eastern European
 Environ/Occup Training Program@, D.O. Carpenter, P.I. \$657,520 total costs.
 1995-2001 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total
 direct costs.
 1998-1999 Environmental Protection Agency, A Indoor Air Risk at Akwesasne - Pilot Project@,
 D.O. Carpenter, P.I. \$9,996 total costs.
 2000-2002 Association Liaison Office for University Cooperation in Development,
 A Cooperative Program in Environmental Health between the Institute of Public
 Health at Makerere University, Kampala, Uganda and the School of Public Health,
 University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
 2001-2007 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Multidisciplinary
 Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
 2006-2011 Pakistan-US Science and Technology Cooperative Program (US National
 Academy of Sciences). "Association of particulate matter with daily morbidity in

- an urban population,” D.O. Carpenter, P.I., \$391,104 total costs.
- 2009-2013 Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2: Environmental contaminants and reproductive health of Akwesasne Mohawk women. \$387,825 for year 1. D.O. Carpenter, Co-PI.
- 2010-2013 Department of the Army, “Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, “Drinking Water Supply, Sanitation, and Hygiene Promotion : Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda”. D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1R01ES019620), “Protecting the health of future generations: Assessing and preventing exposures.” PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2,354,871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson’s Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001. Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.

6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibibility of motoneurons of different sizes. *J. Neurophysiol.*, 28:599-620, 1965.
7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. *J. Neurophysiol.*, 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. *J. Pharmacol.*, 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. *J. Neurophysiol.*, 28:958-965, 1965.
10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. *Arch. Ital. Biol.*, 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. *J. Neurophysiol.*, 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in *Aplysia* neurons. *J. Gen. Physiol.*, 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. *Adv. Pharmacol.*, 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na⁺ pump to membrane potential in *Aplysia* neurons. *J. Gen. Physiol.*, 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. *Arch. Neurol.*, 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na⁺ pump in *Aplysia* neurons. *Comp. Biochem. Physiol.*, 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of *Aplysia* neurons upon Na⁺ and Ca⁺⁺. *J. Cell. Physiol.*, 75:121-127, 1970.
18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepinephrine in the presence of tetrodotoxin. *J. Pharmacol. Exp. Therap.*, 73:416-421, 1970.
19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. *Science*, 169:597-598, 1970.
20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of *Aplysia* neurons and squid axon as determined by a new technique. *Intl. J. Neurosci.*, 2:35-48, 1971.
21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in *Aplysia* nervous and non-nervous tissues. *Int. J. Neurosci.*, 2:49-56, 1971.
22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of *Aplysia* neuron somata. *Science*, 176:1329-1331, 1972.
23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. *Science*, 179:1336-1338, 1973.
24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. *J. Physiol. (Lond.)*, 229:471-493, 1973.
25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. *Aplysia* acetylcholine receptors: Blockage by and binding of α -bungarotoxin. *Brain Res.*, 72:225-240, 1974.
26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. *Brain Res.*, 73:156-160, 1974.

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for the plaintiffs, 12 September 2006. Samuel E. Stubbs, Attorney; 713-425-7345.

Charles W. Adams, et al., vs. Cooper Industries, Inc. et al., deposed for the plaintiffs, 28-
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Attorney. 406-525-2665.

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Ronald Cybart et al., Michael Campanelli, and Donald and Theresa Shea, et al.v. CL&P. Deposed for the plaintiffs. 15 July 2011. Benson A. Snaider, Attorney.

Maria Snoops vs. Lyon Associates, Inc. and Insurance Co of the state of Pennsylvania. Deposed for the plaintiff, 1 November 2011. Matthew J. Witteman, Attorney. 415-363-3106.

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Local Counsel for Plaintiffs

United States District Court

District of Oregon

Portland Division

AHM, by and through
her Guardian *ad litem* and father,
David Mark Morrison, and
David Mark Morrison, individually,

v.

Portland Public Schools,

Defendant.

Civil Action No. 3:11-cv-00739-MO

**Amended Declaration of
Dr. David O. Carpenter, M.D.**

I, Dr. David O. Carpenter, M.D., under penalty of perjury pursuant to 28 U.S.C. § 1746, hereby make the following declaration in support of an injunction against Portland Public Schools' use of WI-FI:

1. I am a public health physician, educated at Harvard Medical School. My current title is Director of the Institute for Health and the Environment at the University at Albany and Professor of Environmental Health Sciences within the School of Public Health. Formerly, I was the Dean of the School of Public Health at the University of Albany and the Director of the Wadsworth Center for Laboratories and Research of the New York State Department of Health.

2. I served as the Executive Secretary to the New York State Powerlines Project in the 1980s, a program of research that showed children living in homes with elevated magnetic fields coming from powerlines suffered from an elevated risk of developing leukemia. After this I became the spokesperson on electromagnetic field (EMF) issues for the state during the time of my employment in the Department of Health. I have published several reviews on the subject and have edited two books.

3. I am a Co-Editor and a Contributing Author of the *BioInitiative: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)*, www.bioinitiative.org. It documents bioeffects, adverse health effects and public health conclusions about impacts of electromagnetic radiation (electromagnetic fields including extremely-low frequency ELF-EMF and radiofrequency /microwave or RF-EMF fields). The public health chapter from this report was subsequently published in a peer-reviewed journal.

4. Additionally, I am a Co-Author of *Setting Prudent Public Health Policy for Electromagnetic Field Exposures*, *Reviews on Environmental Health*, Volume 23, No 2, 2008, attached as Addendum A-2.

5. In addition, in 2009, I was invited to present to the President's Cancer Panel on the subject of powerline and radiofrequency fields and cancer, and have testified on this issue before the United States House of Representatives.

6. In sum, I am a public health physician, professor and former public health school Dean with expertise in electrophysiology, low-frequency electromagnetic fields bioeffects, and

radiofrequency (RF) and microwave (MW) radiation bioeffects.

7. WI-FI deploys pulse-modulated (“PM”) microwave (“MW”) radiation (within the larger RF radiation spectrum) with a carrier frequency that is similar to that used by a microwave oven: about 2.45 GHz. This is the “Agent”. The 2.45 GHz frequency was chosen for the oven because of its wavelength and harmonic resonance with the water molecule, to ensure the most efficient absorption by living tissues and effective heating by way of the agitation of water at the molecular level. The pulse-modulation of a wave with lower frequencies in addition to the high-frequency carrier signal, increases the exposure complexity and in turn the bioeffects in an exposed population.

8. In the context of school development, WI-FI exposes building occupants including children and adults constantly from both computers and infrastructure antennas. Duration may be an even more potent contributing factor to RF/MW radiation bioeffects than exposure levels. Chronic, such as all-day, school exposure, is more likely than short and intermittent exposure, such as cell phone use, to produce harmful health effects, and is likely to do so at lower exposure levels.

9. Persons stationed close to school computers with WI-FI and especially those very near to any WI-FI infrastructure will receive considerably higher exposure than do others.

10. It is generally accepted within the relevant scientific community and has been established beyond any reasonable doubt that adverse human health effects occur at far lower levels of RF/MW radiation exposure than those that cause noticeable heating, particularly where the wavelength approaches body-part size and thus maximizes absorption, where the wavelength has resonance with the water molecule, where there is more complex, modulated wave, where there is chronic exposure duration, and where exposed persons lack the capacity voluntarily to remove themselves from radiation sources.

11. Some effects are shown to occur at several hundred thousand times below the FCC public exposure guidelines, which are set based on the fallacious assumption that there are no adverse health effects at exposures that do not cause easily measureable heating. FCC guidelines

also only apply to 30-minute public exposures; therefore do not even infer safety at durations >30 minutes, such as in a school setting.

12. Exposure to high-frequency RF and MW radiation and also the extreme low frequency (ELF) EM fields that accompany WI-FI exposure have been linked to a variety of adverse health outcomes. Some of the many adverse effects reported to be associated with and/or caused by ELF fields and/or RF/MW radiation include neurologic, endocrine, immune, cardiac, reproductive and other effects, including cancers.

13. Studies of isolated cells have shown that RF/MW exposures may cause changes in cell membrane function, cell communication, metabolism, activation of proto-oncogenes, and can trigger the production of stress proteins at exposure levels below FCC guidelines and also at and less than school WI-FI exposure levels and parameters. Resulting effects in cellular studies include without limitation DNA breaks and chromosome aberrations, cell death including death of brain neurons, increased free radical production, activation of the endogenous opioid system, cell stress and premature aging.

14. Human studies of comparable RF/MW radiation parameters show changes in brain function including memory loss, retarded learning, performance impairment in children, headaches and neurodegenerative conditions, melatonin suppression and sleep disorders, fatigue, hormonal imbalances, immune dysregulation such as allergic and inflammatory responses, cardiac and blood pressure problems, genotoxic effects like miscarriage, cancers such as childhood leukemia, childhood and adult brain tumors, and more.

15. There is consistent evidence for increased incidence of effects in individuals who live near to high-power short-wave, AM, FM and TV transmission towers. This is particularly relevant because, like WI-FI, radio-TV transmission towers give continuous, whole-body radiation, not just radiation to the head, constantly.

16. Since WI-FI transmitters, both infrastructural and on computers, are indoors, where children and teachers may be very close by, and since WI-FI, at 2.45 GHz, deploys a

wavelength, at ~12.2 cm or ~ 4.8 inches, more absorbable by children's and adults' bodies and brains than radio-TV wavelengths, the harmfulness of WI-FI radiation likely exceeds that of radio-TV towers.

17. Like second-hand smoke, EMF and RF/MW radiation involve complex mixtures, where different frequencies, intensities, durations of exposure(s), modulation, waveform and other factors are known to produce variable effects, often more harmful with greater complexity. Decades of scientific study have produced substantial evidence that EMF and RF/MW radiation may be considered neurotoxic, carcinogenic and genotoxic. Sources of fields and radiation, but are not limited to: power lines, navigational radar, cell phones, cordless phones [or Digitally Encoded Cordless Transmission Devices (D.E.C.T.) phones], cell towers, 'smart' meters and their grids or infrastructure, "smart" boards, meters and grids, WiMax and wireless internet (WI-FI).

18. The RF/MW radiation and low-frequency EMF science that currently exists includes tens of thousands of studies dating back to the 1920s. On the basis of this vast body of literature, many public health experts believe, myself included, that it is likely society will face epidemics of neurotoxic effects and degeneration, cancers and genotoxicity in the future, resulting from the extreme and mostly involuntary exposure to RF/MW radiation and EMFs. WI-FI radiation in schools exceeds natural background levels of microwave radiation by trillions of times. Thus, it is important that all of us restrict our use of cell phones, and be as free as possible from exposure to unnatural, background sources of MW radiation, particularly WI-FI.

19. In public health science, it is generally accepted fact that vulnerable subgroups exist within any human population. This is also recognized specifically for RF/MW radiation and fields. These groups include children, pregnant women, the elderly and those with preexisting illnesses and/or impairments. Children are more vulnerable to RF/MW radiation because of the susceptibility of their developing nervous systems. RF/MW penetration is greater relative to head size in children, who have a greater absorption of RF/MW energy in the tissues of the head at WI-FI frequencies.

Such greater absorption results because children's skulls are thinner, their brains smaller, and their brain tissue is more conductive than those of adults, and since it has a higher water content and ion concentrations. The Presidential Cancer Panel found that children 'are at special risk due to their smaller body mass and rapid physical development, both of which magnify their vulnerability to known carcinogens, including radiation.'

http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf

20. FCC public RF/MW radiation exposure guidelines are based on the height, weight and stature of a 6-foot tall man, not children or adults of smaller stature. The guidelines do not take into account the unique susceptibility of growing children to exposures. Since children are growing, their rate of cellular activity and division is more rapid, and they are at more risk for DNA damage and subsequent cancers. Growth and development of the central nervous system is still occurring well into the teenage years, such that the neurological impairments predictable by the extant science may have great impact upon development, cognition, learning, and behavior. Prenatal exposure has been identified as a risk factor for childhood leukemia, and is associated with miscarriage. Children are largely unable to remove themselves from exposures to harmful substances in their environments. Their exposure is involuntary.

21. When WI-FI is in operation in a school, children and their parents have no choice but to allow the school to expose them to trillions of times higher microwave radiation than exists naturally on Earth at the same frequencies. Children and other building users are exposed to as much as 30-40 hours per week of constant, digitally encoded WI-FI signals from each wireless device and infrastructural antenna in a school building. Based upon a review of the Mount Tabor WI-FI Floor Plan, a given child is subject to direct signals from multiple WI-FI transmitters, including rooms full of students and teachers transmitting numerous laptop and other wireless signals. There is a major legal difference between an exposure that an individual chooses to accept and one that is forced upon a person, especially a dependent, who can do nothing about it.

22. WI-FI in the Portland Schools deploys similar PM MW radiation, at 2.45 and 5 GHz, to that of cell and cordless phones and their infrastructure. There is clear and strong evidence that intensive use of cell phones increases incidence of brain cancer, tumors of the auditory nerve, and cancer of the parotid gland, the salivary gland in the cheek by the ear. Cell and cordless phone radiation closely resembles that of WI-FI radiation exposure, except that WI-FI is more hazardous by way of frequency, duration, and the involuntary nature of exposure. While a cell or cordless phone is used only intermittently and primarily voluntarily, a WI-FI radiation microenvironment is constant in duration, with unavoidable radiation exposure even when nearby students are not actively using it. Because WI-FI radiation is essentially the same as, but more hazardous than, that for cell and cordless phones, there is every reason to understand that the health effects will be the same or worse, varying in relation to the total dose of radiation, and intensified by the constancy of duration. There is evidence from Scandinavian studies of cell phone usage that children who use cell phones are about five times more likely to develop brain cancer than if their usage starts as an adult. Thus, it is especially necessary to protect children from pulse-modulated MW radiation such as both cell phones and WI-FI deploy.

23. Based on a high degree of scientific certainty, Portland Public Schools' use of WI-FI is causing and will continue to cause AHM, other students, and school staff and faculty adverse health effects, and should be discontinued immediately. Educating by way of the Internet via cabled systems only decreases MW radiation exposure and is of minimal expense.

24. Having reviewed hundreds, possibly thousands, of studies in RF/MW radiation and ELF fields, published from decades ago to the present, I would provide you the following primary evidence, without limitation. Due to the active suppression of the RF/MW literature, some researchers in public health science are less aware of these studies. However, the forefront experts specializing in these areas, RF/MW radiation and ELF fields, recognize the certainties in this large body of scientific literature, which establishes without limitation that PM MW radiation with chronic duration is quite harmful to humans, particularly children, as well as to animals and plants.

25. It is not surprising that even as of 1990, the US Environmental Protection Agency ("EPA") had determined RF/MW radiation a "probable carcinogen". Now that we have much more confirming study in the interim, the conclusion is yet more certain. And when we focus on MW radiation, particularly pulse-modulated radiation, on long, non-intermittent duration and on more vulnerable subgroups such as children, we see that the cancer outcome is very certain, indeed. Amongst the epidemiologic studies showing cancer outcomes, the following are particularly strong:

- a. Dode AC, Leao M, Tejo FdeAF, gomes ACR, Dode DC, Dode MC, Moreira CW, Condessa VA, Albinatti C and Calaffa WT. Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais State, Brazil. *Sci Total Environ* 409: 3649-3665:2011. This study shows higher rates of cancer in people living close to cell phone towers than for people living further away. Cell phone radiation is similar to but likely not as harmful as 2.45 GHz radiation from WI-FI. The exposure levels in this study are lower than those that Portland school building occupants receive from WI-FI.
- b. Oberfeld G. Environmental Epidemiology Study of Cancer Incidence in the Municipalities of Hausmannstatten & Vasoldsberg (Austria), 2008. This government-commissioned study found significantly increased cancer risk relative to a lower-exposure reference category, 23x higher for breast cancer and 121x higher for brain tumors, with strong exposure-effect relations.
- c. Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, Barca A and Perucci CA. Adult and childhood leukemia near a high-power radiostation in Rome, Italy. *Am J Epidemiol.* 155: 1098-1103: 2002. The authors show that there is a significant elevation of childhood leukemia among residents living near to Vatican Radio, and that the risk declines with distance away from the transmitter. This is RF radiation in frequencies similar to that of WI-FI.

- d. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM and Pack JK. Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am J Epidemiol* 166: 270-279: 2007. Leukemia and brain cancer in children in Korea were investigated in relation to residence within 2 km of AM radio transmitters. There was a significant elevation in rates of leukemia but not of brain cancer. WI-FI radiation is more harmful than AM.
- e. Park SK, Ha M, Im HJ. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. *Int Arch Occup Environ Health*. 2004 Aug;77(6):387-94. This study found higher mortality areas for all cancers and leukemia in some age groups in the area near the AM towers.
- f. Hallberg O. Johansson O. *Med Sci Monit* 2004 Jul;10(7):CR336-40. Malignant melanoma of the skin – not a sunshine story! Increased incidence and mortality from skin melanoma are concluded to result from continuous disturbances of cell repair mechanisms by body-resonant EMFs from FM/TV networks.
- g. Hallberg O. Johansson O. 2005. FM Broadcasting exposure time and malignant melanoma incidence, *Electromagnetic Biology and Medicine* 24;1-8. Age-specific incidence of malignant melanoma of the skin is related to FM broadcasting radiation at whole-body resonant frequencies. This is very relevant to children, since the smaller wavelengths of WI-FI are at resonant frequencies with dimensions of the human head, particularly the child's head.
- h. Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I, Elliot P. Cancer Incidence near radio and television transmitters in Great Britain. I – Sutton-Colfield transmitter, and II. Al high-power transmitters. *Am J Epidemiol* 1997; 145(1):1-9 and 10-17. In the first study, there was a statistically significant

increase in cancer; in the second, a small but significant increase in adult leukemia.

i. Hocking B, Gordon IR, Grain HL, Harfield GE. Cancer incidence and mortality and proximity to TV towers. *Medical J of Australia*. 1985;165:601-605. At extremely low exposure levels, there was an association between increased childhood leukemia incidence and mortality and proximity to TV towers. TV radiation, in the VHF and UHF bands, is similar to but not as harmful as WI-FI radiation at 2.45 GHz.

j. Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: A nested case-control study. *Am J Epidemiol* 1996; 143:480-6. This study found an association between exposure to ELF and RF/MW radiation and brain tumors.

k. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Sci Total Environ* 1996;180:9-17. This study showed huge increases in leukemia and Non-Hodgkin's lymphomas. Though exposure levels are higher in this study than they would be with school WI-FI, it is possible that certain students or teachers stationed immediately next to the WI-FI infrastructure could receive comparable levels in radiation peaks.

26. Additional studies show neurologic, immune, endocrine, reproductive and cardiac, adverse health effects from low-dose, chronic exposure to RF/MW radiation in humans:

a. Papageorgiou CC, Hountala CD, Maganioti AE, Kyprianou MA, Rabavilas AD, Papadimitriou GN, Capsalis CN. Effects of WI-FI signals on the p300 component of event-related potentials during an auditory listening task. *J Integr Neurosci* 2011 Jun;10(2):189-202. This study concludes that WI-FI exposure may exert gender-related alterations on neural activity.

- b. Altpeter ES, Roosli M et al. Effect of Short-wave magnetic fields on sleep quality and melatonin cycle in humans: The Schwarzenburg shut-down study. *Bioelectromagnetics* 27:142-150, 2006. Sleep quality improved and melatonin excretion increased when the transmitter was shut down.
- c. Abelin T et al. Sleep disturbances in the vicinity of the short-wave broadcast transmitter Schwarzenburg. *Somnologie* 9:203-209, 2005. There is strong evidence of a causal relationship between operation of a short-wave radio transmitter and sleep disturbances in the surrounding population.
- d. Hutter HP et al. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup Environ Med* 2006;63:307-313, 2006. There was a significant relation of some symptoms, especially headaches, to measured power density, as well as effects on wellbeing and performance.
- e. Preece AW, Georgious AG, Duunn EJ, Farrow SC. *Occup Environ Med* 2007 Jun;64(6):402-8. Compared to control village, there were highly significant differences in the reporting of migraine, headache and dizziness military and cell phone antenna systems.
- f. Buchner K, Eger, H. Changes of clinically important neurotransmitters under the influence of modulated RF fields – a long-term study under real-life conditions. *Umwelt-Medizin-Gesellschaft* 24(1):44-57, 2011. There is clear evidence of health-relevant effects, including increase in adrenaline/noradrenaline, subsequent decrease in dopamine from a new MW-emitting base station. During counterregulation, trace amine PEA decreased and remained decreased. Clinically documented increases in sleep problems, cephalgia, vertigo, concentration problems and allergies followed the onset of new microwave transmissions.

- g. Eliyahu I, Luria R, Hareuveny R, Margalioth M, Neiran N and Shani G . Effects of radiofrequency radiation emitted by cellular telephones on the cognitive functions of humans. *Bioelectromagnetics* 27: 119-126: 2006. A total of 36 human subjects were exposed to PM MW and were tested on four distinct cognitive tasks. Exposure to the left side of the brain slows left-hand response time in three of the four tasks.
- h. Barth A, Winker R, Ponocny-Seliger E, Mayrhofer W, Ponocny I, Sauter C and Vana N. *Occup Environ Med* 65: 342-345: 2008. A meta-analysis for neurobehavioural effects due to electromagnetic field exposure emitted by GSM mobile phones. The authors looked at 19 studies of cognitive function in cell phone users, and found in the meta-analysis that there is evidence for a decreased reaction time, altered working memory and increased number of errors in exposed persons.
- i. Augner C, Hacker GW, Oberfeld G, Florian M, Hitzl W, Hutter J and Pauser G. Effects of exposure to base station signals on salivary cortisol, alpha-amylase and immunoglobulin A. *Biomed Environ Sci* 23: 199-207: 2010. This was a human experimental study with exposure to PM MW radiation wherein immune indicators were monitored after five 50-minute sessions. The researchers found dose-dependent changes in cortisol and alpha-amylase.
- j. Avendano C, Mata A, Sanchez Sarimiento CA and Doncel GF. Use of laptop computers connected to internet through WI-FI decreases human sperm motility and increases sperm DNA fragmentation. *Fert Steril*, 2012, In press. In this study human sperm were exposed to WI-FI from a laptop, and were found to show reduced motility after a 4-hour exposure. The results are consistent with other publications (see Agarwal et al., *Fert Steril* 89: 124-128: 2008) that reported that those who use cell phone regularly have reduced sperm count.

k. Baste V, Riise T and Moen BE (2008) *Int J Epidemiol* 23: 369-377: 2008. Radiofrequency electromagnetic fields: male infertility and sex ratio of offspring. This is a study of Norwegian Navy personnel chronically exposed to RF fields on the job. The rates of infertility were related to level of exposure in a dose-dependent fashion.

27. Many toxicologic and other animal studies, of which the following are but a few, support conclusions of cancer, genotoxicity, neurotoxicity and other health outcomes from RF/MW radiation.

a. Sinha R. Chronic non-thermal exposure of modulated 2450 MHz microwave radiation alters thyroid hormones and behavior of male rats. *Int. J. Radiation Biol.* 84:6:505-513, 2008. This study of 2.45 GHz at levels and durations comparable to and less than those of school WI-FI concluded that the radiation was sufficient to alter the levels of thyroid hormone as well as emotional reactivity compared to controls.

b. Nittby H, Grafstrom G, Tian DP, Malmgren L, Brun A, Persson BRR, Salfors LG and Eberhardt J. *Bioelectromagnetics* 29: 219-232: 2008. This study showed cognitive impairment in rats after long-term exposure to PM MW radiation. This study of rats shows that after 2 hours per week for 55 weeks there was impaired memory for objects in exposed as compared to sham animals.

c. Kimmel S et al. Electromagnetic radiation: Influences on honeybees (*Apis mellifera*). A significant difference between non-exposed and fully irradiated bees was the result of the influence of high-frequency PM RF/MW radiation.

d. Panagopoulos DJ et al. Bioeffects of mobile telephony radiation in relation to its intensity or distance from the antenna. *Int. J Radiat Biol*, 86;(5):345-357, 2010. The PM MW radiations at 900 and 1800 MHz decreased the reproductive capacity by cell death induction, with an increased bioactivity “window” at 10

uW/cm², and still evident down to 1 uW/cm².

e. Everaert J, Bauwens D. A possible effect of electromagnetic radiation from mobile phone base stations on the number of breeding house sparrow (*passer domesticus*). *Electromagnetic Biology and Medicine*, 26:63-72, 2007.

Long-term exposure to higher-level low-intensity PM MW radiation negatively affects the abundance or behavior of House Sparrows in the wild.

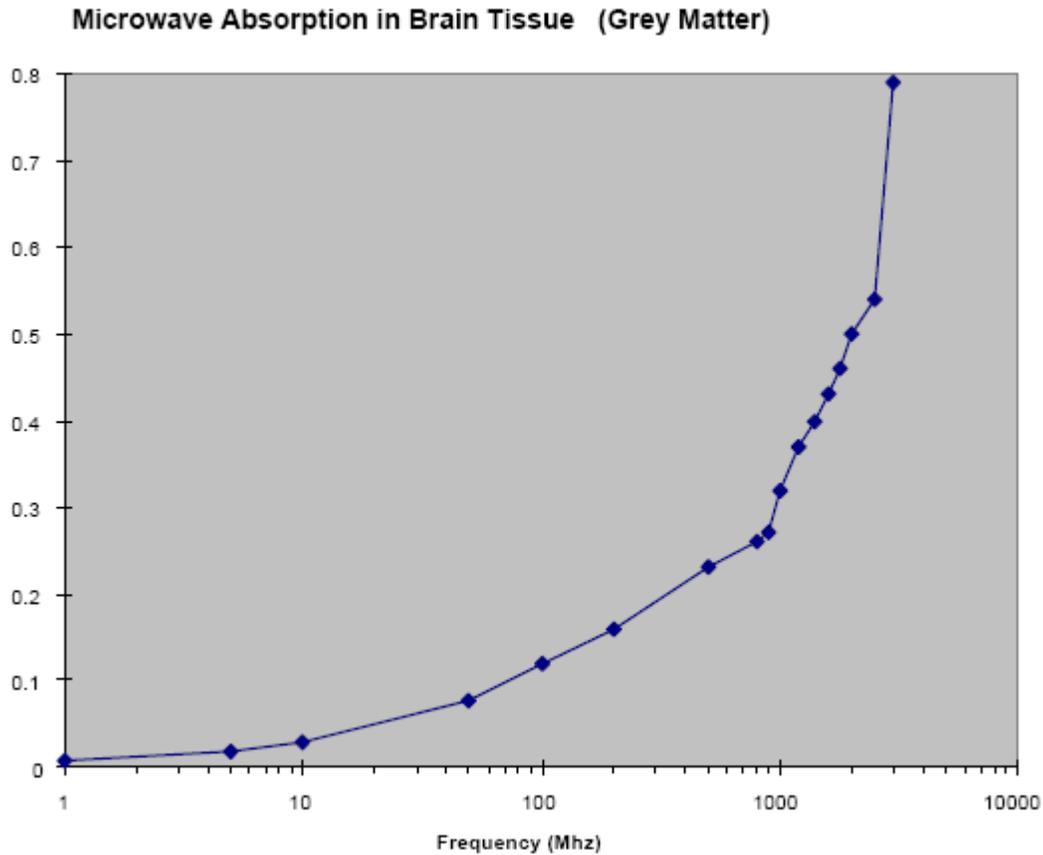
f. Magras I, Xenos T. RF Radiation-Induced Changes in the Prenatal Development of Mice. *Bioelectromagnetics* 18:455-461, 1997. Near almost 100 TV and FM broadcast transmitters, with exposure levels between 0.168 uW/cm² and 1.053 uW/cm², found in the more exposed groups testicular damage and decreasing size of litters to irreversible infertility.

g. Balmori A. Electromagnetic pollution from phone masts. *Effects on wildlife, Pathophysiology* 2009. This large review of wildlife effects concludes, “pulsed telephony microwave radiation can produce effects on nervous, cardiovascular, immune and reproductive systems,” including damage to the nervous system by altering EEG and changes to the blood-brain barrier, disruption of the 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, growth problems, problems in building the nest or impaired fertility, embryonic development, hatching percentage, genetic and developmental problems, problems of locomotion, promotion of tumors and more.

28. Exposure thresholds for harmful effects are lowered in human populations and individuals when duration is increased. Due to the variability of thresholds for harmful effects both in the population and within the individual, there is no exposure power density that is safe. The School's WI-FI deploys arguably the worst possible frequency of 2.45 GHz, that of the

microwave oven, worst because it is most absorbable by the brain and most resonant with the water molecule, such that:

- a. absorption-per-exposure is maximized, dramatically lowering effects thresholds for population and individual effects; and
- b. water molecules in tissues and cells are highly agitated.



Curry, Ph.D., *Wireless LANs in the schoolroom*

29. This above graph, from physicist William Curry PhD's presentation *Wireless LANs in the Schoolroom*, shows how absorption in brain tissue (grey matter) increases exponentially toward the ultra-high frequency (UHF) area of the microwave oven and WI-FI.

30. In the case of the Portland Schools, the additional, unused but still deployed carrier frequency of 5 GHz would likely increase absorption in other, smaller organs, such as the thyroid.

31. The graph also illustrates the problem with the drive of the wireless industry toward ever higher frequencies within the cm microwave band. While nearly all the lower frequency bands have already been allocated by the FCC for specific types of radio transmissions, and transmission of ever more information content on any given channel requires greater bandwidth, each new deployment undermines further the integrity of the population's health. Engineers who design these systems have no training that would qualify them to consider the effects on biologic systems, which is why public health scientists need to be called in to policymaking *prior to* contracting and deployment, not after the fact.

32. The following studies explain the mechanisms of interaction between RF/MW radiation and biologic systems at the cellular level.

- a. The cell membrane recognition process -- which includes signal transduction and 'heat-shock protein' release -- was first discerned by Litovitz and his co-workers at Catholic University of America in the mid-1990s.

Below are a few citations that make the point.

- i. Litovitz, T., C. Montrose, et al. (1994). "Superimposing spatially coherent electromagnetic noise inhibits field induced abnormalities in developing chick embryos." *Bioelectromagnetics* **15**(2): 105-113.
- ii. DiCarlo, A., J. Farrell, et al. (1998). "A simple experiment to study electromagnetic field effects: Protection induced by short term exposures to 60 Hz magnetic fields." *Bioelectromagnetics* **19**(8): 498-500.
- iii. Penafiel, L., T. Litovitz, et al. (1997). "Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929

- cells." *Bioelectromagnetics* **18**(2): 132-141.
- iv. Dicarlo, A. L., Michael T. Hargis, L. Miguel Penafiel, Theodore A. Litovitz, A. (1999). "Short-term magnetic field exposures (60Hz) induce protection against ultraviolet radiation damage." *International journal of radiation biology* **75**(12): 1541-1549.
 - v. Litovitz, T., C. Montrose, et al. (1990). "Amplitude windows and transiently augmented transcription from exposure to electromagnetic fields." *Bioelectromagnetics* **11**(4): 297-312.
 - vi. Litovitz, T., M. Penafiel, et al. (1997). "The role of temporal sensing in bioelectromagnetic effects." *Bioelectromagnetics* **18**(5): 388-395.
 - vii. Litovitz, T., L. Penafiel, et al. (1997). "Role of modulation in the effect of microwaves on ornithine decarboxylase activity in L929 cells." *Bioelectromagnetics* **18**: 132-141.]
 - viii. Litovitz, T., D. Krause, et al. (1993). "The role of coherence time in the effect of microwaves on ornithine decarboxylase activity." *Bioelectromagnetics* **14**(5): 395-403.
- b. Cell membrane reaction is lipid peroxidation.
 - i. Serban, M. and V. Ni (1994). "Lipid peroxidation and change of plasma lipids in acute ischemic stroke." *Romanian journal of internal medicine= Revue roumaine de médecine interne* **32**(1): 51.

- ii. Vileno, B., S. Jeney, et al. (2010). "Evidence of lipid peroxidation and protein phosphorylation in cells upon oxidative stress photo-generated by fullerols." *Biophysical chemistry*.
- iii. Maaroufi, K., E. Save, et al. (2011). "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." *Neuroscience*.
- iv. Nelson, S. K., S. K. Bose, et al. (1994). "The toxicity of high-dose superoxide dismutase suggests that superoxide can both initiate and terminate lipid peroxidation in the reperfused heart." *Free Radical Biology and Medicine* **16**(2): 195-200.
- v. Alvarez, J. G. and B. T. Storey (1989). "Role of glutathione peroxidase in protecting mammalian spermatozoa from loss of motility caused by spontaneous lipid peroxidation." *Gamete research* **23**(1): 77-90.
- vi. Devasagayam, T., K. Bloor, et al. (2003). "Methods for estimating lipid peroxidation: An analysis of merits and demerits." *Indian journal of biochemistry & biophysics* **40**(5): 300-308.
- c. Free-Radical Damage:
 - i. Ozgur, E., G. Güler, et al. (2010). "Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants n-acetyl cysteine and epigallocatechin-gallate." *International journal of radiation biology*(00): 1-11.

- ii. Gutteridge, J. and X. C. Fu (1981). "Enhancement of bleomycin-iron free radical damage to DNA by antioxidants and their inhibition of lipid peroxidation." *FEBS letters* **123**(1): 71.
- d. mRNA:
 - i. Yan, J. G., M. Agresti, et al. (2009). "Qualitative Effect on mRNAs of Injury-Associated Proteins by Cell Phone Like Radiation in Rat Facial Nerves." *Electromagnetic Biology and Medicine* **28**(4): 383-390.
 - ii. Yan, J. G., M. Agresti, et al. (2008). "Upregulation of specific mRNA levels in rat brain after cell phone exposure." *Electromagnetic Biology and Medicine* **27**(2): 147-154.
 - iii. Simbürger, E., A. Stang, et al. (1997). "Expression of connexin43 mRNA in adult rodent brain." *Histochemistry and cell biology* **107**(2): 127-137.
 - iv. Chen, J., H. C. He, et al. (2010). "Effects of Pulsed Electromagnetic Fields on the mRNA Expression of RANK and CAII in Ovariectomized Rat Osteoclast-Like Cell." *Connective Tissue Research* **51**(1): 1-7.
- e. Epigenetic changes.... environmentally induced genetic change:
 - i. Migliore, L. and F. Copped (2009). "Genetics, environmental factors and the emerging role of epigenetics in neurodegenerative diseases." *Mutation Research/Fundamental and Molecular*

Mechanisms of Mutagenesis **667**(1-2): 82-97.

- ii. Currenti, S. (2009). "Understanding and Determining the Etiology of Autism." *Cellular and Molecular Neurobiology* **30**(2): 161-171.
- f. Micronuclei formation:
 - i. Tice, R. R., G. G. Hook, et al. (2002). "Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells." *Bioelectromagnetics*, **23**(2): 113-126.
 - ii. Lerchl, A. (2009). "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)." *Int Arch Occup Environ Health* **82**(2): 275-278.
 - iii. Vijayalaxmi and T. J. Prihoda (2009). "Genetic damage in mammalian somatic cells exposed to extremely low frequency electro-magnetic fields: a meta-analysis of data from 87 publications (1990-2007)." *Int J Radiat Biol* **85**(3): 196-213.
 - iv. Sannino, A., M. Sarti, et al. (2009). "Induction of adaptive response in human blood lymphocytes exposed to radiofrequency radiation." *Radiat Res* **171**(6): 735-742.
- g. DNA repair disruption:
 - i. Brusick, D., R. Albertini, et al. (1998). "Genotoxicity of radiofrequency radiation. DNA/Genetox Expert Panel." *Environ*

- Mol Mutagen* **32**(1): 1-16.
- ii. Belyaev, I. Y., E. Markova, et al. (2009). "Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes." *Bioelectromagnetics* **30**(2): 129-141.
 - iii. Sun, L. X., K. Yao, et al. (2006). "[Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro]." *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* **24**(8): 465-467.
 - h. Immune response suppression:
 - i. Lyle, D. B., P. Schechter, et al. (1983). "Suppression of T-lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields." *Bioelectromagnetics* **4**(3): 281-292.
 - ii. Elekes, E., G. Thuroczy, et al. (1996). "Effect on the immune system of mice exposed chronically to 50 Hz amplitude-modulated 2.45 GHz microwaves." *Bioelectromagnetics* **17**(3): 246-248.
 - iii. DABALA, D., D. SURCEL, et al. (2008). "Oxidative and Immune Response in Experimental Exposure to Electromagnetic Fields." *Electromagnetic field, health and environment: proceedings of EHE'07*: 105.
 - iv. Surcel, D., D. Dabala, et al. (2009). "Free Radicals, Lipid Peroxidation and Immune Response in Experimental Exposure to Electromagnetic Fields." *Epidemiology* **20**(6): S118.

Conclusions

33. To understand the seriousness of this Agent of PM RF/MW radiation in interaction with populations and individuals, we need to consider some basic facts in addition to the many relevant and reliable studies above. For example, where shortwave, AM, FM, TV and cell phone infrastructure frequencies are demonstrated to be harmful, as they consistently are shown to be at low intensities with long duration, then, all other factors being equal, MW radiation at 2.45 GHz will likely be more harmful yet, due to its higher absorption-per-exposure and water molecule resonance. Increasing the constancy and length of exposure toward the maximum of occupational and 24-7 durations will lower the threshold for effects in populations and individuals. Complex radiation microenvironments with pulse-modulated wave and multiple sources, such as are deployed in WI-FI-equipped schools, are more harmful than a single, isolated MW radiation exposure at the same power density and duration. There are only a few of the many studies of RF/MW radiation infrastructure such as base stations that fail to show their studied effect. However, even were the reverse true, i.e., if there existed greater number than those that do show adverse effects, it is the case that positive studies (those that show adverse effects) hold more weight than negative studies (those that show no effect).

34. The FCC-appointed guideline-setting Commission, ASTM-IEEE, in 1991 referred in its conclusions to RF/MW radiation, the Agent, as a ‘Hazard,’ specifically setting a ‘Hazard Threshold.’ It has been discovered that, even amongst the 120 studies chosen by the Committee to prove the validity of its Hazard Threshold, there were 15 studies that concluded adverse effects at levels *lower* than the Hazard Threshold, thus disproving its validity. Three of these studies actually showed adverse effects at less than 10 percent of the Hazard Threshold. Thus the guidelines have no credibility.

35. The large body of scientific literature moreover redundantly proves this Agent to be a hazard. The media-promulgated notion that the relevant scientific studies are inconsistent and inconclusive is false and misleading. Chronic exposure to PM MW radiation harms every individual in a population in some ways, even if these are not always detectable by the individual or consciously attributed to the responsible RF/MW radiation sources. This Agent injures some individuals into a condition in which symptoms will be more easily retriggered with subsequent exposure. And for *a priori* susceptible individuals and those using electronic medical devices, it can respectively exacerbate the extant medical conditions and disrupt medical device operation, even to the point of death. Bassen 1997 discusses the hundreds of excess deaths, even at that time, from wireless communications radiation. See also *Radiofrequency Interference with Medical Devices*, IEEE Engineering in Medicine and Biology Magazine 17(3):111-114(1998), <http://ewh.ieee.org/soc/embs/comar/interfer.htm>.

36. For these reasons, WI-FI must be banned from school deployment.

37. I will receive no compensation for my testimony beyond out-of-pocket expenses.

Dated this 20th day of December, 2011.



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Director, Institute for Health and the Environment
University at Albany

CURRICULUM VITAE

Name: David O. Carpenter

Home Address: 2749 Old State Road
Schenectady, New York 12303

Positions Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Education: 1959 B.A., Harvard College, Cambridge, MA
1964 M.D., Harvard Medical School, Boston, MA

Positions Held:

- 9/61-6/62 Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
- 7/64-6/65 Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
- 7/65-2/73 Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
- 2/73-3/80 Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
- 3/80-9/85 Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 9/85-1/98 Dean, School of Public Health, University at Albany
- 9/85-Pres. Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
- 9/85-7/98 Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 1/98-1/05 Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
- 2001-Pres. Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
- 2005-Pres. Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York

Editor-in-Chief: Cellular and Molecular Neurobiology, 1981 - 1987

Editorial Advisor: Cellular and Molecular Neurobiology, 1987 - Present

Editorial Boards: Journal of Public Health Management and Practice, 1995 - 2002
International Journal of Occupational Medicine & Environmental Health
1996 – Present

Journal of Alzheimer's Disease– Associate Editor,2007-2009
Reviews in Environmental Health; 2008-present
International Archives of Occupational and Environmental Health; 2009-present.
Journal of Environmental and Public Health, 2009-present.
Environmental Health Perspectives, 2010-present

National and International Committees:

- 1978, 1981 Physiology Study Section (Ad hoc member)
- 1979-1985 NIH International Fellowship Study Section
- 1974-1981 Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
- 1981-1989 Member, USA National Committee for the International Brain Research Organization
- 1985-1986 Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
- 1986-1987 Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
- 1987-1989 Member, Science Advisory Council of the American Paralysis Association
- 1987-1990 Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
- 1985-1993 Committee #79, National Council on Radiation Protection and Measurements
- 1986-1997 Member, Legislative and Education Committees, Association of Schools of Public Health
- 1989-1994 Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
- 1994, 1995 Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
- 1994-1997 Member, Legislative Committee of the Association of Schools of Public Health
- 1997 Member, Executive Committee of the Association of Schools of Public Health
- 1997-2000 National Advisory Environmental Health Sciences Council of the National Institutes of Health
- 1998-Pres. Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
- 2000-Pres. Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
- 2001-2008 United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
- 2002-2003 Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
- 2003-2008 Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
- 2003-Pres. Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
- 2007-2011 Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.

State and Local Committees:

- 1980-1987 Executive Secretary, New York State Power Lines Project
- 1985-1989 Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
- 1986-1989 Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
- 1991-1992 Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
- 1991-1992 Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
- 1991-1992 Member, State Task Force for the Reform of Middle Level Education in NY State
- 1992-1993 Member, State Needs Task Force on Health Care and Education
- 1987-1998 Delegate-at-Large, New York State Public Health Association
- 1991-1995 Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
- 1994 Chair, Council of Deans, University at Albany, SUNY
- 1997-2008. Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
- 2000-Pres. Member, Board of Directors, Healthy Schools Network, Inc.
- 2000-2003 Member, Medical Advisory Board, Hepatitis C Coalition, New York
- 2000-2004 Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
- 2001-2008 Member, Board of Directors, Environmental Advocates of New York
- 2004-2007 Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
- 2005-Pres. Member, Schooling Chefs Curriculum Advisory Board
- 2005-2008 Member, Board of Directors, Citizens Environmental Coalition
- 2006-2009 Member, Board of Directors, Marine Environmental Research Institute
- 2007-2009 Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

- 1959 B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, Ambystoma tigrinum" (Thesis advisor, Professor George Wald)
Elected to Phi Beta Kappa and to Sigma Xi
- 1964 M.D. awarded cum laude for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
- 1964 Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
- 1970 Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
- 1971 Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months

- 1982, 1986 Visiting Professor of Physiology, Department of Physiology, Kyushu
 1987 University, Fukuoka, Japan, for a period of three months each
 1989 Awarded Jacob Javits Neuroscience Investigator Award from the National
 Institute of Neurological and Communicative Diseases and Stroke
 1999 Awarded Homer N. Calver Award from the American Public Health
 Association for studies in environmental health.
 2001 Awarded 2001 Academic Laureate from the University at Albany
 Foundation.
 2010 Awarded the Albion O. Bernstein, M.D. Award in recognition of an
 outstanding contribution to public health and the prevention of disease through
 lifelong research of environmental health hazards and for limitless devotion to
 medical education by the Medical Society of the State of New York.

Federal Grants Held: (Principal Investigator Only)

- 1980-1983 United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs",
 \$76,847 total direct costs.
 1982-1988 National Institute of Health, "Mechanisms of Desensitization at Central Synapses",
 \$464,786 total direct costs.
 1984-1986 Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs@,
 \$330,504 total direct costs.
 1986-1996 National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and
 Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct
 costs.
 1989-1993 National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total
 direct costs
 1990-1995 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O.
 Carpenter, P.I. \$5,783,419 total direct costs.
 1995-2001 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Central/Eastern European
 Environ/Occup Training Program@, D.O. Carpenter, P.I. \$657,520 total costs.
 1995-2001 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total
 direct costs.
 1998-1999 Environmental Protection Agency, A Indoor Air Risk at Akwesasne - Pilot Project@,
 D.O. Carpenter, P.I. \$9,996 total costs.
 2000-2002 Association Liaison Office for University Cooperation in Development,
 A Cooperative Program in Environmental Health between the Institute of Public
 Health at Makerere University, Kampala, Uganda and the School of Public Health,
 University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
 2001-2007 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Multidisciplinary
 Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
 2006-2011 Pakistan-US Science and Technology Cooperative Program (US National
 Academy of Sciences). "Association of particulate matter with daily morbidity in

- an urban population,” D.O. Carpenter, P.I., \$391,104 total costs.
- 2009-2013 Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2: Environmental contaminants and reproductive health of Akwesasne Mohawk women. \$387,825 for year 1. D.O. Carpenter, Co-PI.
- 2010-2013 Department of the Army, “Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, “Drinking Water Supply, Sanitation, and Hygiene Promotion : Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda”. D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1R01ES019620), “Protecting the health of future generations: Assessing and preventing exposures.” PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2,354,871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson’s Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001. Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.

6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibibility of motoneurons of different sizes. J Neurophysiol, 28:599-620, 1965.
7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. J Neurophysiol, 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J Pharmacol, 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. J Neurophysiol, 28:958-965, 1965.
10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch Ital Biol, 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J Neurophysiol, 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in *Aplysia* neurons. J Gen Physiol, 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv Pharmacol, 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na⁺ pump to membrane potential in *Aplysia* neurons. J Gen Physiol, 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch Neurol, 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na⁺ pump in *Aplysia* neurons. Comp Biochem Physiol, 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of *Aplysia* neurons upon Na⁺ and Ca⁺⁺. J Cell Physiol, 75:121-127, 1970.
18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepinephrine in the presence of tetrodotoxin. J Pharmacol Exp Therap, 73:416-421, 1970.
19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.
20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of *Aplysia* neurons and squid axon as determined by a new technique. Intl J Neurosci, 2:35-48, 1971.
21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in *Aplysia* nervous and non-nervous tissues. Intl J Neurosci, 2:49-56, 1971.
22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of *Aplysia* neuron somata. Science, 176:1329-1331, 1972.
23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. Science, 179:1336-1338, 1973.
24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. J Physiol (Lond), 229:471-493, 1973.
25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. *Aplysia* acetylcholine receptors: Blockage by and binding of α -bungarotoxin. Brain Res, 72:225-240, 1974.
26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. Brain Res, 73:156-160, 1974.

27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in *Aplysia* suggests neurotransmitter function. *Science*, 185:364-365, 1974.
28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of *Aplysia* neurons. *J. Cell. Physiol.*, 84:463-472, 1974.
29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of *Aplysia*. *Proc. Natl. Acad. Sci. (USA)*, 71:4662-4665, 1975.
30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on *Aplysia* neurons mediate hyperpolarization by increasing membrane conductance. *Nature*, 252:483-485, 1974.
31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. *J. Neurobiol.*, 38:601-612, 1975.
32. Carpenter, D.O. and Gaubatz, G.L. H₁ and H₂ histamine receptors on *Aplysia* neurons. *Nature*, 254:343-344, 1975.
33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal resistivity of giant axons of squid and *Myxicola*. *J. Gen. Physiol.*, 66:139-148, 1975.
34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of *Aplysia*. *Comp. Biochem. Physiol.*, 52C:23-26, 1975.
35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents *in vitro*. *Pflugers Arch.*, 359:349-356, 1975.
36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on *Aplysia* neurons. *Nature*, 258:751-754, 1975.
37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on *Aplysia* neurons. *Science*, 192:806-809, 1976.
38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. *Biophys. J.*, 16:991-1001, 1976.
39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α -bungarotoxin with *Aplysia* acetylcholine receptors. *Mol. Pharmacol.*, 12:999-1006, 1976.
40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in *Aplysia*. *Science*, 195:1004-1006, 1977.
41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in *Aplysia* neurons. *J. Neurobiol.*, 8:119-132, 1977.
42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on *Aplysia* neurons. *Life Sci.*, 20:1441-1448, 1977.
43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. *J. Electrophysiol. Tech.*, 6:34-41, 1977.
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2009. Paula Maccabee, Attorney. 651-775-7128.

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Ronald Cybart et al., Michael Campanelli, and Donald and Theresa Shea, et al.v. CL&P. Deposed for the plaintiffs. 15 July 2011. Benson A. Snaider, Attorney.

Maria Snoops vs. Lyon Associates, Inc. and Insurance Co of the state of Pennsylvania. Deposed for the plaintiff, 1 November 2011. Matthew J. Witteman, Attorney. 415-363-3106.

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United States District Court

District of Oregon

Portland Division

AHM, by and through
her Guardian *ad litem* and father,
David Mark Morrison, and
David Mark Morrison, individually,

v.

Portland Public Schools,

Defendant.

Civil Action No. 3:11-cv-00739-MO

**Amended Declaration of
Dr. David O. Carpenter, M.D.**

I, Dr. David O. Carpenter, M.D., under penalty of perjury pursuant to 28 U.S.C. § 1746, hereby make the following declaration in support of an injunction against Portland Public Schools' use of WI-FI:

1. I am a public health physician, educated at Harvard Medical School. My current title is Director of the Institute for Health and the Environment at the University at Albany and Professor of Environmental Health Sciences within the School of Public Health. Formerly, I was the Dean of the School of Public Health at the University of Albany and the Director of the Wadsworth Center for Laboratories and Research of the New York State Department of Health.

2. I served as the Executive Secretary to the New York State Powerlines Project in the 1980s, a program of research that showed children living in homes with elevated magnetic fields coming from powerlines suffered from an elevated risk of developing leukemia. After this I became the spokesperson on electromagnetic field (EMF) issues for the state during the time of my employment in the Department of Health. I have published several reviews on the subject and have edited two books.

3. I am a Co-Editor and a Contributing Author of the *BioInitiative: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)*, www.bioinitiative.org. It documents bioeffects, adverse health effects and public health conclusions about impacts of electromagnetic radiation (electromagnetic fields including extremely-low frequency ELF-EMF and radiofrequency /microwave or RF-EMF fields). The public health chapter from this report was subsequently published in a peer-reviewed journal.

4. Additionally, I am a Co-Author of *Setting Prudent Public Health Policy for Electromagnetic Field Exposures*, *Reviews on Environmental Health*, Volume 23, No 2, 2008, attached as Addendum A-2.

5. In addition, in 2009, I was invited to present to the President's Cancer Panel on the subject of powerline and radiofrequency fields and cancer, and have testified on this issue before the United States House of Representatives.

6. In sum, I am a public health physician, professor and former public health school Dean with expertise in electrophysiology, low-frequency electromagnetic fields bioeffects, and

radiofrequency (RF) and microwave (MW) radiation bioeffects.

7. WI-FI deploys pulse-modulated (“PM”) microwave (“MW”) radiation (within the larger RF radiation spectrum) with a carrier frequency that is similar to that used by a microwave oven: about 2.45 GHz. This is the “Agent”. The 2.45 GHz frequency was chosen for the oven because of its wavelength and harmonic resonance with the water molecule, to ensure the most efficient absorption by living tissues and effective heating by way of the agitation of water at the molecular level. The pulse-modulation of a wave with lower frequencies in addition to the high-frequency carrier signal, increases the exposure complexity and in turn the bioeffects in an exposed population.

8. In the context of school development, WI-FI exposes building occupants including children and adults constantly from both computers and infrastructure antennas. Duration may be an even more potent contributing factor to RF/MW radiation bioeffects than exposure levels. Chronic, such as all-day, school exposure, is more likely than short and intermittent exposure, such as cell phone use, to produce harmful health effects, and is likely to do so at lower exposure levels.

9. Persons stationed close to school computers with WI-FI and especially those very near to any WI-FI infrastructure will receive considerably higher exposure than do others.

10. It is generally accepted within the relevant scientific community and has been established beyond any reasonable doubt that adverse human health effects occur at far lower levels of RF/MW radiation exposure than those that cause noticeable heating, particularly where the wavelength approaches body-part size and thus maximizes absorption, where the wavelength has resonance with the water molecule, where there is more complex, modulated wave, where there is chronic exposure duration, and where exposed persons lack the capacity voluntarily to remove themselves from radiation sources.

11. Some effects are shown to occur at several hundred thousand times below the FCC public exposure guidelines, which are set based on the fallacious assumption that there are no adverse health effects at exposures that do not cause easily measureable heating. FCC guidelines

also only apply to 30-minute public exposures; therefore do not even infer safety at durations >30 minutes, such as in a school setting.

12. Exposure to high-frequency RF and MW radiation and also the extreme low frequency (ELF) EM fields that accompany WI-FI exposure have been linked to a variety of adverse health outcomes. Some of the many adverse effects reported to be associated with and/or caused by ELF fields and/or RF/MW radiation include neurologic, endocrine, immune, cardiac, reproductive and other effects, including cancers.

13. Studies of isolated cells have shown that RF/MW exposures may cause changes in cell membrane function, cell communication, metabolism, activation of proto-oncogenes, and can trigger the production of stress proteins at exposure levels below FCC guidelines and also at and less than school WI-FI exposure levels and parameters. Resulting effects in cellular studies include without limitation DNA breaks and chromosome aberrations, cell death including death of brain neurons, increased free radical production, activation of the endogenous opioid system, cell stress and premature aging.

14. Human studies of comparable RF/MW radiation parameters show changes in brain function including memory loss, retarded learning, performance impairment in children, headaches and neurodegenerative conditions, melatonin suppression and sleep disorders, fatigue, hormonal imbalances, immune dysregulation such as allergic and inflammatory responses, cardiac and blood pressure problems, genotoxic effects like miscarriage, cancers such as childhood leukemia, childhood and adult brain tumors, and more.

15. There is consistent evidence for increased incidence of effects in individuals who live near to high-power short-wave, AM, FM and TV transmission towers. This is particularly relevant because, like WI-FI, radio-TV transmission towers give continuous, whole-body radiation, not just radiation to the head, constantly.

16. Since WI-FI transmitters, both infrastructural and on computers, are indoors, where children and teachers may be very close by, and since WI-FI, at 2.45 GHz, deploys a

wavelength, at ~12.2 cm or ~ 4.8 inches, more absorbable by children's and adults' bodies and brains than radio-TV wavelengths, the harmfulness of WI-FI radiation likely exceeds that of radio-TV towers.

17. Like second-hand smoke, EMF and RF/MW radiation involve complex mixtures, where different frequencies, intensities, durations of exposure(s), modulation, waveform and other factors are known to produce variable effects, often more harmful with greater complexity. Decades of scientific study have produced substantial evidence that EMF and RF/MW radiation may be considered neurotoxic, carcinogenic and genotoxic. Sources of fields and radiation, but are not limited to: power lines, navigational radar, cell phones, cordless phones [or Digitally Encoded Cordless Transmission Devices (D.E.C.T.) phones], cell towers, 'smart' meters and their grids or infrastructure, "smart" boards, meters and grids, WiMax and wireless internet (WI-FI).

18. The RF/MW radiation and low-frequency EMF science that currently exists includes tens of thousands of studies dating back to the 1920s. On the basis of this vast body of literature, many public health experts believe, myself included, that it is likely society will face epidemics of neurotoxic effects and degeneration, cancers and genotoxicity in the future, resulting from the extreme and mostly involuntary exposure to RF/MW radiation and EMFs. WI-FI radiation in schools exceeds natural background levels of microwave radiation by trillions of times. Thus, it is important that all of us restrict our use of cell phones, and be as free as possible from exposure to unnatural, background sources of MW radiation, particularly WI-FI.

19. In public health science, it is generally accepted fact that vulnerable subgroups exist within any human population. This is also recognized specifically for RF/MW radiation and fields. These groups include children, pregnant women, the elderly and those with preexisting illnesses and/or impairments. Children are more vulnerable to RF/MW radiation because of the susceptibility of their developing nervous systems. RF/MW penetration is greater relative to head size in children, who have a greater absorption of RF/MW energy in the tissues of the head at WI-FI frequencies.

Such greater absorption results because children's skulls are thinner, their brains smaller, and their brain tissue is more conductive than those of adults, and since it has a higher water content and ion concentrations. The Presidential Cancer Panel found that children 'are at special risk due to their smaller body mass and rapid physical development, both of which magnify their vulnerability to known carcinogens, including radiation.'

http://deainfo.nci.nih.gov/advisory/pcp/annualReports/pcp08-09rpt/PCP_Report_08-09_508.pdf

20. FCC public RF/MW radiation exposure guidelines are based on the height, weight and stature of a 6-foot tall man, not children or adults of smaller stature. The guidelines do not take into account the unique susceptibility of growing children to exposures. Since children are growing, their rate of cellular activity and division is more rapid, and they are at more risk for DNA damage and subsequent cancers. Growth and development of the central nervous system is still occurring well into the teenage years, such that the neurological impairments predictable by the extant science may have great impact upon development, cognition, learning, and behavior. Prenatal exposure has been identified as a risk factor for childhood leukemia, and is associated with miscarriage. Children are largely unable to remove themselves from exposures to harmful substances in their environments. Their exposure is involuntary.

21. When WI-FI is in operation in a school, children and their parents have no choice but to allow the school to expose them to trillions of times higher microwave radiation than exists naturally on Earth at the same frequencies. Children and other building users are exposed to as much as 30-40 hours per week of constant, digitally encoded WI-FI signals from each wireless device and infrastructural antenna in a school building. Based upon a review of the Mount Tabor WI-FI Floor Plan, a given child is subject to direct signals from multiple WI-FI transmitters, including rooms full of students and teachers transmitting numerous laptop and other wireless signals. There is a major legal difference between an exposure that an individual chooses to accept and one that is forced upon a person, especially a dependent, who can do nothing about it.

22. WI-FI in the Portland Schools deploys similar PM MW radiation, at 2.45 and 5 GHz, to that of cell and cordless phones and their infrastructure. There is clear and strong evidence that intensive use of cell phones increases incidence of brain cancer, tumors of the auditory nerve, and cancer of the parotid gland, the salivary gland in the cheek by the ear. Cell and cordless phone radiation closely resembles that of WI-FI radiation exposure, except that WI-FI is more hazardous by way of frequency, duration, and the involuntary nature of exposure. While a cell or cordless phone is used only intermittently and primarily voluntarily, a WI-FI radiation microenvironment is constant in duration, with unavoidable radiation exposure even when nearby students are not actively using it. Because WI-FI radiation is essentially the same as, but more hazardous than, that for cell and cordless phones, there is every reason to understand that the health effects will be the same or worse, varying in relation to the total dose of radiation, and intensified by the constancy of duration. There is evidence from Scandinavian studies of cell phone usage that children who use cell phones are about five times more likely to develop brain cancer than if their usage starts as an adult. Thus, it is especially necessary to protect children from pulse-modulated MW radiation such as both cell phones and WI-FI deploy.

23. Based on a high degree of scientific certainty, Portland Public Schools' use of WI-FI is causing and will continue to cause AHM, other students, and school staff and faculty adverse health effects, and should be discontinued immediately. Educating by way of the Internet via cabled systems only decreases MW radiation exposure and is of minimal expense.

24. Having reviewed hundreds, possibly thousands, of studies in RF/MW radiation and ELF fields, published from decades ago to the present, I would provide you the following primary evidence, without limitation. Due to the active suppression of the RF/MW literature, some researchers in public health science are less aware of these studies. However, the forefront experts specializing in these areas, RF/MW radiation and ELF fields, recognize the certainties in this large body of scientific literature, which establishes without limitation that PM MW radiation with chronic duration is quite harmful to humans, particularly children, as well as to animals and plants.

25. It is not surprising that even as of 1990, the US Environmental Protection Agency ("EPA") had determined RF/MW radiation a "probable carcinogen". Now that we have much more confirming study in the interim, the conclusion is yet more certain. And when we focus on MW radiation, particularly pulse-modulated radiation, on long, non-intermittent duration and on more vulnerable subgroups such as children, we see that the cancer outcome is very certain, indeed. Amongst the epidemiologic studies showing cancer outcomes, the following are particularly strong:

- a. Dode AC, Leao M, Tejo FdeAF, gomes ACR, Dode DC, Dode MC, Moreira CW, Condessa VA, Albinatti C and Calaffa WT. Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais State, Brazil. *Sci Total Environ* 409: 3649-3665:2011. This study shows higher rates of cancer in people living close to cell phone towers than for people living further away. Cell phone radiation is similar to but likely not as harmful as 2.45 GHz radiation from WI-FI. The exposure levels in this study are lower than those that Portland school building occupants receive from WI-FI.
- b. Oberfeld G. Environmental Epidemiology Study of Cancer Incidence in the Municipalities of Hausmannstatten & Vasoldsberg (Austria), 2008. This government-commissioned study found significantly increased cancer risk relative to a lower-exposure reference category, 23x higher for breast cancer and 121x higher for brain tumors, with strong exposure-effect relations.
- c. Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, Barca A and Perucci CA. Adult and childhood leukemia near a high-power radiostation in Rome, Italy. *Am J Epidemiol.* 155: 1098-1103: 2002. The authors show that there is a significant elevation of childhood leukemia among residents living near to Vatican Radio, and that the risk declines with distance away from the transmitter. This is RF radiation in frequencies similar to that of WI-FI.

- d. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM and Pack JK. Radio-frequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. *Am J Epidemiol* 166: 270-279: 2007. Leukemia and brain cancer in children in Korea were investigated in relation to residence within 2 km of AM radio transmitters. There was a significant elevation in rates of leukemia but not of brain cancer. WI-FI radiation is more harmful than AM.
- e. Park SK, Ha M, Im HJ. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. *Int Arch Occup Environ Health*. 2004 Aug;77(6):387-94. This study found higher mortality areas for all cancers and leukemia in some age groups in the area near the AM towers.
- f. Hallberg O. Johansson O. *Med Sci Monit* 2004 Jul;10(7):CR336-40. Malignant melanoma of the skin – not a sunshine story! Increased incidence and mortality from skin melanoma are concluded to result from continuous disturbances of cell repair mechanisms by body-resonant EMFs from FM/TV networks.
- g. Hallberg O. Johansson O. 2005. FM Broadcasting exposure time and malignant melanoma incidence, *Electromagnetic Biology and Medicine* 24;1-8. Age-specific incidence of malignant melanoma of the skin is related to FM broadcasting radiation at whole-body resonant frequencies. This is very relevant to children, since the smaller wavelengths of WI-FI are at resonant frequencies with dimensions of the human head, particularly the child's head.
- h. Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt I, Elliot P. Cancer Incidence near radio and television transmitters in Great Britain. I – Sutton-Colfield transmitter, and II. A1 high-power transmitters. *Am J Epidemiol* 1997; 145(1):1-9 and 10-17. In the first study, there was a statistically significant

increase in cancer; in the second, a small but significant increase in adult leukemia.

i. Hocking B, Gordon IR, Grain HL, Harfield GE. Cancer incidence and mortality and proximity to TV towers. *Medical J of Australia*. 1985;165:601-605. At extremely low exposure levels, there was an association between increased childhood leukemia incidence and mortality and proximity to TV towers. TV radiation, in the VHF and UHF bands, is similar to but not as harmful as WI-FI radiation at 2.45 GHz.

j. Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: A nested case-control study. *Am J Epidemiol* 1996; 143:480-6. This study found an association between exposure to ELF and RF/MW radiation and brain tumors.

k. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. *Sci Total Environ* 1996;180:9-17. This study showed huge increases in leukemia and Non-Hodgkin's lymphomas. Though exposure levels are higher in this study than they would be with school WI-FI, it is possible that certain students or teachers stationed immediately next to the WI-FI infrastructure could receive comparable levels in radiation peaks.

26. Additional studies show neurologic, immune, endocrine, reproductive and cardiac, adverse health effects from low-dose, chronic exposure to RF/MW radiation in humans:

a. Papageorgiou CC, Hountala CD, Maganioti AE, Kyprianou MA, Rabavilas AD, Papadimitriou GN, Capsalis CN. Effects of WI-FI signals on the p300 component of event-related potentials during an auditory listening task. *J Integr Neurosci* 2011 Jun;10(2):189-202. This study concludes that WI-FI exposure may exert gender-related alterations on neural activity.

- b. Altpeter ES, Roosli M et al. Effect of Short-wave magnetic fields on sleep quality and melatonin cycle in humans: The Schwarzenburg shut-down study. *Bioelectromagnetics* 27:142-150, 2006. Sleep quality improved and melatonin excretion increased when the transmitter was shut down.
- c. Abelin T et al. Sleep disturbances in the vicinity of the short-wave broadcast transmitter Schwarzenburg. *Somnologie* 9:203-209, 2005. There is strong evidence of a causal relationship between operation of a short-wave radio transmitter and sleep disturbances in the surrounding population.
- d. Hutter HP et al. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup Environ Med* 2006;63:307-313, 2006. There was a significant relation of some symptoms, especially headaches, to measured power density, as well as effects on wellbeing and performance.
- e. Preece AW, Georgious AG, Duunn EJ, Farrow SC. *Occup Environ Med* 2007 Jun;64(6):402-8. Compared to control village, there were highly significant differences in the reporting of migraine, headache and dizziness military and cell phone antenna systems.
- f. Buchner K, Eger, H. Changes of clinically important neurotransmitters under the influence of modulated RF fields – a long-term study under real-life conditions. *Umwelt-Medizin-Gesellschaft* 24(1):44-57, 2011. There is clear evidence of health-relevant effects, including increase in adrenaline/noradrenaline, subsequent decrease in dopamine from a new MW-emitting base station. During counterregulation, trace amine PEA decreased and remained decreased. Clinically documented increases in sleep problems, cephalgia, vertigo, concentration problems and allergies followed the onset of new microwave transmissions.

- g. Eliyahu I, Luria R, Hareuveny R, Margalioth M, Neiran N and Shani G . Effects of radiofrequency radiation emitted by cellular telephones on the cognitive functions of humans. *Bioelectromagnetics* 27: 119-126: 2006. A total of 36 human subjects were exposed to PM MW and were tested on four distinct cognitive tasks. Exposure to the left side of the brain slows left-hand response time in three of the four tasks.
- h. Barth A, Winker R, Ponocny-Seliger E, Mayrhofer W, Ponocny I, Sauter C and Vana N. *Occup Environ Med* 65: 342-345: 2008. A meta-analysis for neurobehavioural effects due to electromagnetic field exposure emitted by GSM mobile phones. The authors looked at 19 studies of cognitive function in cell phone users, and found in the meta-analysis that there is evidence for a decreased reaction time, altered working memory and increased number of errors in exposed persons.
- i. Augner C, Hacker GW, Oberfeld G, Florian M, Hitzl W, Hutter J and Pauser G. Effects of exposure to base station signals on salivary cortisol, alpha-amylase and immunoglobulin A. *Biomed Environ Sci* 23: 199-207: 2010. This was a human experimental study with exposure to PM MW radiation wherein immune indicators were monitored after five 50-minute sessions. The researchers found dose-dependent changes in cortisol and alpha-amylase.
- j. Avendano C, Mata A, Sanchez Sarimiento CA and Doncel GF. Use of laptop computers connected to internet through WI-FI decreases human sperm motility and increases sperm DNA fragmentation. *Fert Steril*, 2012, In press. In this study human sperm were exposed to WI-FI from a laptop, and were found to show reduced motility after a 4-hour exposure. The results are consistent with other publications (see Agarwal et al., *Fert Steril* 89: 124-128: 2008) that reported that those who use cell phone regularly have reduced sperm count.

k. Baste V, Riise T and Moen BE (2008) *Int J Epidemiol* 23: 369-377: 2008. Radiofrequency electromagnetic fields: male infertility and sex ratio of offspring. This is a study of Norwegian Navy personnel chronically exposed to RF fields on the job. The rates of infertility were related to level of exposure in a dose-dependent fashion.

27. Many toxicologic and other animal studies, of which the following are but a few, support conclusions of cancer, genotoxicity, neurotoxicity and other health outcomes from RF/MW radiation.

a. Sinha R. Chronic non-thermal exposure of modulated 2450 MHz microwave radiation alters thyroid hormones and behavior of male rats. *Int. J. Radiation Biol.* 84:6:505-513, 2008. This study of 2.45 GHz at levels and durations comparable to and less than those of school WI-FI concluded that the radiation was sufficient to alter the levels of thyroid hormone as well as emotional reactivity compared to controls.

b. Nittby H, Grafstrom G, Tian DP, Malmgren L, Brun A, Persson BRR, Salfors LG and Eberhardt J. *Bioelectromagnetics* 29: 219-232: 2008. This study showed cognitive impairment in rats after long-term exposure to PM MW radiation. This study of rats shows that after 2 hours per week for 55 weeks there was impaired memory for objects in exposed as compared to sham animals.

c. Kimmel S et al. Electromagnetic radiation: Influences on honeybees (*Apis mellifera*). A significant difference between non-exposed and fully irradiated bees was the result of the influence of high-frequency PM RF/MW radiation.

d. Panagopoulos DJ et al. Bioeffects of mobile telephony radiation in relation to its intensity or distance from the antenna. *Int. J Radiat Biol*, 86;(5):345-357, 2010. The PM MW radiations at 900 and 1800 MHz decreased the reproductive capacity by cell death induction, with an increased bioactivity “window” at 10

uW/cm², and still evident down to 1 uW/cm².

e. Everaert J, Bauwens D. A possible effect of electromagnetic radiation from mobile phone base stations on the number of breeding house sparrow (*passer domesticus*). *Electromagnetic Biology and Medicine*, 26:63-72, 2007.

Long-term exposure to higher-level low-intensity PM MW radiation negatively affects the abundance or behavior of House Sparrows in the wild.

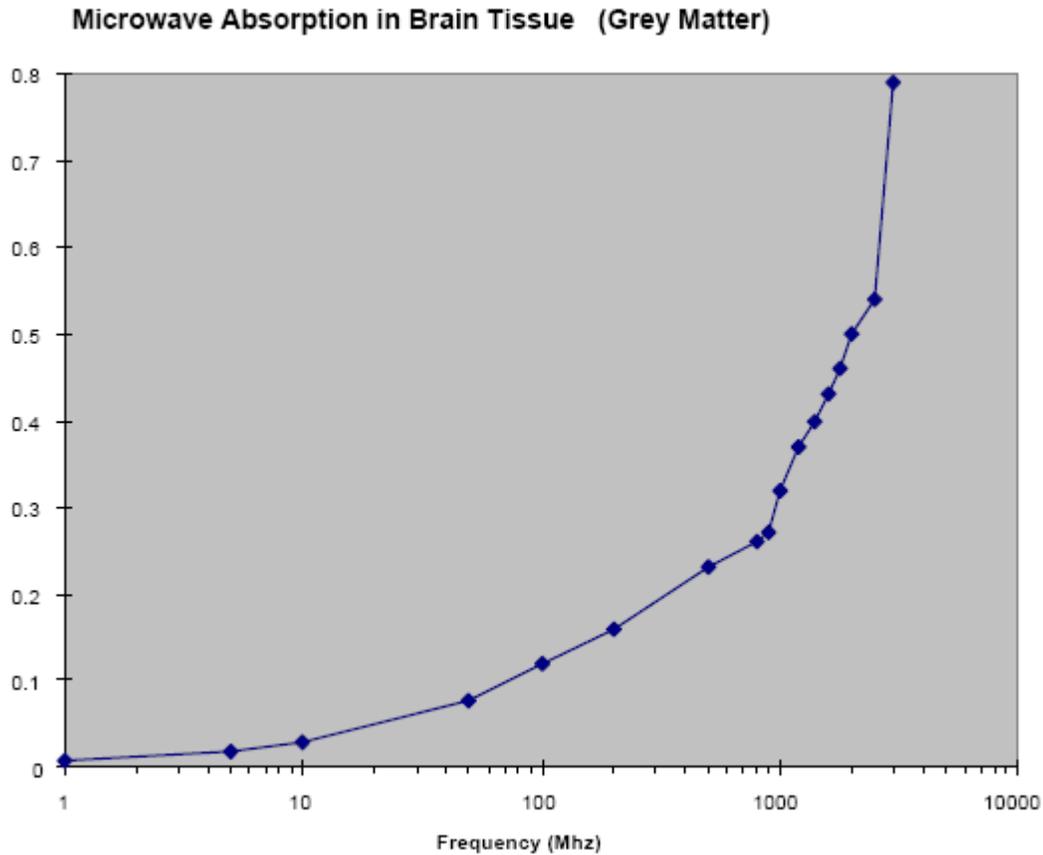
f. Magras I, Xenos T. RF Radiation-Induced Changes in the Prenatal Development of Mice. *Bioelectromagnetics* 18:455-461, 1997. Near almost 100 TV and FM broadcast transmitters, with exposure levels between 0.168 uW/cm² and 1.053 uW/cm², found in the more exposed groups testicular damage and decreasing size of litters to irreversible infertility.

g. Balmori A. Electromagnetic pollution from phone masts. *Effects on wildlife, Pathophysiology* 2009. This large review of wildlife effects concludes, “pulsed telephony microwave radiation can produce effects on nervous, cardiovascular, immune and reproductive systems,” including damage to the nervous system by altering EEG and changes to the blood-brain barrier, disruption of the 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, growth problems, problems in building the nest or impaired fertility, embryonic development, hatching percentage, genetic and developmental problems, problems of locomotion, promotion of tumors and more.

28. Exposure thresholds for harmful effects are lowered in human populations and individuals when duration is increased. Due to the variability of thresholds for harmful effects both in the population and within the individual, there is no exposure power density that is safe. The School's WI-FI deploys arguably the worst possible frequency of 2.45 GHz, that of the

microwave oven, worst because it is most absorbable by the brain and most resonant with the water molecule, such that:

- a. absorption-per-exposure is maximized, dramatically lowering effects thresholds for population and individual effects; and
- b. water molecules in tissues and cells are highly agitated.



Curry, Ph.D., *Wireless LANs in the schoolroom*

29. This above graph, from physicist William Curry PhD's presentation *Wireless LANs in the Schoolroom*, shows how absorption in brain tissue (grey matter) increases exponentially toward the ultra-high frequency (UHF) area of the microwave oven and WI-FI.

30. In the case of the Portland Schools, the additional, unused but still deployed carrier frequency of 5 GHz would likely increase absorption in other, smaller organs, such as the thyroid.

31. The graph also illustrates the problem with the drive of the wireless industry toward ever higher frequencies within the cm microwave band. While nearly all the lower frequency bands have already been allocated by the FCC for specific types of radio transmissions, and transmission of ever more information content on any given channel requires greater bandwidth, each new deployment undermines further the integrity of the population's health. Engineers who design these systems have no training that would qualify them to consider the effects on biologic systems, which is why public health scientists need to be called in to policymaking *prior to* contracting and deployment, not after the fact.

32. The following studies explain the mechanisms of interaction between RF/MW radiation and biologic systems at the cellular level.

- a. The cell membrane recognition process -- which includes signal transduction and 'heat-shock protein' release -- was first discerned by Litovitz and his co-workers at Catholic University of America in the mid-1990s.

Below are a few citations that make the point.

- i. Litovitz, T., C. Montrose, et al. (1994). "Superimposing spatially coherent electromagnetic noise inhibits field induced abnormalities in developing chick embryos." *Bioelectromagnetics* **15**(2): 105-113.
- ii. DiCarlo, A., J. Farrell, et al. (1998). "A simple experiment to study electromagnetic field effects: Protection induced by short term exposures to 60 Hz magnetic fields." *Bioelectromagnetics* **19**(8): 498-500.
- iii. Penafiel, L., T. Litovitz, et al. (1997). "Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929

- cells." *Bioelectromagnetics* **18**(2): 132-141.
- iv. Dicarlo, A. L., Michael T. Hargis, L. Miguel Penafiel, Theodore A. Litovitz, A. (1999). "Short-term magnetic field exposures (60Hz) induce protection against ultraviolet radiation damage." *International journal of radiation biology* **75**(12): 1541-1549.
 - v. Litovitz, T., C. Montrose, et al. (1990). "Amplitude windows and transiently augmented transcription from exposure to electromagnetic fields." *Bioelectromagnetics* **11**(4): 297-312.
 - vi. Litovitz, T., M. Penafiel, et al. (1997). "The role of temporal sensing in bioelectromagnetic effects." *Bioelectromagnetics* **18**(5): 388-395.
 - vii. Litovitz, T., L. Penafiel, et al. (1997). "Role of modulation in the effect of microwaves on ornithine decarboxylase activity in L929 cells." *Bioelectromagnetics* **18**: 132-141.]
 - viii. Litovitz, T., D. Krause, et al. (1993). "The role of coherence time in the effect of microwaves on ornithine decarboxylase activity." *Bioelectromagnetics* **14**(5): 395-403.
- b. Cell membrane reaction is lipid peroxidation.
 - i. Serban, M. and V. Ni (1994). "Lipid peroxidation and change of plasma lipids in acute ischemic stroke." *Romanian journal of internal medicine= Revue roumaine de médecine interne* **32**(1): 51.

- ii. Vileno, B., S. Jeney, et al. (2010). "Evidence of lipid peroxidation and protein phosphorylation in cells upon oxidative stress photo-generated by fullerols." *Biophysical chemistry*.
 - iii. Maaroufi, K., E. Save, et al. (2011). "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." *Neuroscience*.
 - iv. Nelson, S. K., S. K. Bose, et al. (1994). "The toxicity of high-dose superoxide dismutase suggests that superoxide can both initiate and terminate lipid peroxidation in the reperfused heart." *Free Radical Biology and Medicine* **16**(2): 195-200.
 - v. Alvarez, J. G. and B. T. Storey (1989). "Role of glutathione peroxidase in protecting mammalian spermatozoa from loss of motility caused by spontaneous lipid peroxidation." *Gamete research* **23**(1): 77-90.
 - vi. Devasagayam, T., K. Bloor, et al. (2003). "Methods for estimating lipid peroxidation: An analysis of merits and demerits." *Indian journal of biochemistry & biophysics* **40**(5): 300-308.
- c. Free-Radical Damage:
- i. Ozgur, E., G. Güler, et al. (2010). "Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants n-acetyl cysteine and epigallocatechin-gallate." *International journal of radiation biology*(00): 1-11.

- ii. Gutteridge, J. and X. C. Fu (1981). "Enhancement of bleomycin-iron free radical damage to DNA by antioxidants and their inhibition of lipid peroxidation." *FEBS letters* **123**(1): 71.
- d. mRNA:
 - i. Yan, J. G., M. Agresti, et al. (2009). "Qualitative Effect on mRNAs of Injury-Associated Proteins by Cell Phone Like Radiation in Rat Facial Nerves." *Electromagnetic Biology and Medicine* **28**(4): 383-390.
 - ii. Yan, J. G., M. Agresti, et al. (2008). "Upregulation of specific mRNA levels in rat brain after cell phone exposure." *Electromagnetic Biology and Medicine* **27**(2): 147-154.
 - iii. Simbürger, E., A. Stang, et al. (1997). "Expression of connexin43 mRNA in adult rodent brain." *Histochemistry and cell biology* **107**(2): 127-137.
 - iv. Chen, J., H. C. He, et al. (2010). "Effects of Pulsed Electromagnetic Fields on the mRNA Expression of RANK and CAII in Ovariectomized Rat Osteoclast-Like Cell." *Connective Tissue Research* **51**(1): 1-7.
- e. Epigenetic changes.... environmentally induced genetic change:
 - i. Migliore, L. and F. Copped (2009). "Genetics, environmental factors and the emerging role of epigenetics in neurodegenerative diseases." *Mutation Research/Fundamental and Molecular*

Mechanisms of Mutagenesis **667**(1-2): 82-97.

- ii. Currenti, S. (2009). "Understanding and Determining the Etiology of Autism." *Cellular and Molecular Neurobiology* **30**(2): 161-171.

f. Micronuclei formation:

- i. Tice, R. R., G. G. Hook, et al. (2002). "Genotoxicity of radiofrequency signals. I. Investigation of DNA damage and micronuclei induction in cultured human blood cells." *Bioelectromagnetics*, **23**(2): 113-126.
- ii. Lerchl, A. (2009). "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)." *Int Arch Occup Environ Health* **82**(2): 275-278.
- iii. Vijayalaxmi and T. J. Prihoda (2009). "Genetic damage in mammalian somatic cells exposed to extremely low frequency electro-magnetic fields: a meta-analysis of data from 87 publications (1990-2007)." *Int J Radiat Biol* **85**(3): 196-213.
- iv. Sannino, A., M. Sarti, et al. (2009). "Induction of adaptive response in human blood lymphocytes exposed to radiofrequency radiation." *Radiat Res* **171**(6): 735-742.

g. DNA repair disruption:

- i. Brusick, D., R. Albertini, et al. (1998). "Genotoxicity of radiofrequency radiation. DNA/Genetox Expert Panel." *Environ*

- Mol Mutagen* **32**(1): 1-16.
- ii. Belyaev, I. Y., E. Markova, et al. (2009). "Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes." *Bioelectromagnetics* **30**(2): 129-141.
 - iii. Sun, L. X., K. Yao, et al. (2006). "[Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro]." *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* **24**(8): 465-467.
 - h. Immune response suppression:
 - i. Lyle, D. B., P. Schechter, et al. (1983). "Suppression of T-lymphocyte cytotoxicity following exposure to sinusoidally amplitude-modulated fields." *Bioelectromagnetics* **4**(3): 281-292.
 - ii. Elekes, E., G. Thuroczy, et al. (1996). "Effect on the immune system of mice exposed chronically to 50 Hz amplitude-modulated 2.45 GHz microwaves." *Bioelectromagnetics* **17**(3): 246-248.
 - iii. DABALA, D., D. SURCEL, et al. (2008). "Oxidative and Immune Response in Experimental Exposure to Electromagnetic Fields." *Electromagnetic field, health and environment: proceedings of EHE'07*: 105.
 - iv. Surcel, D., D. Dabala, et al. (2009). "Free Radicals, Lipid Peroxidation and Immune Response in Experimental Exposure to Electromagnetic Fields." *Epidemiology* **20**(6): S118.

Conclusions

33. To understand the seriousness of this Agent of PM RF/MW radiation in interaction with populations and individuals, we need to consider some basic facts in addition to the many relevant and reliable studies above. For example, where shortwave, AM, FM, TV and cell phone infrastructure frequencies are demonstrated to be harmful, as they consistently are shown to be at low intensities with long duration, then, all other factors being equal, MW radiation at 2.45 GHz will likely be more harmful yet, due to its higher absorption-per-exposure and water molecule resonance. Increasing the constancy and length of exposure toward the maximum of occupational and 24-7 durations will lower the threshold for effects in populations and individuals. Complex radiation microenvironments with pulse-modulated wave and multiple sources, such as are deployed in WI-FI-equipped schools, are more harmful than a single, isolated MW radiation exposure at the same power density and duration. There are only a few of the many studies of RF/MW radiation infrastructure such as base stations that fail to show their studied effect. However, even were the reverse true, i.e., if there existed greater number than those that do show adverse effects, it is the case that positive studies (those that show adverse effects) hold more weight than negative studies (those that show no effect).

34. The FCC-appointed guideline-setting Commission, ASTM-IEEE, in 1991 referred in its conclusions to RF/MW radiation, the Agent, as a ‘Hazard,’ specifically setting a ‘Hazard Threshold.’ It has been discovered that, even amongst the 120 studies chosen by the Committee to prove the validity of its Hazard Threshold, there were 15 studies that concluded adverse effects at levels *lower* than the Hazard Threshold, thus disproving its validity. Three of these studies actually showed adverse effects at less than 10 percent of the Hazard Threshold. Thus the guidelines have no credibility.

35. The large body of scientific literature moreover redundantly proves this Agent to be a hazard. The media-promulgated notion that the relevant scientific studies are inconsistent and inconclusive is false and misleading. Chronic exposure to PM MW radiation harms every individual in a population in some ways, even if these are not always detectable by the individual or consciously attributed to the responsible RF/MW radiation sources. This Agent injures some individuals into a condition in which symptoms will be more easily retriggered with subsequent exposure. And for *a priori* susceptible individuals and those using electronic medical devices, it can respectively exacerbate the extant medical conditions and disrupt medical device operation, even to the point of death. Bassen 1997 discusses the hundreds of excess deaths, even at that time, from wireless communications radiation. See also *Radiofrequency Interference with Medical Devices*, IEEE Engineering in Medicine and Biology Magazine 17(3):111-114(1998), <http://ewh.ieee.org/soc/embs/comar/interfer.htm>.

36. For these reasons, WI-FI must be banned from school deployment.

37. I will receive no compensation for my testimony beyond out-of-pocket expenses.

Dated this 20th day of December, 2011.



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Director, Institute for Health and the Environment
University at Albany

CURRICULUM VITAE

Name: David O. Carpenter

Home Address: 2749 Old State Road
Schenectady, New York 12303

Positions Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Education: 1959 B.A., Harvard College, Cambridge, MA
1964 M.D., Harvard Medical School, Boston, MA

Positions Held:

- 9/61-6/62 Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
- 7/64-6/65 Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
- 7/65-2/73 Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
- 2/73-3/80 Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
- 3/80-9/85 Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 9/85-1/98 Dean, School of Public Health, University at Albany
- 9/85-Pres. Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
- 9/85-7/98 Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
- 1/98-1/05 Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
- 2001-Pres. Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
- 2005-Pres. Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York

Editor-in-Chief: Cellular and Molecular Neurobiology, 1981 - 1987

Editorial Advisor: Cellular and Molecular Neurobiology, 1987 - Present

Editorial Boards: Journal of Public Health Management and Practice, 1995 - 2002
International Journal of Occupational Medicine & Environmental Health
1996 – Present

Journal of Alzheimer's Disease– Associate Editor,2007-2009
Reviews in Environmental Health; 2008-present
International Archives of Occupational and Environmental Health; 2009-present.
Journal of Environmental and Public Health, 2009-present.
Environmental Health Perspectives, 2010-present

National and International Committees:

- 1978, 1981 Physiology Study Section (Ad hoc member)
- 1979-1985 NIH International Fellowship Study Section
- 1974-1981 Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
- 1981-1989 Member, USA National Committee for the International Brain Research Organization
- 1985-1986 Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
- 1986-1987 Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
- 1987-1989 Member, Science Advisory Council of the American Paralysis Association
- 1987-1990 Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
- 1985-1993 Committee #79, National Council on Radiation Protection and Measurements
- 1986-1997 Member, Legislative and Education Committees, Association of Schools of Public Health
- 1989-1994 Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
- 1994, 1995 Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
- 1994-1997 Member, Legislative Committee of the Association of Schools of Public Health
- 1997 Member, Executive Committee of the Association of Schools of Public Health
- 1997-2000 National Advisory Environmental Health Sciences Council of the National Institutes of Health
- 1998-Pres. Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
- 2000-Pres. Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
- 2001-2008 United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
- 2002-2003 Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
- 2003-2008 Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
- 2003-Pres. Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
- 2007-2011 Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.

State and Local Committees:

- 1980-1987 Executive Secretary, New York State Power Lines Project
- 1985-1989 Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
- 1986-1989 Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
- 1991-1992 Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
- 1991-1992 Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
- 1991-1992 Member, State Task Force for the Reform of Middle Level Education in NY State
- 1992-1993 Member, State Needs Task Force on Health Care and Education
- 1987-1998 Delegate-at-Large, New York State Public Health Association
- 1991-1995 Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
- 1994 Chair, Council of Deans, University at Albany, SUNY
- 1997-2008. Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
- 2000-Pres. Member, Board of Directors, Healthy Schools Network, Inc.
- 2000-2003 Member, Medical Advisory Board, Hepatitis C Coalition, New York
- 2000-2004 Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
- 2001-2008 Member, Board of Directors, Environmental Advocates of New York
- 2004-2007 Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
- 2005-Pres. Member, Schooling Chefs Curriculum Advisory Board
- 2005-2008 Member, Board of Directors, Citizens Environmental Coalition
- 2006-2009 Member, Board of Directors, Marine Environmental Research Institute
- 2007-2009 Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

- 1959 B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, Ambystoma tigrinum" (Thesis advisor, Professor George Wald)
Elected to Phi Beta Kappa and to Sigma Xi
- 1964 M.D. awarded cum laude for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
- 1964 Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
- 1970 Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
- 1971 Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months

- 1982, 1986 Visiting Professor of Physiology, Department of Physiology, Kyushu
 1987 University, Fukuoka, Japan, for a period of three months each
 1989 Awarded Jacob Javits Neuroscience Investigator Award from the National
 Institute of Neurological and Communicative Diseases and Stroke
 1999 Awarded Homer N. Calver Award from the American Public Health
 Association for studies in environmental health.
 2001 Awarded 2001 Academic Laureate from the University at Albany
 Foundation.
 2010 Awarded the Albion O. Bernstein, M.D. Award in recognition of an
 outstanding contribution to public health and the prevention of disease through
 lifelong research of environmental health hazards and for limitless devotion to
 medical education by the Medical Society of the State of New York.

Federal Grants Held: (Principal Investigator Only)

- 1980-1983 United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs",
 \$76,847 total direct costs.
 1982-1988 National Institute of Health, "Mechanisms of Desensitization at Central Synapses",
 \$464,786 total direct costs.
 1984-1986 Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs@,
 \$330,504 total direct costs.
 1986-1996 National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and
 Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct
 costs.
 1989-1993 National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total
 direct costs
 1990-1995 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O.
 Carpenter, P.I. \$5,783,419 total direct costs.
 1995-2001 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Central/Eastern European
 Environ/Occup Training Program@, D.O. Carpenter, P.I. \$657,520 total costs.
 1995-2001 National Institute of Environmental Health Sciences, Superfund Basic Research
 Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total
 direct costs.
 1998-1999 Environmental Protection Agency, A Indoor Air Risk at Akwesasne - Pilot Project@,
 D.O. Carpenter, P.I. \$9,996 total costs.
 2000-2002 Association Liaison Office for University Cooperation in Development,
 A Cooperative Program in Environmental Health between the Institute of Public
 Health at Makerere University, Kampala, Uganda and the School of Public Health,
 University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
 2001-2007 Fogarty International Center, National Institutes of Health, International Training
 Program in Environmental and Occupational Health. A Multidisciplinary
 Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
 2006-2011 Pakistan-US Science and Technology Cooperative Program (US National
 Academy of Sciences). "Association of particulate matter with daily morbidity in

- an urban population,” D.O. Carpenter, P.I., \$391,104 total costs.
- 2009-2013 Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2: Environmental contaminants and reproductive health of Akwesasne Mohawk women. \$387,825 for year 1. D.O. Carpenter, Co-PI.
- 2010-2013 Department of the Army, “Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, “Drinking Water Supply, Sanitation, and Hygiene Promotion : Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda”. D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1R01ES019620), “Protecting the health of future generations: Assessing and preventing exposures.” PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2,354,871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson’s Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001. Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.

6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibibility of motoneurons of different sizes. *J. Neurophysiol.*, 28:599-620, 1965.
7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. *J. Neurophysiol.*, 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. *J. Pharmacol.*, 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. *J. Neurophysiol.*, 28:958-965, 1965.
10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. *Arch. Ital. Biol.*, 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. *J. Neurophysiol.*, 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in *Aplysia* neurons. *J. Gen. Physiol.*, 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. *Adv. Pharmacol.*, 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na⁺ pump to membrane potential in *Aplysia* neurons. *J. Gen. Physiol.*, 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. *Arch. Neurol.*, 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na⁺ pump in *Aplysia* neurons. *Comp. Biochem. Physiol.*, 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of *Aplysia* neurons upon Na⁺ and Ca⁺⁺. *J. Cell. Physiol.*, 75:121-127, 1970.
18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepinephrine in the presence of tetrodotoxin. *J. Pharmacol. Exp. Therap.*, 73:416-421, 1970.
19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. *Science*, 169:597-598, 1970.
20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of *Aplysia* neurons and squid axon as determined by a new technique. *Intl. J. Neurosci.*, 2:35-48, 1971.
21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in *Aplysia* nervous and non-nervous tissues. *Int. J. Neurosci.*, 2:49-56, 1971.
22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of *Aplysia* neuron somata. *Science*, 176:1329-1331, 1972.
23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. *Science*, 179:1336-1338, 1973.
24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. *J. Physiol. (Lond.)*, 229:471-493, 1973.
25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. *Aplysia* acetylcholine receptors: Blockage by and binding of α -bungarotoxin. *Brain Res.*, 72:225-240, 1974.
26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. *Brain Res.*, 73:156-160, 1974.

27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in *Aplysia* suggests neurotransmitter function. *Science*, 185:364-365, 1974.
28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of *Aplysia* neurons. *J. Cell. Physiol.*, 84:463-472, 1974.
29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of *Aplysia*. *Proc. Natl. Acad. Sci. (USA)*, 71:4662-4665, 1975.
30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on *Aplysia* neurons mediate hyperpolarization by increasing membrane conductance. *Nature*, 252:483-485, 1974.
31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. *J. Neurobiol.*, 38:601-612, 1975.
32. Carpenter, D.O. and Gaubatz, G.L. H₁ and H₂ histamine receptors on *Aplysia* neurons. *Nature*, 254:343-344, 1975.
33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal resistivity of giant axons of squid and *Myxicola*. *J. Gen. Physiol.*, 66:139-148, 1975.
34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of *Aplysia*. *Comp. Biochem. Physiol.*, 52C:23-26, 1975.
35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents *in vitro*. *Pflugers Arch.*, 359:349-356, 1975.
36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on *Aplysia* neurons. *Nature*, 258:751-754, 1975.
37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on *Aplysia* neurons. *Science*, 192:806-809, 1976.
38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. *Biophys. J.*, 16:991-1001, 1976.
39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α -bungarotoxin with *Aplysia* acetylcholine receptors. *Mol. Pharmacol.*, 12:999-1006, 1976.
40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in *Aplysia*. *Science*, 195:1004-1006, 1977.
41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in *Aplysia* neurons. *J. Neurobiol.*, 8:119-132, 1977.
42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on *Aplysia* neurons. *Life Sci.*, 20:1441-1448, 1977.
43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. *J. Electrophysiol. Tech.*, 6:34-41, 1977.
44. Yarowsky, P.J. and Carpenter, D.O. Receptors for gamma-aminobutyric acid (GABA) on *Aplysia* neurons. *Brain Res.*, 144:75-94, 1978.
45. Carpenter, D.O., Gaubatz, G., Willis, J.A. and Severance, R. Effects of irradiation of *Aplysia* pacemaker neurons with 20 MeV electrons. *Rad. Res.*, 76:32-47, 1978.
46. Yarowsky, P.J. and Carpenter, D.O. A comparison of similar ionic responses to gamma-aminobutyric acid and acetylcholine. *J. Neurophysiol.*, 41:531-541, 1978.
47. Blum, B., Auker, C.R. and Carpenter, D.O. A head holder and stereotaxic device for the rattlesnake. *Brain Res. Bull.*, 3:271-274, 1978.

48. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Dopamine-induced muscle contractions and modulation of neuromuscular transmission in *Aplysia*. Brain Res., 157:167-172, 1978.
49. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Evidence for identified dopamine motor neurons to the gill of *Aplysia*. Neurosci. Lett., 10:275-280, 1978.
50. Kebabian, P.R., Kebabian, J.W. and Carpenter, D.O. Regulation of cyclic AMP in heart and gill of *Aplysia* by the putative neurotransmitters, dopamine and serotonin. Life Sci., 24:1757-1764, 1979.
51. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Res. Bull., 4:149-152, 1979.
52. Pellmar, T.C. and Carpenter, D.O. Voltage-dependent calcium current induced by serotonin. Nature, 277:483-484, 1979.
53. Ruben, P.C., Swann, J.W. and Carpenter, D.O. Neurotransmitter receptors on gill muscle fibers and the gill peripheral nerve plexus in *Aplysia*. Canad. J. Physiol. Pharmacol., 57:1088-1097, 1979.
54. Pellmar, T.C. and Carpenter, D.O. Serotonin induces a voltage-sensitive calcium current in neurons of *Aplysia californica*. J. Neurophysiol., 44:423-439, 1980.
55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthalmol., 89:641-646, 1980.
56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homogenates by high-performance liquid chromatography. Neurochem. Res., 5:1089-1096, 1980.
57. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Thyrotropin-releasing hormone has multiple actions in cortex. Brain Res., 194:244-248, 1980.
58. Meszler, R.M., Auker, C.R. and Carpenter, D.O. Fine structure and organization of the infrared receptor relay, the lateral descending nucleus of the trigeminal nerve in pit vipers. J. Comp. Neurol., 196:571-584, 1981.
59. Auker, C.R., Parver, L.M., Doyle, T. and Carpenter, D.O. Choroidal blood flow: I. Ocular tissue temperature as a measure of flow. Arch. Ophthalmol., 100:1323-1326, 1982.
60. Parver, L.M., Auker, C., Carpenter, D.O. and Doyle, T. Choroidal blood flow: II. Reflexive control in the monkey. Arch. Ophthalmol., 100:1327-1330, 1982.
61. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Lateral olfactory tract transmitter: Glutamate, aspartate or neither? Cell. Mol. Neurobiol., 1:115-120, 1981.
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PREVIOUS DEPOSITIONS AND TESTIMONY (past seven years):

Antonia Tolbert et al. vs. Monsanto Company, Pharmacia Corp., and Solutia Inc.,
deposed for the plaintiffs, 21-22 January 2003. Mark Englehart, Attorney 334-269-2343.

Aaron et al. vs. Chicago Housing Authority et al., deposed for the plaintiffs, 5-6 March
2003.

Kellum et al., vs. Kuhlman Corporation, deposed for the plaintiffs, 4 September 2004.
Douglas Mercier, Attorney, 601-914-2882.

Allgood et al. vs. General Motors Corporation, deposed for the plaintiffs, 8-10 December
2004. Brian J. Leinbach, Attorney. 310-552-3800.

Maggie T. Williams et al. vs. Kuhlman Corporation, deposed for the plaintiffs, 1
February and 25 February 2005. Douglas Mercier, Attorney, 601-914-2882.

Solutia Inc. et al., Debtors, vs. Monsanto Company and Pharmacia Corporation; deposed
for the plaintiffs, 12 September 2006. Samuel E. Stubbs, Attorney; 713-425-7345.

Charles W. Adams, et al., vs. Cooper Industries, Inc. et al., deposed for the plaintiffs, 28-
29 September 2006. Donna Keene Holt, Attorney. 865-212-3294.

Arthur D. Dyer et al. vs. Waste Management et al., deposed for the plaintiffs, 2
November 2006. Mark L. Thomsen, Attorney. Cannon & Dunply, Brookfield, WI 53008.

Clopten et al. vs. Monsanto, deposed for the plaintiffs, 31 January 2007. Robert Roden,
Attorney. 406-525-2665.

Marty Paulson et al. vs. Monsanto, deposed for the plaintiffs, 7 August 2007. Torger
Oaas, Attorney. 406-525-2665.

John Edward Martinez and Gladys Yolanda Martinez vs. Entergy Corporation et al.,
deposed for the plaintiffs, 16 April 2008. Julie Jacobs, Attorney. 504-566-1704.

Fannie Wayne et al. vs. Pharmacia Corporation, et al., deposed for the plaintiffs, 29
October 2008. John E. Norris, Attorney. 205-541-7759.

Fannie Wayne et al. vs. Pharmacia Corporation et al., testified for the plaintiffs, 31
March-1 April, 2009. John E. Norris, Attorney. 205-541-7759.

Clement Passariello, et al., vs. CL&P, et al.; William Korzon, et al., vs. CL&P, et al.;
Louis Gherlone et al., vs. CL&P, et al.; and William Ho, et al., vs. CL&P et al., deposed for the
plaintiffs, 13 April 2009. Benson A. Snaider, Attorney. 203-777-6426.

Before the Pennsylvania Public Utility Commission, docket No A-2009-2082652, et al.
Testified on behalf of the Saw Creek Estates Community Association, 2 September 2009. Paul
M. Schmidt, Attorney. 215-569-2800 x161.

James Alford et al. v. Kuhlman Corporation, et al., pending in the USDC, Southern
District of Mississippi, Deposed for plaintiffs, 20 August 2009. Shiela Bossier, Attorney. 601-
352-5450

Fannie Wayne et al. v. Pharmacia Corporation. Deposed for plaintiffs, 23 September
2009, Timothy C. Davis, Attorney. 205-327-9115.

Before the Minnesota Public Utilities Commission in the matter of the route permit
application by Great river energy and Xcel Energy for a 345 kV transmission line from
Brookings County, South Dakota to Hampton, Minnesota. Testified for plaintiffs, 16 December

2009. Paula Maccabee, Attorney. 651-775-7128.

Highland Lakes Estates et al.v. Republic Services of Florida et al., Deposed for the plaintiffs, 23 April 2010. John W. Frost II, Attorney. 863-533-8985.

Zina G. Bibb, et al. v Monsanto Company et al. Deposed for plaintiffs, 28 April 2010. W. Stuart Calwell, Attorney, 304-343-4323.

Highland Lakes Estates et al., v. Republic Services of Florida et al., Testified for the plaintiffs, 13 May 2010.

Nora Williams, et al., v. City of Jacksonville, et al. Deposed for the plaintiffs.15 July 2010. Samuel W. Wethern, Attorney.

Ronald Cybart et al., Michael Campanelli, and Donald and Theresa Shea, et al.v. CL&P. Deposed for the plaintiffs. 15 July 2011. Benson A. Snaider, Attorney.

Maria Snoops vs. Lyon Associates, Inc. and Insurance Co of the state of Pennsylvania. Deposed for the plaintiff, 1 November 2011. Matthew J. Witteman, Attorney. 415-363-3106.

John Edward Martinez and Gladys Yolanda Martinez v. Entergy Corporation, et al., Deposed for the plaintiff, 19 December 2011. J. Patrick Connick, Attorney. 504-347-4535.

Re: PZC Meeting 10/13/22 Public Comments Recieved

David Neel <david.neel@hotmail.com>

Tue 10/18/2022 3:02 PM

To: Kayla DiCristina <kayla@landofsky.org>

Be Advised: This email originated from outside Land of Sky

Kayla,

I thought so but wanted to make sure.

It's not my battle, but the doctor's lengthy court amendment, which none of the Board members will likely read (I did), appears to have been debunked by a recent, American Cancer Society article saying, **"there's no strong evidence that exposure to RF waves from cell phone towers causes any noticeable health effects."**

Jun 1, 2020

<https://www.google.com/amp/s/amp.cancer.org/healthy/cancer-causes/radiation-exposure/cellular-phone-towers.html>

Again, this isn't my battle, but his documentation seemed dated, so I just looked for more current data on Google.

Hope this helpful. Toss it if it's not needed. 😊

David

Sent from my iPhone

On Oct 18, 2022, at 1:21 PM, Kayla DiCristina <kayla@landofsky.org> wrote:

Hi David,

SO SORRY. This was not meant for the BOA.

Best,

Kayla DiCristina, AICP

Land of Sky Regional Council | Regional Planner | kayla@landofsky.org

Town of Montreat | Zoning Administrator | zoning@townofmontreat.org (inquiries are answered in the order they are received Tuesday through Thursday)

Message sent from the tiny pocket computer.

From: David Neel <david.neel@hotmail.com>
Sent: Tuesday, October 18, 2022 12:00:51 PM
To: Kayla DiCristina <kayla@landofsky.org>
Cc: Angela (Angie) Murphy Montreat Clerk <amurphy@townofmontreat.org>
Subject: Re: PZC Meeting 10/13/22 Public Comments Recieved

Be Advised: This email originated from outside Land of Sky

Kayla and Angie,

Why were the Board of Adjustments members copied on the email forwarded to the Planning and Zoning Board?

Was it a "friendly" FYI?

Thanks,
David

Sent from my iPhone

On Oct 18, 2022, at 9:52 AM, Kayla DiCristina <kayla@landofsky.org> wrote:

Good morning all,

The email below was transmitted to the Board before the meeting 10/13/22. I wanted to be sure you all received this information.

Best,

[Kayla DiCristina, AICP](#)

*(*For inquiries regarding the Town of Montreat, please see below)*

Regional Planner | Economic and Community Development

Land of Sky Regional Council

339 New Leicester Hwy., Suite 140 • Asheville, NC 28806

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From: Kayla DiCristina

Sent: Thursday, October 13, 2022 10:13 AM

To: crawfordjohna@aol.com; danielbluedean@gmail.com; johndora@charter.net; julie19923@gmail.com; lhjohnson62@gmail.com; sfstansill58@gmail.com; wdbmountainliving@gmail.com; wscheu@rtlaw.com

Cc: bblackburn@townofmontreat.org; Angela Murphy <amurphy@townofmontreat.org>

Subject: PZC Meeting 10/13/22 Public Comments Recieved

Good morning Board Members,

Attached is a message I received moments ago from Angela Murphy, Town Clerk. This is an email from Jane Warner regarding the Wireless Communications Ordinance. Ms. Warner's email is **not** regarding the proposed text amendments, but rather her concerns about the effect of wireless communication equipment on health. I have printed copies of her email for the Board to review at the meeting (since I received this email only a few minutes ago and not in time to make the board packet) but did not print the attached study as it is over 40 pages. I have attached digital copies of both the email and study to this message.

Best,

Kayla DiCristina, AICP

*(*For inquiries regarding the Town of Montreat, please see below)*

Regional Planner | Economic and Community Development

Land of Sky Regional Council

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Wireless communications regulation

ltlredwagon <ltlredwagon@mailfence.com>

Mon 11/14/2022 8:56 PM

To: thelms@townofmontreat.org <thelms@townofmontreat.org>

Cc: Commissioner Kitty Fouche <kfouche@townofmontreat.org>; Commissioner Mason Blake <mblake@townofmontreat.org>; Mayor Pro-Tem Kent Otto <kotto@townofmontreat.org>; Commissioner Jane Alexander <jalexander@townofmontreat.org>; Commissioner Tom Widmer <twidmer@townofmontreat.org>; Julie Schell <julie19923@gmail.com>; wscheu@rtlaw.com <wscheu@rtlaw.com>; Kayla DiCristina <kayla@landofsky.org>; sfstansill58@gmail.com <sfstansill58@gmail.com>; Angela Murphy <amurphy@townofmontreat.org>

📎 7 attachments (10 MB)

Are Contaminants Silencing Our Genes - Scientific American.pdf; Carpenter EMF.pdf; ICBE-EMF.pdf; EMF Genetic Effects.pdf; Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest.pdf; Oncology Letters.pdf; Why Most Published Research Findings Are False - PMC.pdf;

You don't often get email from ltlredwagon@mailfence.com. [Learn why this is important](#)

Be Advised: This email originated from outside Land of Sky

Dear Mayor and Commissioners of Montreat,

My wife, Jane, recently wrote to you regarding regulations governing wireless communication installations in Montreat. My understanding is that there are no equipment installations planned for the immediate future and that this matter may be examined again in the new year. Perhaps this email can be set aside for review at that time.

Whenever you do take up this matter, I would like to offer the following data for your consideration. I support the recommendation of my wife that Montreat take a more proactive stance to protect Montreaters from electromagnetic frequency radiation (EMF).

Here is the fundamental problem as I see it. Some may disagree, but I would say that history reveals humans to be fantastically creative and capable as problem solvers, but often expedient, even careless at times, in getting things done. I'll provide a few examples below.

In seeking to understand a topic it is sometimes helpful to make comparisons to something which is more familiar. EMF radiation, as a global phenomenon, is a very recent concern. It was only 30 years ago that reduced cell phone size enormously increased the popularity of cell phone usage. Complete internet service on the mobile web has only been available for a little over 20 years – mere seconds when it comes to biological research.

But the phenomenon is not without precedent. A similar situation existed in the mid-20th century with regard to skyrocketing chemical use in agriculture and industry. (In the 1980s I spent about 8 years writing for various trade journals – agriculture, industry, police, firefighting – on human exposure to toxic chemicals; I'm no expert, but I know a bit about the subject.) At that time the general population was being exposed to thousands of toxic chemicals and there were no studies which had examined the effects of prolonged exposure to such chemicals over a lifetime (particularly at low levels) and, through placental blood transfer and other factors, over many generations. The renowned environmental scientist and pathologist, Dr. Rene Dubos of the Harvard Medical School, sounded the alarm in 1968 in the journal *Environmental Scientist*, writing, "The greatest danger of pollution may well be that we shall tolerate levels of it so low as to have no acute nuisance value, but sufficiently high, nevertheless, to cause delayed disease and spoil the quality of life." Dubos is credited with popularizing the environmental maxim, "Think globally, act locally".

Dubos words were prophetic. The US Environmental Protection Agency's National Human Adipose Tissue Survey (NHATS) has found dozens of carcinogenic chemicals (herbicides, pesticides, industrial chemicals such as PCBs) at low levels in virtually every human ever tested. According to a report in the journal, *Environment International*, these chemicals "concentrate in fatty

tissues and bioaccumulate as they move up the food chain; travel long distances in global air and water currents; and have been linked with serious health effects in humans, even at low exposures" (*Environment International*, Vol. 39, Issue 1, Feb., 2012).

We are now in the middle of a decades-long human experiment with low-level chemical exposure on a global scale, the effects of which we simply have not been able to fully study. There are so many substances which were tested and "proven" safe at given levels by top scientists decades ago. Now we know more. If you will allow me to give just one example. This year the EPA reported that PFAS (per- and poly-fluoroalkyl substances), carcinogenic chemicals in use since the 1940s for nonstick cookware, fabrics and flame-retardant equipment, are far more dangerous than previously known. The EPA has set new levels which are 3,000 to 17,000 times lower than previous standards (<https://www.washingtonpost.com/climate-environment/2022/06/15/epa-pfas-forever-chemicals/>). PFAS are in the drinking water of a majority of Americans and in the blood of almost everyone, but one component of the environmental and internal chemical "soup" we now live with. It's worth pausing to consider this: the adverse effects of a chemical in use for 80 years are only now being understood. (Teflon pans – never caused me any problems, right?)

There are also concerns that "chemicals in our environment and in our food can alter genes, leaving people vulnerable to a variety of diseases and disorders." ("Are Contaminants Silencing our Genes?", *Scientific American*, Aug 3., 2009.)

It is my opinion that a very similar global experiment is now occurring with electromagnetic frequency radiation (EMF). My wife has forwarded to you the extensive review of scientific literature on this subject which David Carpenter, M.D. provided when the Portland Public Schools were considering the effects of EMF on children. Dr. Carpenter, a graduate of the Harvard Medical School, is the Director of the Institute for Health and the Environment at the University of Albany. Dr. Carpenter received no compensation for his testimony.

Dr. Carpenter concluded:

"Chronic exposure to PM MW [pulse-modulated microwave] radiation harms every individual in a population in some ways, even if these are not always detectable by the individual or consciously attributed to the responsible RF/MW [radiofrequency/microwave] radiation sources. This Agent injures some individuals into a condition in which symptoms will be more easily retriggered with subsequent exposure."

This year the International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF) published a peer-reviewed paper entitled: "Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G" (*Environmental Health* (2022) 21:92 <https://doi.org/10.1186/s12940-022-00900-9>). My wife has already summarized the findings, as follows:

- **The limits set for radiofrequency radiation established by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) and the Federal Communications Commission (FCC) are based upon invalid assumptions and outdated science; they are not protective of human health.**
- **That there be an independent assessment of the dangers of radio frequency radiation based on scientific evidence from peer-reviewed studies conducted over the past 25 years. They are seeking health standards for workers and the public.**
- **That the public be informed of the health risks of EMF and encouraged to do everything they can to minimize exposures, especially for children, pregnant women and people who are hypersensitive.**
- **That there be an immediate moratorium on further rollout of 5G wireless technology until safety is actually demonstrated.**

As with chemical pollutants, there is also concern over the genetic effects of EMF. The journal, *Electromagnetic Biology and Medicine* (Electromag Biol Med. 2021 Apr 3;40(2):264-273. doi: 10.1080/15368378.2021.1881866. Epub 2021 Feb 4) reported last year:

"Genetic effects of EMF depend on various factors, including field parameters and characteristics (frequency, intensity, wave-shape), cell type, and exposure duration. The types of gene expression affected (e.g., genes involved in cell cycle arrest, apoptosis and stress responses, heat-shock proteins) are consistent with the findings that EMF causes genetic damages. Many studies reported effects in cells and animals after exposure to EMF at intensities similar to those in the public and occupational environments. The mechanisms by which effects are induced by EMF are basically unknown."

You may also wish to review a study published in the journal, *Oncology Letters*, in 2020 (*Oncology Letters*, 2020 October; 20(4): 15), entitled, "Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest". The researchers wrote:

"The fifth generation, 5G, of radio frequency 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, the 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.”

With regard to this last report, I would particularly like to draw your attention to two important peer-reviewed studies. In 2016, 40% of scientists surveyed by the journal, *Nature* (1500 scientists) believed that fraud was always or often a factor in research. This is the scientific community critiquing itself. An astonishing 70% cited the bias of “selective reporting”, the suppression of undesirable facts and findings (*Nature*, Vol. 533, pages 452–454, 2016). John Ioannidis, M.D., of the Stanford University School of Medicine reached a similar conclusion. Ioannidis is an internationally recognized expert in the study of scientific research. In 2005 he published a paper entitled, “Why most published research findings are false” (*PLoS Med.* 2005 Aug;2(8):e124. doi:10.1371/journal.pmed.0020124). Ioannidis reported:

“The greater the financial and other interests and prejudices in a scientific field, the less likely the research findings are to be true. Conflicts of interest are very common in biomedical research, and typically they are inadequately and sparsely reported.”

The question for Montreat is whether or not it will, by default, side with industry and continue the EMF experiment, or whether it will act now to reduce the exposure of Montreaters to EMF. Will there be, to quote Dubos, very little in the way of “acute nuisance value” but eventual “delayed disease”? If you research this field you will likely encounter those who will tell you that there are no studies which have proven long-term adverse health effects on populations exposed to EMF. Of course, they are absolutely right! But those who wish to act responsibly must then ask themselves the *other* question: **While most people do not experience, or do not notice, any acute effects, are there any studies which prove that there are NO long-term adverse health effects from prolonged exposure to EMF radiation?** Who has the burden to answer that question? Industry, yes; medicine, yes. But you and I as well.

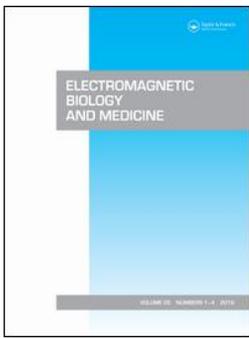
Look at our oceans and rivers, our air, our soil, our food supply. It’s hard to argue the charge that humans tend to be expedient: push ahead now, “get it done” – ask questions later. Are we making the same mistake with EMF?

I believe that there is more than enough evidence in the scientific research literature to support a decision by the government of Montreat to place a moratorium on all 5G installations in Montreat until their apparent dangers are fully researched. I hope the government of Montreat will place the safety of its citizens above all other concerns.

Sincerely,
Bob Warner
346 Chapman Road

ATTACHMENTS:

1. “Are Contaminants Silencing our Genes?”, *Scientific American*, Aug 3., 2009
2. David O. Carpenter, M.D., Declaration on EMF United States District Court, Portland, Oregon, 2011
3. “Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G” (*Environmental Health* (2022) 21:92 <https://doi.org/10.1186/s12940-022-00900-9>).
4. “Genetic effects of non-ionizing electromagnetic fields,” *Electromagnetic Biology and Medicine*, Vol. 40, 2021 - Issue 2
5. “Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest”, *Oncology Letters*, 2020 October; 20(4):15)
6. “1500 scientists lift the lid on reproducibility,” *Nature* 533, 452-454 (2016)
7. “Why Most Published Research Findings Are False,” John Ioannidis, M.D., *PLoS Med.* 2005 Aug; 2(8): e124



Genetic effects of non-ionizing electromagnetic fields

Henry Lai

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REVIEW

Genetic effects of non-ionizing electromagnetic fields

Henry Lai

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ABSTRACT

This is a review of the research on the genetic effects of non-ionizing electromagnetic field (EMF), mainly on radiofrequency radiation (RFR) and static and extremely low frequency EMF (ELF-EMF). The majority of the studies are on genotoxicity (e.g., DNA damage, chromatin conformation changes, etc.) and gene expression. Genetic effects of EMF depend on various factors, including field parameters and characteristics (frequency, intensity, wave-shape), cell type, and exposure duration. The types of gene expression affected (e.g., genes involved in cell cycle arrest, apoptosis and stress responses, heat-shock proteins) are consistent with the findings that EMF causes genetic damages. Many studies reported effects in cells and animals after exposure to EMF at intensities similar to those in the public and occupational environments. The mechanisms by which effects are induced by EMF are basically unknown. Involvement of free radicals is a likely possibility. EMF also interacts synergistically with different entities on genetic functions. Interactions, particularly with chemotherapeutic compounds, raise the possibility of using EMF as an adjuvant for cancer treatment to increase the efficacy and decrease side effects of traditional chemotherapeutic drugs. Other data, such as adaptive effects and mitotic spindle aberrations after EMF exposure, further support the notion that EMF causes genetic effects in living organisms.

ARTICLE HISTORY

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KEYWORDS

Radiofrequency radiation;
static/extremely low
frequency EMF; genetic
effects; genotoxicity; gene
expression

Introduction

This is a review on studies on the genetic effects of non-ionizing electromagnetic fields (EMF). We will concentrate on two parts of the EMF spectrum which are common in our environment: static and extremely low-frequency electromagnetic fields (ELF-EMF) and radio-frequency radiation (RFR).

Studies are summarized in Supplements 1 (RFR) and 2 (static/ELF-EMF). Basically, there are two types of studies: genetic damages and gene expression. The research covers a wide area of biological systems: both in vitro and in vivo involving many animal and cell models, and various exposure conditions. First, a few words have to be said on the exposure set-ups used in these studies. It is relatively easy to set up a reliable exposure system for static and ELF-EMF. Most exposure systems used these studies are generally satisfactory. However, it is difficult to set up good exposure systems for RFR studies. In my opinion, most set-ups are relatively satisfactory, considering that there is no perfect guideline on what is a good system. However, preferably, incident power density and specific absorption rate should be provided in each study. These are generally lacking when telecommunication devices, such as cellular phones, are used in a study. It becomes difficult to

compare the results of these studies with other studies using exposure systems. It is not totally without merit to use these devices for studies. If properly set up, these devices provide more realistic exposure parameters. A general problem is that some researchers generally showed ignorance on the independent variable, i.e., EMF, that they worked on.

Regarding biological measurements, with few exceptions, the researcher are generally knowledgeable in the methodology used. However, there are studies that showed that the researchers are not familiar with the methodology that they used in their studies. An example is the use of the “Comet assay” to determine DNA strand breaks. 31% of the studies listed in Supplements 1 and 2 used the “Comet assay”. A few words have to be said on it. Different versions of the assay have been developed. These versions have different detection sensitivities and can be used to measure different aspects of DNA strand breaks. A comparison of data from experiments using different versions of the assay may be misleading. Another concern is that most of the ‘comet assay’ studies were carried out by experimenters who had no prior experience on the assay. My experience with the ‘Comet assay’ is that it is a very sensitive assay and requires great care in performing. Thus, different detection sensitivities could result from different

experimenters, even following the same procedures. One way to solve this experimental variation problem is for each researcher or laboratory to report their sensitivity of the ‘Comet assay’, e.g., threshold of detecting strand breaks in human lymphocytes exposed to x-rays. This information is generally not available from the EMF-genotoxicity studies. However, in one incidence, an incredibly high sensitivity was even reported (Malyapa et al., 1998), suggesting the inexperience of the researchers on the assay.

Supplements 1 and 2 show that the majority of studies reported genetic effects of EMF (66% for RFR and 79% for static/ELF-EMF). Thus, it is safe to conclude that genotoxic effects of EMF have been reported. The most common effects found are: DNA strand breaks, micronucleus formation, and chromosomal structural changes. There are not many studies on mutation. Thus, it is not known whether these genotoxic effects transform into mutation and involved in carcinogenesis. Interestingly, available data do not suggest mutagenic effect after RFR exposure (Chang et al., 2005; Meltz et al., 1990; Ono et al., 2004; Takahashi et al., 2002); whereas most static/ELF-EMF studies (Chahal et al., 1993; Mairs et al., 2007; Miyakoshi, 1997; Miyakoshi et al., 1998, 1996; Potenza et al., 2004; Wilson et al., 2015) suggested some mutagenic effects. Another interesting speculation is that ELF EMF acts as a promoter of cancer in the presence of an initiator by modulation of signaling pathways involved free radicals and apoptosis (Lacy-Hulbert et al., 1998). Such a possibility has not been well investigated.

There are similarly many studies that showed changes in gene expression after EMF exposure (Supplement 3). Changes in expression of many different genes have been reported. Studies in gene expression by static/ELF-EMF are far more diversified than those of RFR. The most interesting results are the expression of genes related to stress response both in vitro and in vivo in plants and animals. Another important finding is the expression of heat shock proteins, particularly HSP70, which is an important protein involving in protein misfolding and protecting cells from environmental stress.

The data point to four areas of interest: involvement of free radicals, effects at low-intensity of exposure, contributions of exposure parameters and biological system being studied, and interaction with other entities. Let us look at each of these four topics.

Involvement of free radicals (Citations of references in italic in this section are in Supplements 1 and 2)

Effects of EMF on cellular free radical processes have been reported in many experiments (cf. Lai, 2019; Yakymenko et al., 2016). It is conceivable that an

increase in free radicals in cells could cause macromolecular damages including DNA. There are many reports on involvements of free radicals in genetic processes, including both reactive oxygen species and reactive nitrogen species: **RFR** – Agarwal et al., 2009; Alkis et al., 2019a, b, 2021; Bektas et al., 2020; Bourdineaud et al., 2017; Burlaka et al., 2013; De Jullis et al., 2009; Duan et al., 2015; Gajski and Garaj-Vrhovac 2009; Garaj-Vrhovac et al., 2009, 2011; Guler et al., 2010; Gürler et al., 2014; Houston et al., 2019; Kesari et al., 2011, 2014; Khalil et al., 2012; Kumar et al., 2010; Lai and Singh, 1997; Li et al. 2018; Liu et al., 2013a, b; Luukkonen et al., 2009; Manta et al. 2017; Magha et al., 2015b; Meena et al. 2014; Millenbaugh et al., 2008; Odaci et al., 2016; Pandey et al., 2017; Pandey and Giri, 2018; Qin et al., 2019; Sahin et al., 2016; Shahin et al., 2013, 2019; Sharma and Shukla, 2020; Sokolovic et al., 2015; Sun et al. 2017; Tkalec et al., 2013; Vafaei et al. 2020; Varghese et al., 2018; Veerachari and Vasan, 2012; Vilić et al., 2017; Wang et al., 2015; Wu et al., 2008; Xu et al., 2010; Yakymenko et al., 2018; Yao et al., 2008; Zong et al. 2015; Zothansiyama et al., 2017; **Static and ELF EMF** – Alcaraz et al., 2014; Amara et al., 2007b; Ashta et al., 2020; Hosseinabadi et al., 2020; Berteau et al., 2015; Butdak et al., 2012; Consales et al., 2018; Dong et al. 2019; Jajte et al., 2001; Jouni et al., 2012; Kimsa-Dudek et al. 2018; Kindzelskii and Petty, 2000; Lai and Singh, 1997b, 2004; Li et al., 2001; Luukkonen et al., 2014; Rageh et al., 2012; Shokrollahi et al., 2018; Solek et al., 2017; Wang et al., 2020; Wolf et al., 2005; Yin et al., 2016; Yokus et al., 2008; Yuan et al., 2020; Zhang et al., 2016. Brief descriptions of these reports are in Supplements 1 and 2. However, changes in cellular free radical and genetic processes do not imply a cause–effect relationship. A convincing argument on direct involvement of free radicals on EMF-induced genetic changes comes from data showing that the effects could be blocked by free radical scavengers (e.g., antioxidants) e.g., see Lai and Singh (1997; 2004). The free radicals involved probably include both reactive oxidative species (ROS) and reactive nitrogen species (RNS) (Lai and Singh, 2004). RNS (e.g., nitric oxide) have longer mean free path than ROS (e.g., hydroxyl radical) and could cause more widespread cellular molecular damages. Nitric oxide can further enhance iron-mediated free radical formation via its effects on iron metabolism and release of iron from ferritin (Reif and Simmons 1990; Richardson and Ponka 1997) that generates ROS via the Fenton reaction. Nitric oxide can either be mutagenic or cytotoxic. It is mutagenic when the intracellular level of reduced glutathione is low, but cytotoxic (leading to

apoptosis and inhibition of tumor growth) in a thiol-rich environment that favors the formations of toxic nitrosothiols (Felley-Bosco 1998). These situations could occur under EMF exposure.

The mechanisms on how EMF affects free radicals in cells are not known. There are various speculations. Readers may be interested to take a look at these publications: Barnes and Greenebaum (2015); Binhi and Prato (2017); Davila et al. (2005); Dodson et al. (2013); Hore (2019); Hore and Mouritsen (2016); Kirschvink et al. (2001); Landler and Keays (2018); Sheppard et al. (2017); Sherrard et al. (2018); and Sisakht et al. (2020).

Furthermore, it has to be pointed out that EMF-induced genetic effects have been observed without free radical changes (Alcaraz et al., 2014; Ferreira et al., 2006; Furtado-Filho et al., 2014) and free radical changes without genetic effects (Frahm et al., 2006; Senturk et al., 2019; Tiwari et al., 2015; Tomruk et al., 2010) have also been reported. This may imply that mechanisms other than free radicals are involved,

Effects at low exposure intensities

There are many reports of genetic effects induced by low intensities of EMF. The studies are listed in Supplement 4. This is an important topic to consider since living organisms are being constantly exposed to low levels of EMF in the occupational and public environments. This is particularly true for ELF-EMF, since intensities of ELF-EMF in the environment are in microtesla (μT) levels, even exposure to fields from electrical appliances rarely exceed 10 microtesla (i.e., 0.01 mT). However, most laboratory cell and animal studies in ELF-EMF used fields in the millitesla (mT) level.

A survey of level of RFR in the environment of various countries (Amoako et al., 2009; Aris et al., 2020; Bhatt et al., 2016; Dhama, 2012; Dode et al., 2011; Estenberg and Augustsson, 2014; Firlarer et al., 2003; Frei et al., 2009; Hardell et al., 2016, 2017; Henderson and Bangay, 2006; Joseph et al., 2008, 2010; Kim and Park, 2010; Kurnaz and Aygun, 2020; Lahham and Hammash, 2012; Lahham et al., 2015, 2017; Sagar et al., 2018; Tell and Kavet, 2014; Thuroczy et al., 2006; Urbinello et al., 2014; Viel et al., 2009; Waldmann-Selsam et al., 2016) gave a mean power density level of 0.00259 mW/cm^2 and median of 0.000545 mW/cm^2 . Reports (Abuasbi et al. 2018; Al-Badi, 2012; AL-rajhi, 2014; Eskelinen et al., 2002; Ilonen et al., 2008; Lindgren et al., 2001; Rösli et al., 2011) on the levels of magnetic fields in the human environment came up with a mean level of 0.0036 mT and median level of 0.00062 mT. Much higher exposure levels could be found in occupational situations. Operators and technicians in a power plant could be exposed to 0.0126 mT, whereas the

magnetic field level in the vicinity of a power transmission line could be as high as 0.0482 mT (Hosseinsbadi et al., 2020).

Besides genetic effects, other physiological processes have also been reported to be affected by low-intensity EMFs, e.g., **RFR**: retarded development of frog (Balmori, 2010; 88.5–1873.6 MHz cell phone base station emission; $0.00859\text{--}0.00325 \text{ mW/cm}^2$); slowing of circadian rhythm in cockroach (Bartos et al., 2019; broadband RF noise; 0.000429 mT); changes in electrical activities in rat sciatic nerve (Comelekoglu et al., 2018, 1800-MHz RFR; 0.00421 W/kg); delayed growth in rose (Grémiaux et al., 2016; 900 MHz RFR; 0.00072 W/kg); retarded memory in rat (Nittby et al., 2008; 900 MHz GSM signal; 0.0006 W/kg); adrenal gland stimulation in rat (Perov et al., 2019; 171 MHz RFR; 0.0006 W/kg); human blood mononucleus cells showed higher immunological activates (Szymanski et al., 2020; 0.024 W/kg) (see also the Table in Lai, 2018 on low-intensity effect on neurological functions); **static and ELF-EMF**: decreased number of living and quality of movement of sperms of mouse (de Bruyn and de Jager, 2010; 50-Hz MF $0.0005\text{--}0.077 \text{ mT}$) and free radicals (see Table 1: „Free radical effects observed at low intensities of static and ELF-EMF” in Lai, 2019, effects have been observed with exposure to a 50 Hz MF of 0.0005 mT). In addition, mechanisms have evolved for organisms to detect very low levels of static EMF, e.g., 26 nT (i.e., 0.000026 mT) in honey bees (Kirschvink et al., 1992); 20 microV/cm in platypus (Manger and Pettigrew, 1996); and 2–3 nT in songbird (Pakhomov et al., 2017). These capabilities of detecting very low-intensity static/ELF EMF fields is actually not surprising because they are results of evolution over millions of years to enable the survival of the species. On the other hand, these functions are much vulnerable to disturbance from recent man-made EMF. However, it is a little surprising that RFR at very low intensity could also cause biological effect. The RFR studied are mostly man-made and have only existed in the environment in the last several decades. This points to a possibility that EMFs (RFR and static/ELF EMF), in general, act on some common unknown basic biological mechanisms.

Interaction effects (citations of references in italic in this section are in supplements 1 and 2)

Another important observation of the studies is that EMF can interact with other entities and synergistically cause genetic effects. These entities include:

RFR: Chemical mutagens (Baohong et al., 2005); ultraviolet ray (Baohong et al., 2007); 17- β -estradiol (Cervellati et al., 2013); bee venom (Gajski and Garaj-Vrhovac, 2009);

garlic (Gurler et al., 2014); γ -radiation (He et al., 2017; Ji et al., 2016; Jiang et al., 2013); clastogens (Kim et al., 2008); incoherent electromagnetic noise (Lai and Singh, 2005; Wu et al., 2008; Yao et al., 2008); lipopolysaccharide (Lameth et al., 2020; Zuo et al., 2015); mitomycin C (Maes et al., 1996; Sannino et al., 2011, 2017; Zeni et al., 2012a; Zhang et al., 2002); x-rays (Manti et al., 2008; Gapeyev et al., 2014; Sannino et al., 2014); aphidicolin (Tiwari et al., 2008); picrotoxin (López-Martín et al., 2009); bleomycin (Koyama et al., 2003; Zong et al., 2015) and doxorubicin (Zhijian et al., 2010).

Static – and ELF-EMF: Zinc (Amara et al., 2007); Tremozolomide (Ashta et al., 2020); Cisplatin (Buldak et al., 2012; El-Bialy et al., 2013; Chen et al., 2010; Mahmoudinasab and Saadat, 2018a; Sanie-Jahromi and Saadat, 2017; Sanie-Jahromi et al., 2016); Bleomycin (Cho et al., 2007; Sanie-Jahromi and Saadat, 2017); Gadolinium (Cho et al., 2014); alkaline-ph (Fan et al., 2018); natural radioactivity in soil (Jouni et al., 2012); sodium fluoride (Kimsa-Dudek et al., 2018, 2020); gamma radiation (Arruda-Neto et al., 2009; Kubinyi et al., 2010; Lagroye and Poney, 1997; Mairs et al., 2007); hydrogen peroxide and methyl methane sulfonate (Koyama et al., 2008); menadione (Luukkunan et al., 2011, 2014, 2017; Markkanen et al., 2008), morphine (Mahmoudinasab and Saadat, 2018b); X-ray (Miyakoshi et al., 1996b; 1999, 2000; Teodori et al., 2014; Udroui et al., 2015); Xenobiotics (Moretti et al., 2005); lipopolysaccharide (Nakayama et al., 2016); heat (Robison et al., 2002); N-methyl-N'-nitro-N-nitrosoguanidine, 4-nitroquinoline N-oxide, benzene, 1,4-benzenediol, 1,2,4-benzenetriol (Scassellati Sforzolini et al., 2004; Villarini et al., 2000); mineral oil (Skyberg et al., 2001); Paclitaxel (Sun et al., 2012); IR (Yoon et al., 2014); FeCl₂ (Zmyslony et al., 2000); UV (Zmyslony et al., 2000).

Most of the compounds that have been shown to interact with EMF are mutagens. This is important because in real-life situations, a person is usually exposed simultaneously to EMF and many different environmental factors, including mutagens. On the other hand, some of these entities are drugs used in cancer chemotherapy. EMF can possibly be used as an adjuvant in chemotherapy to enhance the anticancer efficacy of these drugs and decrease their side-effects. Thus, synergism of these entities with EMF should be further studied.

However, it is important to point out that are reports (listed below) that showed no significant interaction effects.

RFR: Mitomycin C (Hansteen et al., 2009; Kerbacher et al., 1990; Maes et al., 1997, 2000, 2001, 2006; Zhijian et al., 2009); Adrimycin (Kerbacher et al., 1990); x-ray

(Maes et al., 2000; Stronati et al., 2006); proflavin (Meltz et al., 1990); 3-Chloro-4-(dichloromethyl)-5-Hydroxy-2 (5 H)-furanone (an environmental mutagen) (Sannino et al., 2009; Verschaeve et al., 2006).

Static – and ELF-EMF: Methylmethane sulfonate, chromate (Cantoni et al., 1996); UV (Cantoni et al., 1996; Mizuno et al., 2014); ionizing radiation, H₂O₂, mitomycin C (Jin et al., 2011, 2014); IR and H₂O₂ (Jin et al., 2015; Yoon et al., 2014); chemical mutagens (Verschaeve et al., 2011); heat (Williams et al., 2006).

Effects of waveform

Two other important findings of recent studies are that the effects of EMF are waveform specific and cell-type specific (Supplement 5). These findings underscore the complicity of interaction of EMF with biological tissues and may partially explain why effects were observed in some studies and not others. It is essential to understand why and how certain wave-characteristics of an EMF are more effective than other characteristics in causing biological effects, and why certain types of cells are more susceptible to the effect of EMF? The fact that “there are different biological effects elicited by different EMF wave-characteristics” is a critical proof for the existence of non-thermal effects.

Wave-form dependency is one of the major puzzles of Bioelectromagnetics research. In the 1970s, research in the laboratories of Ross Adey (Bawin et al., 1975; 1978) and Carl Blackman (Blackman et al., 1979) showed the importance of modulations on the EMF-carrier frequency on calcium efflux from cells. Other biological effects of EMF also showed wave-form dependency, e.g., see discussion in Lai (2018) on neurological effects of RFR. And, research presented here also showed similar dependency in EMF-induced genetic effects. So far, there has not been a credible unifying explanation for the “wave-form dependency effect”.

Regarding cell-type specificity, one can speculate that:

1. Cells that are metabolic active are more susceptible to EMF effects with an increase in generation of free radical in the mitochondria;
2. Cells that have higher anti-oxidative activities are less susceptible;
3. Transitional elements, e.g., iron, may play a role in the effect via the Fenton reaction (see Lai, 2019). Brain cells contain a relatively high concentration of free iron, particularly intercalated in the DNA molecules, and are more susceptible;
4. Cell cycle arrests are common in cells exposed to EMF. It may be a response to repair genetic damages caused by EMF. If damage could not be repaired, cell death occurs, particularly via apoptosis, which is a common outcome after EMF exposure. These effects are consistent with the gene expression

studies, showing activation of genes involved in both cell death and repair. 5. If genetic damaged cells are allowed to survive, cancer may occur. However, if they die, the risk of cancer would actually be reduced. But, other detrimental health outcomes may occur, e.g., death of brain cells could lead to neurodegenerative diseases. Increased incidences of degenerative diseases (including Alzheimer's disease, amyotrophic lateral sclerosis, dementia, and motor dysfunctions) after EMF exposure, particularly under occupational conditions, have been reported (Gervasi et al. 2019; Gunnarsson and Bodin 2018, 2019; Huss et al. 2018; Koeman et al. 2017; Jalilian et al. 2018; Pedersen et al. 2017; Sorahan and Mohammed 2014).

Discussion

The main question is whether EMF exposure could cause genetic effects? It is pertinent here to quote a recent statement made by two prominent bioelectromagnetic researchers (Barnes and Greenebaum, 2020): "The evidence that weak radiofrequency (RF) and low-frequency fields can modify human health is still less strong, but the experiments supporting both conclusions are too numerous to be uniformly written off as a group due to poor technique, poor dosimetry, or lack of blinding in some cases, or other good laboratory practices." All in all, in the studies reviewed in Supplements 1 and 2, approximately 70% of them showed effects. One could say that EMF exposure can lead to genetic changes. Some genetic damages could eventually lead to detrimental health effects. However, the mechanisms remain to be uncovered. But, knowing the mechanism is not necessary to accept that the data are valid. It is also a general criticism that most EMF studies cannot be replicated. I think it is a conceptual and factual misstatement. Replication is also not a necessary and sufficient condition to believe that certain data are true. Scientific studies are hardly replicated. Rational funders do not generally fund replications. All scientists should know that it is very difficult to replicate exactly an experiment carried out by another lab. This is particularly true when the effects of EMF depend on many unknown factors. By the way, not many replication experiments have been carried out in EMF genetic-effect research to justify the statement that "data from EMF are not replicable". In some cases, the experimenters deliberately changed the procedures of an experiment that they were supposed to be replicating and claimed that their experiment was a replication, for example, compare the experimental procedures of Lai and Singh (1995) and Malyapa et al. (1998).

To prove an effect, one should look for consistency in data. Genetic damage studies have shown similar effects with different set-up and in various biological systems. And, the gene expression results (Supplement 3) also support the studies on genetic damages. Expression of genes related to cell differentiation and growth, apoptosis, free radical activity, DNA repair, and heat-shock proteins have been reported. These changes could be consequences of EMF-induced genetic damages. In addition, other effects of EMF, such as mitotic-spindle disruption (De Amicis et al., 2015; Hintzsche et al., 2011; Li et al., 2013; Schrader et al., 2011, 2008; Tkalec et al., 2009) and "adaptive" effects, i.e., the ability of concomitant exposure of RFR to decrease the genotoxic effects of other agents, such as ionizing radiation (He et al., 2017; Ji et al., 2016; Jiang et al., 2012, 2013; Sannino et al., 2014, 2017, 2011; Sun et al., 2016; Zeni et al., 2012; Zong et al., 2015) also support the notion that EMF exposure could affect genetic processes in cells. In conclusion, there are enough reasons to believe that genetic effects of EMF are real and possible.

During cell phone use, a relatively constant mass of tissue in the brain is exposed to the radiation at relatively high intensity (peak specific absorption rate (SAR) of 4–8 W/kg). Many papers have reported genetic effect/DNA damage at much lower SAR (or power density) (see Supplement 4). This questions the wisdom of the several exposure standard-setting organizations in using the obsolete data of 4 W/kg (whole-body averaged SAR) as the threshold for exposure-standard setting. Furthermore, since critical genetic mutations in one single cell are sufficient to lead to cancer and there are millions of cells in a gram of tissue, it is inconceivable that some standards have changed the SAR from averaged over 1 gm to 10 gm of tissue. (The limit of localized tissue exposure has been changed from 1.6 W/kg averaged over 1 gm of tissue to 2 W/kg over 10 gm of tissue. Since distribution of radiofrequency energy is non-homogenous inside tissues, this change allows a higher peak level of exposure.) What actually needed is a better refinement of SAR calculation to identify 'peak values' of SAR inside the brain.

Any effect of EMF has to depend on the energy absorbed by a biological entity and on how the energy is delivered in space and time. Aside from influences that are not directly related to experimentation (Huss et al., 2007), many factors could influence the outcome of an experiment in bioelectromagnetics research. Frequency, intensity, exposure duration, and the number of exposure episodes can affect the response, and

these factors can interact with each other to produce different effects. In addition, in order to understand the biological consequences of EMF exposure, one must know whether the effect is cumulative, whether compensatory responses result, and when homeostasis will break down. A drawback in the interpretation and understanding of experimental data from bioelectromagnetic research is that there is no general accepted mechanism on how EMF affects biological systems. Since the energy level is not sufficient to cause direct breakage of chemical bonds within molecules, the effects are probably indirect and secondary to other induced chemical changes in the cell. The mechanisms by which EMF causes genetic effects are unknown. This author suspects that biological effects of EMF exposure are caused by multiple inter-dependent biological mechanisms.

Disclosure of Interest

The author declares no conflict of interest

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Supplement 1

**Genetic effects of radiofrequency electromagnetic radiation (*study with no effect observed)
Study reported effect =237 (66%); study reported no effect = 124 (34%) (Literature up to
January 2021).**

	Exposure conditions	Results
*Agarwal et al. (2009)	Human semen sample to cell phone radiation in talk mode for 1 h	No significant DNA damage, increase in reactive oxygen species; decrease in sperm motility and viability.
Aitken et al. (2005)	Mice to 900-MHz RFR for 7 days at 12 h/day; SAR 0.09 W/kg	Significant damage to Mitochondrial genome and nuclear β -globin locus in epididymal spermatozoa.
Akdag et al. (2016)	Male Wistar-Albino rats to 2400 MHz RFR from a Wi-Fi signal generator for a year; SAR 0.000141 (min)- 0.007127 (max) W/kg	No significant change in DNA single strand breaks (Comet assay) in brain, kidney, liver, and skin tissues, increased in testes.
Akdag et al. (2018)	Men who used cell phone for different durations per day; peak head SAR 0.45-0.79 W/kg	Increased DNA single strand breaks (Comet assay) in ear canal hair follicle cells; a dose-response relationship was observed.
Akhavan-Sigari et al. (2014)	Resected Glioblastoma multiforme (GBM) brain tumors from human patients	Increased mutant type of p53 expression in the peripheral zone of GBM in patient who use cell phone form ≥ 3 h/day; the increase was significantly correlated with shorter overall survival time.
Alkis et al. (2019a)	Rats exposed to 900 MHz (brain SAR 0.0845 W/kg), 1800 MHz (0.04563 W/kg), and 2100 MHz (0.03957 W/kg) RFR 2 h/day for 6 months	Increased DNA single strand break (Comet assay), oxidative DNA damage, and oxidative stress in brain frontal lobe.
Alkis et al. (2019b)	Rats exposed to 900 MHz, 1800 MHz, and 2100 MHz RFR 2 h/day for 6 months; maximum SAR over the rat 0.017 W/kg	Increased DNA single strand beak (Comet assay), oxidative DNA damage and oxidative stress in testicular tissue.
Alkis et al. (2021)	Rats exposed to 1800	Significant increases in liver in 8-hydroxydeoxyguanosine, DNA single strand

	MHz (SAR 0.62 W/kg), 1800 MHz (0.04563 W/kg), or 2100 MHz (0.2 W/kg) RFR 2 h/day for 7 months	breaks (Comet assay), malondialdehyde, total oxidant status, oxidative stress index,
*Al-Serori et al. (2017)	Human U87 (wild-type) and U251 (mutated) glioblastoma cells exposed to intermittent (5 mi ON/10 min OF) UMTS 1750 MHz signal for 16 h, SAR 0.25, 0.5, and 1 W/kg	No effect on micronucleus frequency. Apoptosis was induced in U231 cells.
Al-Serori et al. (2018)	Ten human cell types exposed to intermittent (5 mi ON/10 min OF) UMTS 1750 MHz signal for 16 h, SAR 0.25, 0.5, and 1 W/kg	Increased in single strand breaks (Comet assay) in U87 p52- proficient glioblastoma cells grew under serum free condition; no effect on double strand breaks (γ H2AX foci); nucleotide excision repair induced.
*Antonopoulos et al. (1997)	Human blood samples exposed to 380 MHz (17.65 Hz modulation, 0.08 W/kg); 900 MHz (217 Hz modulation, 0.208 W/kg); or 1700 MHz (217 Hz modulation, 1.7 W/kg) for 48-68 h	No significant effect on cell cycle progression and frequency of sister-chromatin exchange in lymphocytes.
Atasoy et al. (2013)	Male Wister rats exposed to 2437 MHz (Wi-Fi) RFR; 24 h/day for 20 weeks; maximum SAR 0.091 W/kg	Increased oxidative DNA damage and decreased catalase and glutathione activities in blood and testes.
Atlı Şekeroğlu et al (2013)	Immature (whole body SAR 0.38-0.78 W/kg) and mature (0.31-0.52 W/kg) rats exposed to 900 MHz RFR 2 h/day for 45 days	Increased bone marrow cell chromosome aberration, micronucleus frequency, mitotic index and ratio of polychromatic erythrocytes. Cytogenetic damages in immature rats were significantly higher than in the mature rats. No recovery on day 15 post-exposure.
Balode (1996)	Blood samples from female Latvian Brown cows lived close to and in front of the Skrundra	Significantly higher micronucleus concentration was found in the erythrocytes of the exposed cows.

	Radar and from a control area	
Banerjee et al. (2016)	Buccal mucosal cells from subjects who used their cellular phone less than five years and less than three hours a week (low), and those who used more than five years and more than 10 hours a week comprised of the second group.	Micronucleated frequency in buccal mucosal cells was found to be significantly increased in longer cellular phone users.
Baohong et al. (2005)	Human lymphocytes exposed in vitro to 1800 MHz RFR (SAR 3 W/kg) for two hours and also co-treated with various mutagens	DNA strand break assayed (Comet assay) at 0 and 21 h after treatment. No effect when cells were exposed to RFR alone. But, RFR co-exposure enhanced the DMA damage induced by mitomycin C and 4-nitroquinoline-1-oxide.
Baohong et al. (2007)	Human lymphocytes exposed in vitro to 1800 MHz RFR (SAR 3 W/kg) for 0, 1.5, and 4 h. Cells were also co-treated with ultraviolet ray C	DNA damage as assayed by the Comet assay showed no significant effect with RFR alone. But, RFR co-exposure reduced DNA damage induced by ultraviolet C.
Beaubois et al. (2007)	Tomato plant leaves exposed to a 900-MHz RFR or 10 min at 0.066 mW/cm ²	Evoked rapid and substantial accumulation of basic leucine-zipper transcription factor (bZIP) mRNA in the terminal leaf with kinetics very similar to that seen in response to wounding. (Effect attenuated by calcium antagonist.)
Bektas et al (2020)	Pregnant women who used cell phone and Wi-Fi; placenta and cord blood samples were analyzed	Samples from cell phone users showed increased oxidative DNA damage and oxidative stress; Wi-Fi users showed increased oxidative DNA damage but no oxidative stress; more DNA single strand breaks (Comet assay) in cell phone users than in control (did not use cell phone nor Wi-Fi) and Wi-Fi users; Wi-Fi and cell phone uses were synergistic.
Belyaev et al. (1992)	X-irradiated E. coli cells exposed to 51.62-51.84 GHz and 41.25-41.50 GHz millimeter-wave	Power density of 1 μW/cm ² was sufficient to suppress X-radiation-induced repair of genome conformational state.

	RFR	
Belyaev et al. (2005)	Lymphocytes from human subjects exposed to GSM 915 MHz RFR for 2 h ; SAR 0.037 W/kg;	Increased condensation of chromatin; no significant difference between responses of blood samples of healthy and electro-hypersensitive subjects.
Belyaev et al. (2006)	Rats exposed to GSM 915 MHz RFR for 2 h, SAR 0.4 W/kg	Affected gene expression in brain cells; no significant effect on chromatin conformation and double strand DNA breaks.
Belyaev et al. (2009)	Human lymphocytes exposed to UMTS cell phone signal(1947.4 MHz, 5 MHz band width) for 1 h; SAR 0.04 W/kg	Chromatin affected and inhibition of DNA double-strand break co-localizing 53BPI/gamma-H2AX DNA repair foci; lymphocytes from electro-hypersensitive subjects responded differently to UMTS and GSM signals in the formation of DNA repair foci than in healthy subjects.
*Bisht et al. (2002)	Mouse embryo sarcoma fibroblast C3H 10T½ cells exposed to FDMA (835.62 MHz; SAR 3.2 or 5.1 W/kg) and CDMA (847.74 MHz; SAR 3.2 or 4.8 W/kg) RFR for 3, 8, 16 or 24h	No significant effect on micronucleus formation.
Bourdineaud et al. (2017)	earthworms (<i>Eisenia fetida</i>) exposed to 900 MHz for 2 h; SAR 0.00013-0.00933 W/kg	DNA genotoxic effect persisted for at least 24 h; gene expressions up regulated for HSP70 (heat shock protein), MEKK1 (signal transduction); oxidative stress; and chemical and immune defenses.
*Bourthoumieu et al. (2010)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25 W/kg	No significant genotoxic effect was observed at 0 and 24 h after exposure by visual examination of chromosomal rearrangement.
*Bourthoumieu et al. (2011)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25, 1,2, and 4 W/kg	No significant change in the rate of aneuploidy of chromosomes 11 and 17 was found.
*Bourthoumieu et al. (2013)	Human amniotic cells exposed to GSM-900 MHz RFR for 24 h; SAR 0.25, 1,2, and 4 W/kg	No significant change in the expression and activation of the p53 protein was found. (p53 can cause cell cycle arrest and allow time for DNA repair or apoptosis.)
Burlaka et al. (2013)	Male Wister rats exposed to 245 MHz RFR for 2 h a day. 7 days a week for 2, 8, 15, or 30 days at 5-	Increased micronucleus formation was found in bone marrow erythropoietic cells after 15- day exposure; erythrocyte count, haemoglobin and haematocrit were

	10 mW/cm ² .	increased in peripheral blood after 8 and 15 days of exposure.
Buttiglione et al. (2007)	Human SH-SY5Y neuroblastoma cells exposed to modulated 900 MHz RFR for 24 h; SAR 1 W/kg	Increased Egr-1 gene expression paralleled with activation of the MAPK subtypes ERK1/2 and SAPK/JNK, and decrease in mRNA of Bcl-2 and surviving genes. RFR has anti-proliferative effect and causes cell cycle arrest at G2-M.
Cam and Seyhan (2012)	Hair root cells of human subjects after 15-30 min use of a 900-MHz GSM cell phone	Increased in DNA single strand breaks (Comet assay) was observed; more damages resulted after 30 min than after 15 min use.
Campisi et al. (2010)	Rat neocortical astroglial to 50 Hz-modulated or CW 900 MHz RFR for 5, 10, or 20 min; incident power density 0.0265 mW/cm ²	Significant increases in DNA fragmentation and reactive oxygen species were observed at 20 min only after exposure to the modulated RFR.
Cervellati et al. (2013)	Human placenta trophoblast-derived HTR-8/SVneo cells exposed to 1.8 GHz GSM RFR amplitude modulated by rectangular pulses of 217 Hz for 1 h; SAR 2 W/kg	Increased connexin Cx40 and Cx43 mRNA expression; decreased Integrin alpha1 and β 1 mRNA levels but enhanced Int alpha5 mRNA expression.
Chandel et al. (2019a)	Onion roots (<i>Allium cepa</i> L.) were exposed to 2350 MHz RFR for 1, 2, or 4 h, SAR 0.313 W/kg	Increased in mitotic index and chromosomal aberration; significant increase in DNA single strand break (Comet assay) at 2 and 4 h.
Chandel et al. (2019b)	Onion roots (<i>Allium cepa</i> L.) were exposed to 2100 MHz RFR for 1 or 4 h, SAR 0.282 W/kg	Increased mitotic index, chromosomal aberration, and DNA single-strand breaks (Comet assay) after 4 h of exposure.
*Chang et al. (2005)	<i>Escherichia coli</i> and <i>Salmonella typhimurium</i> exposed to 835 MHz RFR for 48h; SAR 4W/kg	835-MHz RFR neither affected the reverse mutation frequency nor accelerated DNA degradation in vitro. (Some interaction effects with mutagens were observed.)
Chaturvedi et al. (2011)	Male mice exposed to 2450 MHz RFR, 2 h/day for 30 days; SAR 0.03561 W/kg	Increased DNA single strand breaks (Comet assay) in brain cells.
*Chauhan et al. (2006a)	Human lymphoblastoma cells (TK6) exposed to pulsed-modulated,	No evidence of a general stress response with proto-oncogene and heat-shock protein gene transcriptions.

	intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 6 h; SAR 1 or 6 W/kg	
*Chauhan et al. (2006b)	Human –derived immune cell-lines HL-60 and MM6 cells exposed to pulsed-modulated, intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 6 h; SAR 1 or 10 W/kg	No evidence of detectable change in stress-related gene expression.
*Chauhan et al. (2007)	Human glioblastoma-derived cell-line (U87MG) and human monocyte-derived cell-line (MM6) exposed to pulsed-modulated, intermittent (5 min ON, 10 min OFF) 1900-MHz RFR for 24 and 6 h; SAR 0.1-10 W/kg	No evidence that the RFR exposure altered late onset gene expression in either cultured cell-lines.
Chavdoula et al. (2010)	Drosophila melanogaster flies exposed to GSM-900 MHz and DCS-1800 MHz cell phone radiation; 6 min per day for 5 days	Decreased insect’s reproductive capacity with fragmented DNA (apoptosis) in the egg chamber.
*Chemeris et al. (2004)	Frog (<i>Xenopus laevis</i>) erythrocytes exposed to high peak power pulsed RFR (8.8 GHz, 180 ns pulse width, peak power 65 kW, repetition rate 50 Hz) for 40 min; SAR 1.6 kW/kg (peak SAR 300 MW/kg)	Increased DNA single strand breaks (Comet assay) caused by temperature rise.
*Chemeris et al. (2006)	Human whole blood leukocytes and isolated lymphocytes exposed to pulsed 8.8 Hz RFR (180 ns pulse width, peak power 65 kW, pulse repetition frequency 50 Hz) for 40 min: average SAR 1.6 kW/kg (peak	No change in DNA single strand breaks (Comet assay)

	300 mW/kg)	
Chen et al. (2012)	Saccharomyces cerevisiae yeast cells exposed to 1800 MHz RFR for 6 h; SAR 4.7 W/kg	Expression of several genes.
*Choi et al. (2020)	Human adipose tissue-derived stem cells (ASCs), Huh7 and Hep3B liver cancer stem cells (CSCs), HeLa and SH-SY5Y cancer cells, and normal fibroblast IMR-90 cells exposed to WCDMA-signal 1.7-GHz RFR for 72 h, SAR 1 and 2 W/kg	No significant effect on double strand breaks; increased intracellular reactive oxygen species and decreased proliferation.
*Ciaravino et al. (1991)	Chinese hamster ovary cells exposed to 2450-MHz pulsed RFR (SAR 33.8 W/kg) simultaneously with adriamycin for 2 h	RFR did not affect changes in cell progression and number of sister chromatid exchanges induced by adriamycin.
d'Ambrosio et al. (1995)	Human blood exposed to 9 GHz RFR (continuous-wave or 50-Hz amplitude modulated) for 10 min; SAR 90 W/kg	Increased in micronucleus frequency in lymphocytes after exposure to the amplitude modulated RFR.
d'Ambrosio et al. (2002)	Human blood cultures exposed to 1748 MHz RFR (continuous –wave or phase modulated (GMSK)) for 15 min: SAR ~5 W/kg	Micronucleus frequency in lymphocytes was increased only after exposure to phase-modulated RFR.
Danese et al. (2017)	Human whole blood exposed to 900 MHz RFR from a cell phone for 30 min	No change in frequency of γ -H2AX foci (double strand DNA breaks) in lymphocytes.
De Amicis et al. (2015)	Human fetal fibroblasts exposed to THz radiation (0.1-0.15 THz) for 20 min; SAR 15-20 W/kg	Increased total number of micronuclei and centromere positive micronuclei that could lead to chromosome loss. No significant effect on DNA strand breaks (Comet assay), phosphorylation of H2AX histone and apoptosis.
De Iuliis et al. (2009)	Human spermatozoa exposed to 1800-MHz	Increased oxidative DNA damage and fragmentation (apoptosis) and reactive

	RFR; SAR 0.4 – 27.5 W/kg for 16 h	oxygen species; sperm motility and vitality were reduced.
*de Oliveira et al. (2017)	Human buccal cells from cell phone users; Averaged years of use 11.4 yrs; mean duration of daily use 2.8 min	Cells ipsilateral to cell phone use did not have a statistically significantly higher micronucleus frequency, compared to cells contralateral to exposure.
Del Re et al. (2019)	Human HeLa, BE2C and SH-SY5Y cells exposed to 900 MHz 217-Hz pulse-modulated RFR for 48 h; SAR 1 W/kg	Increased transcription of repetitive DNA, type of transcription depended on cell type. (Alteration of repetitive DNA transcription can be induced by environmental stress conditions, causing human pathological effects.)
Del Vecchio et al. (2009)	Murine SN56 cholinergic cell line (48 and 72 h) and rat primary cortical neurons (24, 72, 120 h) exposed to GSM-modulate 900 MHz RFR; SAR 1 W/kg	Increased expression of beta-thymosin (cytoskeleton regulating factor) m-RNA, and reduced neurite generation.
Demia et al. (2004)	Rats exposed to 910-MHz RFR 2 h/day for 30 days; SAR 0.42 W/kg.	Increased of micronuclei in polychromatic polymorphonuclear cells in bone marrow smears. Effects less in female rats.
Deshmukh et al. (2013)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 30 days.	Increased DNA single strand breaks (Comet assay) in brain tissues.
Deshmukh et al. (2015)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 180 days.	Increased DNA single strand breaks (Comet assay) in brain tissues; elevated heat-shock protein-70 level.
Deshmukh et al. (2016)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week	Increased DNA single strand breaks (Comet assay) in brain tissues; elevated heat-shock protein-70 level.

	for 90 days.	
Diem et al.(2005)	Human diploid fibroblasts and cultured rat granulosa cells exposed to 1800 MHz intermittent (5 min On/10 min Off) or continuous –wave; SAR 1.2 or 2 W/kg	Increased in DNA single and double strand breaks (Comet assay) in both cell types after 16 h exposure. Intermittent wave showed a higher effect than continuous wave.
Duan et al (2015)	Mouse spermatocyte-derived GC-2 cells exposed to intermittent (5 min On/10 min Off) 1800 MHz RFR (from a GSM cell phone in talk mode) for 24 h; SAR 1.2 , or 4 W/kg	Increased oxidative DNA damage a 4 W/kg; no significant with Comet assay.
*Durdik et al. (2019)	Umbilical cord blood (UCB) cells exposed to a GSM900 (1-17 h, 0.004 or 0.04 W/kg) or UMTS-1947.4 MHz (3 h, 0.04 /kg) cell phone signals fed to a TEM cell	No changes in DNA single and double strand breaks (Comet assay), and apoptosis; increased reactive oxygen species was observed.
Eker et al. (2018)	Female Wistar-albino rats exposed to 1800-MHz RFR for 2h/day for 8 weeks; SAR 0.06 W/kg	Caspase-3 and p38MAPK gene expressions increased in eye tissues.
Engelmann et al. (2008)	Cell suspension cultures of Arabidopsis thaliana exposed to 1900 MHz UMTS-modulated RFR for 24 h; SAR peak 2 W/kg, average 0.75 W/kg	Significant changes in transcription of 10 genes.
Esmekaya et al. (2011)	Human peripheral blood lymphocytes exposed to 1800 MHz GSM- (217 HZ) modulated RFR for 6, 8, 24, or 48 h; SAR 0.21 W/kg	Chromatin changes and increase in sister chromatin exchange.
*Falzone et al. (2010)	Human spermatozoa exposed to pulse-modulated 900-MHz RFR for 1 h; SAR: 2.0 and 5.7 W/kg	No significant effects on DNA fragmentation, reactive oxygen species, and capase-3 activity.

Ferreira et al. (2006)	Pregnant rats exposed to a cell phone at 834 MHz for 8.5 h/day from conception to birth; SAR 0.55-1.23 W/kg	Increased erythrocyte micronucleus frequency but no significant effects in oxidative parameters in blood and liver of newborn pups.
Figueiredo et al. (2004)	Human whole blood exposed to 2.5 GHz RFR (from a microwave oven) for 40 sec (SAR 626.67 W/kg) or 10.5 GHz RFR for 5 min (SAR 0.25 W/kg)	No chromosomal aberrations observed in lymphocytes; no alteration in radiosensitivity to gamma radiation; cell mortality increased markedly after RFR exposure.
*Finnie et al. (2006)	Pregnant mice exposed to 900-MHz RFR (modulated at 217 Hz with pulse-width of 0.6 ms) for 60 min per day from day 1-19 of gestation; SAR 4 W/kg	No significant effect on c-fos expression in brain of offspring.
Fragopoulou et al. (2018)	C57BL/6 adult male mice exposed to 2 hr to GSM 1800-MHz RFR (from a phone) for 2 h at an average power density of 0.0049-0.081 mW/cm ²	In the hippocampus, the expression of 178 genes changed significantly, revealing an impact on genes involved in critical biological processes, such as cell cycle, DNA replication and repair, cell death, cell signaling, nervous system development and function, immune system response, lipid metabolism, and carcinogenesis.
Franchini et al. (2018a)	Human fetal and adult fibroblasts exposed to 25 GHz RFR for 20 min; SAR 20W/kg	Increased total number of micronuclei and centromere positive micronuclei in exposed samples. No significant effect on DNA single strand break (Comet assay).
Franchini et al. (2018b)	Human adult fibroblasts exposed to 0.15 THz (150 GHz) RFR (4 μs pulses at 25 Hz) for 20 min; SAR 15-20 W/kg	Increased centromere-positive micronuclei frequencies and chromosomal nondisjunction events, indicating induction of aneuploidy and not by DNA breakage.
Franzellitti et al. (2008)	Human trophoblasts HTR-8/SVneo exposed to 1800 MHz continuous-wave, GSM-217-Hz, and GSM-Talk signals for 4-24 h, time averaged SAR 2 W/kg	Levels of the inducible HSP70C transcript were significantly enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals. No effect on inducible HSP70A, HSP70B and the constitutive HSC70 transcripts.
Franzellitti et al. (2010)	Human trophoblast HTR-8/SVneo cells exposed to 1800 MHz	GSM signals increased DNA single strand breaks (Comet assay) after 16 and 24 h exposure; recovered within 2 h post-

	continuous –wave. GSM (217 Hz modulated) and GSM intermittent (5 min on/10 min off) RFR for 4, 16, or 24 h: SAR 2 W/kg	exposure; continuous-wave RFR was without effect.
*Fritze et al. (1997)	Rats expose to GSM 90 MHz RFR for 4 h, brain average SAR 0.3- 1.5 W/kg	No effect on C-jun and GFAP expression in brain.
Fucic et al. (1992)	Lymphocytes from humans occupationally exposed to RFR; 1250-1350 MHz, 10 μ W/cm ² -20 mW/cm ²	Showed preferentially clastogenic effect measured by micronucleus. Effect on genetic material similar to both of a chemical agent and of ionizing radiation.
Furtado-Filho et al. (2014)	Rats of different ages (0-30 days) exposed 950 MHz RFR for 0.5 h/day for 51 days (21 days of gestation and 6-30 days old): SAR pregnant rat 0.01-0.03 W/kg; neonate 0.88 W/kg, 6-day old 0.51 W/kg, 15-day old 0.18 W/kg, 30-day old 0.06 W/kg.	Decreased DNA single strand breaks (Comet assay) in liver of 15-day old and increased breaks in 30-day old rats, no oxidative stress detected.
*Furtado-Filho et al. (2015)	At exposed to 950 MHz RFR. 0.5 h/day to 27 days (throughout pregnancy and 6 days postnatal); SAR 0.44-0.35 W/kg, neonatal rat 1.32 W/kg, 6-day old 1.14 W/kg	Right cerebral cortex showed an increase in DNA single strand breaks (Comet assay), but no significant effect in the left cerebral cortex in RFR-exposed 6-day old rats. No oxidative effects observed.
Gadhia et al. (2003)	Blood samples of cell phone and non-cell phone users	Increased dicentric chromosomes and sister chromatid exchange in lymphocytes of cell phone users.
Gajski and Garaj-Vrhovac (2009)	Blood samples from Wistar rats exposed to GSM-modulated 915 MHz RFR for 30 min, SAR 0.6 W/kg	Increased basal (single strand) and oxidative DNA damage (Comet assay) in lymphocytes.
Gandhi and Anita (2005)	Blood from cell phone users (most for 2-5 yrs)	Increased DNA single strand breaks (Comet assay) and micronucleus found in cell phone users.
Gandhi and Singh	Blood and buccal cells	Increased micronucleated buccal cells and

(2005)	from cell phone users (3-4,5 yrs); controls never used cell phone	chromosomal aberration in peripheral lymphocytes.
Gandhi et al. (2015)	People lived within 300 m of a cell phone base station (average power density= 1.149 mW/cm ²) for an average of 7.45 yrs, controls average power density = 0.0045 mW/cm ² .	Increased DNA single strand breaks (Comet assay) in peripheral blood leukocytes. Daily cell phone usage, location of residence, and power density are significant predictor of DNA damage.
Gapeyev et al. (2014)	Mouse blood samples exposed to 1-Hz pulse-modulated 42.2 GHz RFR for 20 min, SAR 1.5 W/kg; and x-rays	Pre-exposure to pulse-modulated RFR (not continuous-wave) reduced x-ray-induced DNA single strand breaks (Comet assay) in lymphocytes Effect may be related induction of reactive oxygen species by RFR.
Garaj-Vrhovac et al. (1990)	V79 Chinese hamster cells exposed to 7.7 GHz RFR for 15, 30, or 60 min; power density 30 mW/cm ²	Inhibited [³ H]thymidine into DNA with stoppage of cell cycle at S phase; chromosome aberration observed.
Garaj-Vrhovac et al. (1991)	V79 Chinese hamster fibroblast cells exposed to 7.7 GHz RFR for 15, 30, or 60 min; power density 0.5 mW/cm ²	Increased chromosome aberration (dicentric and ring chromosomes) and micronucleus.
Garaj-Vrhovac et al. (1992)	Human whole blood samples exposed to 7.7 GHz RFR for 10, 30, or 60 min; power density 0.5, 10, or 30 mW/cm ²	Increased chromosome aberration (dicentric and ring chromosomes) and micronucleus in lymphocytes.
Garaj-Vrhovac and Fucic (1993)	Air traffic controllers who did repair on radar devices two days ago and exposed to 1250-1350 MHz RFR of unknown intensity (pulse power 100 kW). (presumably higher than normal exposure of 10 μW/cm ² -20 mW/cm ²)	Lymphocytes showed increased number of chromosome breaks, acentric fragments, dicentric and polycentric chromosomes with accompanying fragments, ring chromosomes and chromatid interchange. Most aberrations returned to normal after 30 weeks, except dicentrics and ring chromosomes.
Garaj-Vrhovac. (1999)	Peripheral blood lymphocytes of workers on radar equipment and antenna system service, 1250-1350 MHz; power	Exposed subjects shows an increase in the number of micronucleus and number of micronucleus per cell; disturbance of cells in the cell cycle.

	density 10 $\mu\text{W}/\text{cm}^2$ -20 mW/cm^2 ; average employment duration 13.3 yrs	
Garaj-Vrhovac and Orescanin (2009)	Peripheral blood lymphocytes of workers on radar equipment and antenna system service, 1250-1350 MHz; power density 10 $\mu\text{W}/\text{cm}^2$ -20 mW/cm^2 ; average employment duration 13.3 yrs	Increased DNA single strand breaks (Comet assay) and bleomycin-induced chromatid breakage.
Garaj-Vrhovac et al. (2009)	Wistar rats exposed to 915 MHz RFR 1 h/day for two weeks, SAR 0.6 W/kg	Increased basal DNA single strand break and oxidative DNA damages (Comet assay) in blood leukocytes.
Garaj-Vrhovac et al. (2011)	Workers occupationally exposed to marine radar pulsed RFR (3, 5.5, and 9.4 GHz)	Increased DNA single strand break (Comet assay) and micronucleus in blood lymphocytes; increased oxidative stress.
*Garson et al. (1991)	Blood samples of radiolinenmen occupationally exposed to 400 kHz – 20 GHz	No increase in chromosomal damage in lymphocytes.
Ghatei et al. (2017)	Mice exposed pre- and post-natally to radiation from a cellular phone jammer (900 and 1800 MHz)	At 8-10 weeks old, in the cerebellum, no effect on expression level of bcl-2 and p53 genes, but gene expression level of <i>bax</i> was decreased and gene expression level of <i>p21</i> was increased.
*Glaser et al. (2016)	Human hematopoietic stem cells and leukemia HL-60 cells exposed to GSM (900 MHz), UMTS (1,950 MHz) and LTE (2,535 MHz) for 4, 20 or 66 h; SAR 0-4 W/kg	No effect on apoptosis, oxidative stress, cell cycle, DNA damage (DNA single strand breaks (Comet assay)) and DNA repair. A significant decrease in DNA breaks was found in hematopoietic stem cells exposed for 4 h to GSM signal.
Gökçek-Saraç et al. (2020)	Rats exposed to UMTS 2100 MHz RFR 2h/day for 7 days; whole body average SAR 0.47 or 2.17 W/kg	Decreased RNA expressions of acetylcholinesterase (AChE), choline acetyltransferase (ChAT), and vesicular acetylcholine transporter (VACHT) in the hippocampus; deficit in object location and Y-maze tests.
*Görlitz et al. (2005)	B6C3F1 mice exposed to GSM900 or DCS 1800 signals for 2 h/day for 1	No effect on micronucleus frequency in erythrocytes of the bone marrow or peripheral blood, in keratinocytes, or in

	week (SAR 0-33.2 W/kg) or 6 weeks (SAR 0-24.9 W/kg)	spleen lymphocytes.
Gorpinchenko et al. (2014)	Human sperms exposed to a cell phone in stand-by/talk mode for 5 h	Increased DNA fragmentation (apoptosis) and decreased motility in spermatozoa.
Gulati et al. (2016)	Blood and buccal cells of people lived close (<400 meters) to a cell tower; 1800 MHz, Maximum power density (at 150 meters) $1.22 \mu\text{W}/\text{cm}^2$, some subjects lived in the area for more than 9 yrs	Increased DNA single strand breaks (Comet assay) in lymphocytes and micronucleus in buccal cells. Female subjects had significantly higher effects than males.
Gulati et al. (2018)	Blood samples from subjects lived 400 m from cell towers for 8-9 years, power density $0.037\text{-}12.20 \text{ mW}/\text{cm}^2$	A significant association of genetic polymorphism of antioxidant genes (for MnSOD and CAT) with oxidative damage has been observed in human population exposed to radiations emitted from mobile towers. Decreased MnSOD and CAT activities and increased lipid peroxidation observed in blood serum.
Gulati et al. (2020)	Human lymphocytes exposed to UMTS signals at 1923, 1947.47, and 1977 MHz for 1 or 3 hr; SAR 40 mW/kg	Observed DNA damage (Comet assay) depending on UMTS frequency with maximal effect at 1977 MHz; no effects on ROS, apoptosis, preleukemic fusion genes, and mutations in TP53 gene.
Guler et al (2010)	Pregnant and non-pregnant New Zealand white rabbit exposed to GSM 1800-MHz RFR for 15 min/day for 7 days (15 th to 22 nd days of gestation); power density $0.052 \text{ mW}/\text{cm}^2$	Increased oxidative DNA damage and lipid peroxidation in brain tissues in adult rabbits, no significant effect in newborn rats
Guler et al. (2012)	New Zealand white rabbits exposed to GSM 180-MHz RFR for 15 min/day in utero between 15 th to 22 nd days of gestation and at 1-month old 15 min/day 7 days for female and 14 days for male; SAR 1.8 W/kg	Increased DNA oxidative damage in liver of female rabbits (not in male) and increased lipid peroxidation in liver of both male and female rabbits.
*Gurbuz et al. (2010)	Female Wistar rats	No significant effect on micronucleus

	exposed to GSM 1800-MHz RFR 20 min/day, 5 days/week for 1 month; power density 0.0054 mW/cm ²	frequency in bladder cells.
*Gurbuz et al. (2014)	Male Wistar rats exposed to 1800- or 2100-MHz RFR 30 min/day, 6 days/week for 1 or 2 months; SAR 0.23 W/kg	No significant effect on micronucleus frequency in bladder cells.
*Gurbuz et al. (2015)	Normal and diabetic rats exposed to a 2100-MHz RFR 30 min/day, 5 days/week for 1 month; SAR 0.24 W/kg	No effect on micronucleus frequency in exfoliated bladder cells in both normal rats and rats with chronic disorder.
*Gurisik et al. (2006)	Two human cell lines (neuronal SK-N-SH) and monocytoid U937) exposed to a GSM 900-MHz RFR for 2 h; SAR 0.2W/kg	No significant effects on gene expression, heat shock protein level, and cell cycle distribution in SK-N-SH cells; and no effects on cell viability and cell cycle in U937 cells.
Gürler (2014)	Wistar rats exposed to 2450 MHz RFR 1 h/day for 30 consecutive days; power density 0.0036 mW/cm ²	Increased oxidative DNA damage in brain and blood, and oxidative protein products in blood.
Gustavino et al. (2016)	Secondary roots of Vicia faba (broad bean) seedlings exposed to continuous-wave 915-MHz RFR for 2 h; SAR 0.4-1.5 W/kg	Increased micronucleus frequency up to 7-fold.
Habauzit et al. (2014)	Human keratinocytes exposed to 60.4 GHz RFR for 3 hr, incident power density of 20 mW/cm ² : SAR 594 W/kg (average), 1233 M/kg (peak)	7 gene expressions showed specific electromagnetic effect under hyperthermia condition (i.e., not mimicked by heat-shock controls).
* Habauzit et al. (2020)	Male hairless rats exposed to 94 GHz RFR 3 h/day, 3 days/week for 5 months, incident power density 10 mW/cm ²	No significant modification of gene expression in skin cells.
Haider et al. (1994)	Plant cutting bearing young flower buds	Increased micronucleus was found in all conditions (compared to lab controls).

	exposed for 30 h to short-wave 10-21 MHz RFR on both sides of a slewable curtain antenna (0.424-7.67 mW/cm ²), at 15 m (2.15 mW/cm ²) and 30 m (1.3 mW/cm ²) from a cage antenna; and 200 m from a broadcasting station (0.00027-0.0024 mW/cm ²)	
Hanci et al. (2013)	Pregnant rats exposed 1 h/day on days 13-21 of pregnancy to 900-MHz RFR at power density 0.0265 mW/cm ² .	Testicular tissue of 21-day old offspring showed increased DNA oxidative damage, apoptotic index, and lipid peroxidation.
*Hansteen et al. (2009a)	Human lymphocytes exposed to 18 GHz or pulsed 16.5 GHz RFR for 53 h	No significant effect on chromosomal aberration frequency.
*Hansteen et al. (2009b)	Human lymphocytes exposed to 2.3 GHz continuous-wave or pulsed (200 Hz, 50% duty cycle) RFR	No significant effect on chromosomal aberration frequency.
Hao et al. (2010)	Murine N9 microglial cells were exposed to pulsed 2450-MHz RFR for 20 min, SAR 6.2 W/kg	Significant induced phosphorylation of STAT3, increased transcription levels of the inflammation-associated genes, iNOS and TNF-alpha, which are reported to contain STAT-binding elements in their promoter region. (STAT3 is a transcription activator that mediates the expression of a variety of genes in response to cell stimuli, and thus plays a key role in many cellular processes such as cell growth and apoptosis.)
He et al. (2016)	Mouse bone marrow stromal cells exposed to a 900 MHz RFR 3 h/day for 5 days; peak and average SAR 4.1 x 10 ⁻⁴ and 2.5 x 10 ⁻⁴ W/kg	Increased expression of PARP-1 mRNA. (PARP-1 involved in DNA repair, genomic stability and apoptosis and is activated by DNA single strand breaks.)
He et al. (2017)	Mouse bone marrow stromal cells exposed to a 900 MHz RFR 3 h/day for 5 days; peak and average SAR 4.1 x 10 ⁻⁴	Induced PARP-1. Cells exposed to RFR and gamma ray showed significantly decreased genetic damage (DNA single strand break (Comet assay)) as well as faster kinetics of repair compared with those exposed to GR

	and 2.5×10^{-4} W/kg, some cells were challenged with one dose of gamma ray.	alone.
Hekmat et al. (2013)	Calf thymus exposed to 940 MHz RFR for 45 min; SAR 0.04 W/kg	Altered DNA structure at 0 and 2 h after exposure; conformational changes and disaggregation caused by increment in surface charge and size of DNA.
*Hintzsche and Stopper (2010)	Oral cavity mucosa cells from human subjects who used cell phones for different durations weekly (0, <3 h, and > 3h)	No significant change in micronucleus frequency in mucosa cells with cell phone use.
*Hintzsche et al. (2012a)	Human HaCaT cells and A(L) human-hamster hybrid cells exposed to continuous-wave or GSM-modulated 900 MHz RFR for 30 min or 22 h; power density 0.0066-2.15 mW/cm ²	No significant effect on micronucleus frequency.
*Hintzsche et al. (2012b)	Human keratinocytes (HaCaT) and human dermal fibroblasts (HDF) exposed to 0.106 THz (106 GHz) RFR for 2, 8, 24 h; 0.88 -2 mw/cm ² (2mw/cm ² gave a SAR of 13.34 W/kg)	No effect on micronucleus frequency and DNA single strand breaks (Comet assay).
*Hirose et al. (2006)	Human glioblastoma A172 cells exposed to 2.1425 GHz W-CDMA radiation at SARs of 0.08, 0.25, and 0.8 W/kg, and continuous-wave radiation at 0.08 W/kg for 24 or 48 h; and human IMR-90 fibroblasts from fetal lungs exposed to both W-CDMA and continuous-wave RFR at a SAR of 0.08 W/kg for 28 h	No significant changes in induction of p53-dependent apoptosis, DNA damage, or other stress response
*Hirose et al. (2007)	Human glioblastoma	No significant induction of phosphorylation

	A172 cells were exposed to W-CDMA radiation at SARs of 0.08 and 0.8 W/kg for 2-48 h, and continuous-wave 2.1425 GHz RFR at 0.08 W/kg for 24 h, and human IMR-90 fibroblasts from fetal lungs were exposed to W-CDMA at 0.08 and 0.8 W/kg for 2 or 28 h, and continuous-wave at 0.08 mW/kg for 28 h.	of hsp27 or expression of heat shock protein gene family.
*Hook et al. (2004)	Human Molt-4 T lymphoblastoid cells exposed to 847.74 MHz code-division multiple-access (CDMA) (SAR 3.2 W/kg), 835.62 MHz frequency-division multiple-access (FDMA) (3.2 W/kg), 813.56 MHz iDEN(R) (iDEN) (0.0024 or 0.024 W/KG), and 836.55 MHz time-division multiple-access (TDMA) (0.0026 or 0.026 W/kg) for up to 24 h	No significant changes in DNA single strand breaks (Comet assay) and apoptosis.
*Hou et al. (2015)	Mouse embryonic fibroblasts (NIH/3T3) exposed to intermittent (5 min on/10 min off) 1800-MHz GSM-talk mode RFR from 0.5 to 8 h; SAR 2 W/kg.	No effect on γ H2AX foci frequency (Increased reactive oxygen species and late apoptotic cells).
Houston et al. (2019)	Male mice exposed to 906 MHz RFR for 12 h/day for 1, 3, or 5 weeks; SAR 2.2 W/kg	Increased DNA oxidative and fragmentation (Comet assay) in spermatozoa across all exposure periods, increased mitochondrial reactive oxygen species.
*Huang et al. (2008a)	Jurkat human T lymphoma cells exposed for 24 h to 1763 MHz RFR; SAR 10 W/kg	Alterations in cell proliferation, cell cycle progression, DNA integrity (Comet assay) or global gene expression were not detected.
*Huang et al. (2008b)	HEI-OC1 immortalized mouse auditory hair cells	No significant effects on cycle distribution, DNA damage (Comet assay), stress response

	exposed to 1763 MHz (CDMA) RFR for 24 or 48 h; SAR 20 W/kg	and gene expression.
*Jeong et al. (2018)	14-month old C57BL/6 mice exposed to 1950 MHz RFR for 2 h/day, 5 day/wk, 8 months; SAR 5 W/kg	No significant effects on levels of oxidative stress, oxidative DNA damage, apoptosis, astrocyte, or microglia markers in brain tissues.
Jeong et al. (2020)	2 and 12-month old C57BL/6 mice exposed to 1950-MHz RFR 2h/day, 5 day/wk for 8 months; SAR 5 W/kg	Increased expression of Epha8 and Wnt6 genes in the hippocampi at 20 months after exposure, although 13 additional genes showed no significant changes. Cognitive enhancement detected in 1-month mice after exposure may be associated with increases in neurogenesis-related signals.
Ji et al (2004)	Human subjects used cell phones for 4 h.	DNA single strand breaks (Comet assay) increased in peripheral blood cells (T-cells, B-cells, granulocytes).
Ji et al. (2016)	Mouse bone-marrow stromal cells (BMSC) exposed to 900-MHz RFR for 4 h/day for 5 days; power density 0.12 mW/cm ² ; some cells were also irradiated with 1.5 Gy γ -radiation after RFR exposure	RFR followed by γ -radiation exposure significantly decreased number of DNA strand breaks (Comet assay) and resulted in faster kinetics of repair of DNA strand breaks compared to γ -radiation alone. Thus, data suggest that RFR preexposure protected cells from damage induced by γ -radiation.
Jiang et al. (2012)	Mice were pre-exposed to a 900-MHz RFR for 4 h/day for 1, 3, 5, 7, and 14 days; power density 0.12 mW/cm ² and then subjected to an acute dose of 3 Gy γ -radiation	DNA single strand breaks (Comet assay) in blood leukocytes from mice pre-exposed to RFR for 3, 5, 7, and 14 days showed progressively decreased damage and was significantly different from those exposed to γ -radiation alone.
Jiang et al. (2013)	Mice exposed to a 900-MHz RFR 4/day for 7 days, SAR 0.548 W/kg and also γ -radiation	Pre-exposure to RFR decreased micronucleus frequency induced by γ -radiation in immature erythrocytes in peripheral blood and bone marrow.
*Juutilainen et al. (2007)	Female CBA/S mice were exposed for 78 weeks (1.5 h/day, 5 day/week) to either a continuous 902.5-MHz signal similar to that emitted by analog NMT (Nordic Mobile	No significant effects of RFR on micronucleus frequency in polychromatic or normochromatic erythrocytes.

	Telephone) phones at a whole-body SAR of 1.5 W/kg, or to a pulsed 902.4-MHz signal similar to that of digital GSM phones at 0.35 W/kg and also 4 Gy of X-ray on the first three weeks; female transgenic mice (line K2) and their nontransgenic littermates were exposed for 52 weeks (1.5 h/day, 5 day/week) to two digital mobile phone signals, GSM and DAMPS at SAR 0.5 W/kg, and repeated ultraviolet radiation	
Karaca et al. (2012)	Mouse brain cells exposed to a 10.715 GHz RFR for 6 h/day for three days, SAR 0.725 W/kg	Increased micronucleus apoptosis and necrosis, and decreased expression of the STAT3 genes.
*Kerbacher et al. (1990)	Chinese Hamster Ovary cells exposed for 2 h to pulsed 2450 MHz RFR; SAR 33.8 W/kg	No significant effect on chromosome aberration; no interactions with Mitomycin C and Adriamycin.
Kesari and Behari (2009)	Male Wistar rats exposed to 50-GHz RFR 2 h/day for 45 days; SAR 0.0008 W/kg	Increased in brain tissue DNA double strand breaks (Comet assay); decreased antioxidant enzymes superoxides dismutase and glutathione peroxidase, and increased catalase activity.
Kesari et al. (2010)	Male Wistar rats exposed to 2.45-GHz RFR 2 h/day for 35 days; SAR 0.11 W/kg	Increased in brain tissue DNA double strand breaks (Comet assay); decreased antioxidant enzymes superoxides dismutase and glutathione peroxidase, and increased catalase activity.
Kesari et al. (2011)	Male Wistar rats exposed to 900 MHz-GSM signal 2 h/day for 35 days; SAR 0.9 W/kg	Decreased micronucleus frequency, change in cell cycle and increased oxidative stress in sperm cells.
Kesari et al. (2014)	Male Wistar rats exposed to a 3D cell phone. 2h/day for 60 days; SAR 0.26 W/kg	Increased DNA double strand breaks (comet assay), micronuclei, Caspase 3 and apoptosis in brain cells; activation of hsp27/p38MAPK stress pathway.
*Khalil et al (2011)	Mice exposed to 900	No effects on plasma, brain, and spleen 8-

	MHz-GSM signal 30 min/day for 30 days; SAR 1 W/kg	oxo-7, 8-dihydro-2'- deoxyguanosine and oxidative stress.
Khalil et al. (2012)	Male Sprague-Dawley rats exposed for 2 h to 1800-MHz GSM signal, SAR 1 W/kg	Urine samples collected 0.5, 1, 2, and 4 h from the beginning of exposure showed elevated 8-oxo-7, 8-dihydro-2'-deoxyguanosine (from repair of oxidative DNA damage) level.
*Khalil et al. (2014)	Saliva of cellular phone users collected before as well as after 15 and 30 min use of phones.	No change in 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-Oxo-dG). There was no relationship between cell phone use and changes in the salivary oxidant/antioxidant profile.
Kim et al. (2008)	Mouse lymphoma cells and Chinese hamster lung cells exposed to 835-MHz RFR for 48 h; SAR 4W/kg	RFR increased clastogens-induced DNA single strand breaks (Comet assay).
*Komatsubara et al. (2005)	Mouse m5S cells exposed for 2 h to 2450 MHz CW RFR (SAR 5,10, 20, 50 and 100 W/kg) or pulsed RFR (SAR mean 100W/kg, peak 900 W/kg)	No chromosomal aberration observed.
Korenstein-Ilan et al (2008)	Human dividing lymphocytes exposed to 0.1 THz RFR (0.031 mW/cm ²) for 1, 2, or 24 h	Change in chromosomes number in chromosomes 11 and 17 were most vulnerable (about 30% increase in aneuploidy after 2 and 24 h of exposure), while chromosomes 1 and 10 were not affected, and in the asynchronous mode of replication of centromeres 11, 17 and 1 (by 40%) after 2 h of exposure. 0.1 THz radiation induces genomic instability. It is speculated that these effects are caused by radiation-induced low-frequency collective vibration modes of proteins and DNA.
Koyama et al. (2003)	Chinese hamster ovary (CHO)-K1 cells exposed to 2450 MHz RFR for 18 h; SAR 13-100 W/kg	Higher micronucleus frequency after exposure at 78 W/kg and higher. Synergistic with bleomycin in micronucleus formation.
Koyama et al. (2004)	Chinese hamster ovary K1 cells exposed to 2450 MHz RFR for 2h; SAR5-200 W/kg	Increased micronucleus formation above 50 W/kg (May be related to temperature rise).

*Koyama et al. (2016a)	Human corneal epithelial (HCE-T) cells exposed to 0.12 THz radiation at 5 mW/cm ² for 24 h	No effect on micronucleus formation, morphological change and heat shock protein expression (Hsp27, Hsp70, and Hsp90 α).
*Koyama et al. (2016b)	Human corneal epithelial (HCE-T) and human lens epithelial (SRA01/04) cells exposed to 60 gigahertz (GHz) RFR for 24 h; 1 mW/cm ²	No effect on micronucleus formation DNA single strand breaks (Comet assay) and heat shock protein expression.
Kumar A. et al. (2020)	Allium cepa (onion) root meristematic cells exposed to 900- (0.0902 W/kg) and 1800-MHz (0.169 W/kg) RFR for 0.5, 1, 2, and 4 h	Increased chromosomal aberrations and increased DNA single strand breaks (Comet assay).
*Kumar G. et al. (2011)	Long bone (femur and tibia) of male Sprague – Dawley rats exposed to 900-MHz continuous-wave RFR for 30 min; SAR 2 W/kg	No significant effect on DNA single-strand breaks (Comet assay) in bone marrow lymphocytes.(Assayed at 72 h after exposure.)
*Kumar G. et al. (2015)	Long bone (femur and tibia) of male Sprague – Dawley rats exposed to 900 and 1800 MHz continuous-wave and pulsed RFR; 900-MHz CW at 2 and 10 W/kg for 90 min and 1800-MHz CW and PW at 2.5 and 12.4 W/kg for 120 min	No significant effect on DNA single-strand breaks (Comet assay) in bone marrow lymphoblasts. (Assayed at 1 h after exposure.)
Kumar R. et al. (2020)	male Wistar rats exposed to 900 MHz, 1800 MHz and 2450 MHz RFR at a specific absorption rate (SAR) of 5.84×10^4 W/kg, 5.94×10^4 W/kg and 6.4×10^4 W/kg, respectively for 2 h per day for 1-month, 3-month and 6-month periods.	RFR exposure caused significant epigenetic modulations (DNA and histone methylation) which alter gene expression in the hippocampus.
Kumar S. et al. (2010)	Male Wistar rats exposed to 10-GHz RFR 2 h a day for 45 days, SAR	Increased micronucleus and reactive oxygen species in blood cells.

	0.014 W/kg	
Kumar S. et al. (2013)	Male Wistar rats exposed to a 10 GHz RFR 2h/day for 45 days; SAR 0.014 W/kg	Increased micronucleus frequency in blood lymphocytes and increased single strand breaks (Comet assay) in spermatozoa. Decreased testosterone and testicular size.
Kumar S. et al. (2014)	Male Wistar rats exposed to 1910.6 MHz RFR from a cell phone in “talk mode” for 60 days (2 h/day, 6 days a week); SAR 0.28 (Max.) and 0.0226 (Min.)	Increased DNA single strand breaks (Comet assay) and lipid peroxidation in spermatozoa,
*Lagroye et al. (2004a)	Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h; SAR 1.2 W/kg	No significant change in DNA single strand breaks (Comet assay) (with or without proteinase-k treatment of samples-for detection of DNA-protein crosslinks) in brain cells.
*Lagroye et al. (2004b)	Clonal mouse embryo C3H 10T(1/2) cells exposed 2450-MHz continuous-wave RFR for 2 h; SAR 1.9 W/kg	No significant change in DNA single strand breaks (Comet assay) (with or without proteinase-k treatment of samples.)
Lai and Singh (1995)	Male Sprague-Dawley rats exposed to pulsed or continuous-wave 2450-MHz RFR for 2 h; SAR 0.6 and 1.2 W/kg	Increased DNA single strand breaks (Comet assay) in brain cells was observed at 4 h after exposure to pulsed RFR and at 0 and 4 h after continuous-wave exposure.
Lai and Singh (1996)	Male Sprague-Dawley rats exposed to pulsed or continuous-wave 2450-MHz RFR for 2 h; SAR 1.2 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells was observed at 4 h after exposure to pulsed or continuous-wave RFR.
Lai and Singh (1997)	Male Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h; SAR 1.2 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effects blocked by melatonin or the spin-trap compound N-tert-butyl-alpha-phenylnitron. (Free radicals are involved in the effects).
Lai and Singh (2005)	Male Sprague-Dawley rats exposed to continuous-wave 2450-MHz RFR for 2 h; SAR 0.6 W/kg	Increased DNA single- and double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effects blocked by a temporally incoherent magnetic field.
Lai et al. (1997)	Male Sprague-Dawley rats exposed to pulsed 2450-MHz RFR for 2 h;	Increased DNA double-strand breaks (Comet assay) in brain cells at 4 h after exposure. Effect blocked by naltrexone. (Involvement

	SAR 1.2 W/kg	of endogenous opioids in the effects).
Lakshmi et al. (2010)	Human subjects professionally using VDTs	No effect on DNA single strand break (comet assay) and micronucleus frequency in blood cells of subjects exposed for 2 years; increased in long-term (>10 years) users.
Lameth et al. (2020)	Healthy rats, rats undergoing an acute neuroinflammation triggered by a lipopolysaccharide (LPS) treatment, and transgenic hSOD1 ^{G93A} rats that modeled a presymptomatic phase of human amyotrophic lateral sclerosis (ALS) exposed head only to a GSM-1800 MHz RFR for 2 h, SAR 3.22 W/kg.	Cortical cell gene modulations triggered by GSM-RFR in the course of an acute neuroinflammation and indicate that GSM-induced gene responses can differ according to pathologies affecting the CNS.
*Lamkowski et al. (2018)	Human peripheral blood cells exposed to 900 MHz RFR for 30, 60, and 90 min; SAR 9.3 W/kg	No significant effect on gene expression.
Le Quément et al. (2012)	Primary human skin cells exposed to a 60.4-GHz RFR for 1, 6, or 24 h, SAR 42.4 W/kg.	Expression of 130 transcripts was found to be potentially modulated. PCR confirmed 5 genes as differentially expressed after 6 h of exposure.
*Lerchl et al. (2020)	Pregnant mice exposed to UMTS ~1960 MHz RFR from day 7 post-conception (p.c.) at SAR 0.04 and 0.4 W/kg (24 h/day, 7 days/week); at day 14 p.c., injected with ethylnitrosoures(ENU)	No DNA adenylyl adduct formation was observed in the brain of fetuses at 24, 36, and 72 h after ENU injection.
Lee et al. (2005)	Human HL-60 cells exposed to a pulsed 2450 MHz RFR for 2 or 6 h; SAR 10 W/kg	Many genes apoptosis-related genes were affected. Apoptosis-related genes were among the upregulated ones and the cell cycle genes among the downregulated ones.
*Li et al. (2001)	Murine C3H 10T(1/2) fibroblasts exposed to 847.74 MHz code-division multiple access (CDMA) and 835.62 frequency-division	No significant effect on DNA single strand breaks (Comet assay).

	multiple access (FDMA) RFR for 2, 4, or 24 h; SAR 3.2 - 5.1 W/kg	
Li et al. (2018)	Mouse spermatocyte-derived cells (GC-2) were exposed to 1800-MHz RFR for 24 h, SAR 1, 2 or 4 W/kg	No effect on DNA double strand break, increased DNA single strand breaks (Comet assay); free radicals involved.
Li et al. (2020)	Pregnant female rats exposed to 1800 (1 mW/cm ²) and 2400 (0.1 mW/cm ²) MHz RFR during the 21st day of pregnancy (8 pm- 8 am). Offspring tested from 3-9 weeks postnatal	Up- and down-regulation expressions of different forms (NR1, NR2A, NR2B, NR2C, NR2D, NR3A, NR3B) of methyl-D-aspartate receptors (NMDARs) in the hippocampus were observed; animals showed behavioral and cognitive development effects which may be associated with altered mRNA expression of NMDARs.
Lin et al. (2016)	Budding yeast exposed to 2-GHz RFR for 96 h, SAR 0.12 W/kg	Upregulation of the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle.
Liu et al. (2013a)	Mouse spermatocyte-derived GC-2 cell line exposed to 1800-MHz Global System for Mobile Communication (GSM) signals (5 min on and 10 min off) for 24 h; SAR 1, 2, or 4 W/kg	Increased DNA single strand breaks (comet assay) and DNA adduct 8-oxoguanine at SAR of 4 W/kg; increased reactive oxygen species generation.
Liu et al. (2013b)	Mouse spermatocyte-derived GC-2 cell line was exposed to a commercial mobile phone handset once every 20 minutes in standby, listen, dialed or dialing modes for 24 h; power density 0.0059-0.0122 mW/cm ²	Increased DNA single strand breaks (Comet assay) (attenuated by melatonin).
Lixia et al. (2006)	Human lens epithelial cells exposed to GSM-1.8 GHz RFR for 2 h, SAR 1, 2, 3 W/kg	Increased DNA single strand breaks (comet assay) at 3 W/kg at 0 and 30 min post-exposure; Increased mRNA and protein expression of Hsp70.
López-Martín et al. (2009)	Picrotoxin-pretreated male Sprague-Dawley rats exposed to 900-MHz GSM-modulated or unmodulated RFR for 2	Increased c-fos expression in brain areas.

	h, SAR modulated RFR 0.03 W/kg average– peak 0.14 W/kg in brain; unmodulated RFR average 0.26 W/kg- peak 1.4 w/kg in brain	
Luukkonen et al. (2009)	Human SH-SY5Y neuroblastoma cells exposed to 872-MHz (CW and GSM) RFR for 1 h; SAR 5 W/kg	CW RFR increased DNA single strand breaks (Comet assay) and reactive oxygen species in cells treated with menadione (a chemical that induces intracellular ROS production and DNA damage) compared to cells treated with menadione alone. GSM- modulated RFR had no significant effect.
*Luukkonen et al. (2010)	Human SH-SY5Y neuroblastoma cells exposed to 872-MHz (CW and GSM) RFR for 3 h (DNA damage) and 1 h (reactive oxygen species) ; SAR 5 W/kg	CW and modulated RFR had no significant effect on DNA single strand breaks (Comet assay) and reactive oxygen species production in cells treated with ferrous chloride,
Maes et al (1993)	Human peripheral blood lymphocytes exposed to pulsed 2450-MHz RFR for 30 or 120 min, SAR 75 W/kg	Increase in the frequency of chromosome aberrations (including dicentric chromosomes and acentric fragments) and micronuclei.
Maes et al (1996)	Human whole blood samples exposed to GSM 954- MHz emitting antenna for 2 h, SAR 1.5 W/kg, some samples also incubated with mitomycin C after exposure	Synergistic effect between RFR and mitomycin C was observed the frequencies of sister chromatid exchanges in metaphase figures.
Maes et al. (1995)	Human whole blood cells exposed to 954 MHz RFR from an antenna for 2 h; SAR 1.5 W/kg. Blood from maintenance workers of transmission antenna (450, 900 MHz) exposed at least 1 h/day for a year.	Increased chromosome aberration (dicentric chromosome) in lymphocytes. No effect found in blood of antenna maintenance workers.
*Maes et al. (1997)	Human whole blood cells exposed to 935.2 MHz RFR alone and in	No significant effects of RFR on chromosome aberration, sister chromatid exchange, and DNA single strand breaks

	combination with mitomycin C for 2 h; SAR 0.3-0.4 W/kg	(comet assay). No synergistic effect with mitomycin C.
*Maes et al (2000)	Human lymphocytes exposed to 455.7 MHz RFR from antenna of a car phone for 2 h; SAR 6.5 W/kg	No significant effects of RFR on chromosome aberration and sister chromatid exchange. No synergistic effect with mitomycin C.
*Maes et al (2001)	Human lymphocytes exposed to 900-MHz RFR for 2 h, SAR 0-10 W/kg	No significant effects of RFR on chromosome aberration and sister chromatid exchange. No synergistic effect with mitomycin C.
*Maes et al (2006)	Peripheral blood lymphocytes from subjects who were professionally exposed to cell phone RFR	No evidence of RFR-induced genetic effects: DNA single strand breaks (Comet assay), chromosome aberration, and sister chromatid exchange.
*Malini (2017)	Blood and semen samples from subjects who used cellular phones for 1-5, 6-10, and >10h/day.	No DNA damages (ladder assay) and oxidative changes observed.
*Malyapa et al. (1997a)	U87MG and C3H 10T1/2 cells exposed to 2450-MHz continuous-wave RFR for 2 h; SAR 0.7 and 1.9 W/kg	No significant effects on DNA single strand breaks (Comet assay).
*Malyapa et al. (1997b)	Mouse C3H 10T1/2 fibroblasts and human glioblastoma U87MG cells exposed to 835.62 MHz (FMCW) and 847.74 MHz (CDMA) RFR up to 24 h; SAR 0.6 W/kg	No significant effects on DNA single strand breaks (Comet assay).
*Malyapa et al. (1998)	Male Sprague-Dawley rats exposed to 2450 MHz continuous-wave (CW) RFR for 2 h; SAR 1.2 W/kg	No significant effects on DNA single strand breaks (Comet assay) in cerebral cortex or hippocampus.
Manti et al. (2017)	Four days-old adult female flies (<i>Drosophila melanogaster</i>) exposed to GSM-1800 talk mode RFR emitted by a commercial cellular	168 genes were differentially expressed associated with multiple and critical biological processes, such as basic metabolism and cellular subroutines related to stress response and apoptotic death. Free radicals may be involved.

	phone for 30 min; SAR 0.15 W/kg	
Manti et al. (2008)	Human peripheral blood lymphocytes exposed a UMTS 1.95 GHz signal for 24 h; SAR 0.5 and 2.0 W/kg; some samples also exposed to x-ray	X-ray induced chromosome exchange per cell was increased by RFR exposure. (RFR may either influence the repair of X-ray-induced DNA breaks or alter the cell death pathways of the damage response.)
Marinelli et al. (2004)	acute T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2-48 h, SAR 0.0035 W/kg	Increased DNA damage (DNA ladder) and activation genes involved in pro-survival signaling.
Markova et al. (2005)	Human lymphocytes exposed to 905 and 915 MHz GSM signals for 1 h. SAR 0.037 W/kg	RFR from GSM cell phone affected chromatin conformation and 53BP1/gamma-H2AX foci similar to heat shock. No significant difference between lymphocytes from healthy and electro-hypersensitive subjects.
Markova et al. (2010)	Human diploid VH-10 fibroblasts and human adipose-tissue derived mesenchymal stem cells exposed to GSM (905 MHz or 915 MHz) or UMTS (1947.4 MHz, middle channel) RFR for 1, 2, or 3 hr; SAR 0.037-0.039 W/kg	915 MHz and 1947.4 MHz signals inhibited tumor suppressor TP53 binding protein 1 (53BP1) foci that are typically formed at the sites of DNA double strand break location in both cell types. 905 MHz RFR did not inhibit 53BP1 foci in differentiated cells but in stem cells. (Inability to form DNA repair foci has been correlated to radiosensitivity, genomic instability, and other repair deficits.)
Martin et al. (2020)	Human neonatal foreskin keratinocytes (HEK-3N, HEK-1N, and NHEK-3N) and human skin keratinocytes HeCAT exposed to a 60-GHZ RFR for 3 h, Average SAR 513 W/kg and peak SAR 1233 W/kg	Different cell types showed different patterns of expression of ADAMTS6, IL7R, and NOG genes.
Mashevich et al. (2003)	Human peripheral blood lymphocytes exposed to 830 MHz RFR for 72 hr, SAR 1.6-8.8 W/kg	A linear increase in chromosome 17 aneuploidy (loss and gain of chromosome) and abnormal chromosome-17 replication were observed as a function of the SAR value, demonstrating that this radiation has a genotoxic effect.
Mazor et al. (2008)	Human lymphocytes exposed to continuous-wave 800 MHz for 72 hr;	Increased levels of aneuploidy depending on the chromosome studied as well as on the level of exposure. In chromosomes 1 and 10,

	SAR 2,9 and 4,1 W/kg	there was increased aneuploidy at the higher SAR, while for chromosomes 11 and 17, the increases were observed only for the lower SAR.
*McNamee et al. (2002a)	Human blood cultures exposed to continuous-wave 1900 MHz RFR for 2 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) in leukocytes.
*McNamee et al. (2002b)	Human blood cultures exposed to pulsed 1900 MHz RFR for 2 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) and micronucleus formation in leukocytes.
*McNamee et al. (2003)	Human blood cultures exposed to continuous-wave or pulsed 1900 MHz RFR for 24 h; SAR 0-10 W/kg	No effect on DNA single strand breaks (Comet assay) and micronucleus formation in leukocytes.
*McNamee et al. (2016)	Male C57BL/6 mice exposed to pulse-modulated or continuous-wave 1900 MHz RFR for 4 h/day for 5 consecutive days; whole body average SAR ~0.2 W/kg and ~1.4 W/kg.	No differentially expressed gene expressions were identified in various regions of the brain.
Meena et al. (2014)	Wistar rats exposed to 2.45 MHz RFR 2 h/day for 45 days; SAR 0.14 W/kg. Rats also treated with melatonin.	Increased in DNA single strand breaks (Comet assay) and oxidative stress in testicular tissue. Effects attenuated by melatonin.
Megha et al. (2015a)	Fischer rats exposed to 900 and 1800 MHz RFR for 30 days (2 h/day, 5 days/week); SAR 0.00059 and 0.00058 W/kg	Reduced levels of neurotransmitters dopamine, norepinephrine, epinephrine, and serotonin, and downregulation of mRNA of tyrosine hydroxylase and tryptophan hydroxylase (synthesizing enzymes for the transmitters) in the hippocampus.
Megha et al. (2015b)	Fischer rats exposed to 900, 1800, and 2450 MHz RFR for 60 days (2 h/day, 5 days/week); SAR 0.00059, 0.00058, and 0.00066 W/kg	Increased DNA single-strand breaks (Comet assay) in hippocampus, increased oxidative stress and pro-inflammatory cytokines (IL-2, IL-6, TNF- α , and IFN- γ)
*Meltz et al. (1990)	Mouse leukemic cells exposed to pulsed 2450	No evidence in any mutagenic action by the RFR exposure alone or interaction with

	MHz RFR for 4 h, SAR 40 W/kg	proflavin, a DNA-intercalating drug.
Mildažienė et al. (2019)	Sunflower seeds exposed to 5.28 MHz RFR for 5, 10, 15 min, 12.7 kV/m	RFR exposure induced a long-term effect on gene expression in leaves, mostly stimulating expression of proteins involved in photosynthetic processes and their regulation.
Millenbaugh et al. (2008)	Rats exposed to 35 GHz RFR at 75 mW/cm ² until colonic temperature reached 41-41°C, skin was assayed	Changes were detected in 56 genes at 6 h and 58 genes at 24 h post-exposure. Genes associated with regulation of transcription, protein folding, oxidative stress, immune response, and tissue matrix turnover were affected at both times. At 24 h, more genes related to extracellular matrix structure and chemokine activity were altered.
*Miyakoshi et al. (2002)	Human brain tumor derived M54 cells exposed to 2450 MHz RFR for 2 h; SAR 50 or 100 W/kg	No effect on DNA single strand breaks (Comet assay) observed.
*Mizuno et al. (2015)	WI38VA13 subcloned 2RA human fibroblast cells exposed to wireless power transfer (WPT) 12.5 MHz resonant frequency for 48, 96, or 144 h; SAR 21 W/kg	No effects on cell growth, cell cycle distribution, DNA single strand breaks (Comet assay), micronucleus formation, and hypoxanthine-guanine phosphoribosyltransferase (HPRT) gene mutation.
*Nakatani-Enomoto et al. (2016)	Human spermatozoa exposed to 1950 MHz Wideband Code Division Multiple Access (W-CDMA)-like RFR for 1 h; SAR 2.0 or 6.0 W/kg	No effect on percentage of 8-hydroxy-2'-deoxyguanosine positive spermatozoa.
Narasimhan and Huh (1991)	Lambdaphage DNA exposed to short pulses of RFR	Observed conformational anomalies in DNA probably resulting from single strand breaks and localized strand separations induced by RFR.
Nikolova et al. (2005)	Mouse embryonic neural progenitor stem cells exposed to 1710-MHz GSM RFR for 6 or 48 h; SAR 1.5 W/kg	Exposure for 6 h, but not for 48 h, resulted in a low and transient increase of DNA double-strand breaks and the transcript level of genes related to apoptosis and cell cycle control..
Nittby et al. (2008)	Fischer 344 rats exposed to 1800 MHz GSM RFR	Expression in cortex and hippocampus of genes connected with membrane functions.

	for 6 h; SAR whole body average 0.013 W/kg, head 0.03 W/kg	
Nylund and Leszczynski (2006)	Human endothelial cell line: EA.hy926 and EA.hy926v1 exposed to 900-MHz GSM RFR for 1 h; SAR 2.8 W/kg	Gene and protein expression were altered dependent on the cell type.
Odaci et al. (2016)	Pregnant Sprague - Dawley rats exposed to 900 MHz RFR 1 h each day during days 13 - 21 of pregnancy; SAR whole body average 0.024 W/kg	Testis and epididymis of offspring showed higher DNA oxidation and lipid peroxidation at 60 days postnatal.
Ohtani et al. (2016)	Sprague-Dawley rats exposed to wideband code division multiple access 2140 MHz RFR for 6 h or 3 or 6 h/day for 4 days, SAR 4 or 0.4 W/kg	Exposure at 4 W/kg (at 6 h/day) increased core temperature and upregulation of some stress markers, heat-shock proteins and heat-shock transcription factors family, in the cerebral cortex and cerebellum.
*Ohtani et al. (2019)	Mice exposed to 85 kHz (for charging electrical vehicles) EMF at 25.3 mT, 1 h/day for 10 days	No significant change in gene transcriptional expression in brain and liver.
*Ono et al. (2004)	Pregnant lacZ-transgenic mice exposed intermittently (10 sec On, 50 sec OFF) 16 h/day to 2450-MHz RFR from embryonic days of 0 to 15; SAR whole body average 0.71 W/kg	No significant effects on mutation frequencies at the lacZ gene in spleen, liver, brain, and testis in offspring. The RFR is not mutagenic in utero.
Ozgun et al. (2014)	Hepatocarcinoma cells exposed to intermittent (15 min ON, 15 min OFF) GSM 900- and 1800-MHz RFR for 1, 2, 3, or 4 h; SAR 2 W/kg	Cells showed irregular nuclei pattern and DNA damage (apoptosis).
Pacini et al. (2002)	Human skin fibroblasts exposed to GSM 904.2-MHz RFR for 1 h (from a cell phone); SAR 0.6 W/kg	Increased the expression of mitogenic signal transduction genes (e.g., MAP kinase kinase 3, G2/mitotic-specific cyclin G1), cell growth inhibitors (e.g., transforming growth factor-beta), and genes controlling apoptosis (e.g., bax).

Panagopoulos et al. (2007)	Flies (<i>Drosophila melanogaster</i>) exposed to either GSM 900-MHz or DCS 1800-MHz signals from a digital cell phone, for few minutes per day during the first 6 days of their adult life.	Degeneration of large numbers of egg chambers after DNA fragmentation (apoptosis) of their constituent cells, induced by both types of mobile telephony radiation.
Panagopoulos (2019)	Human peripheral blood lymphocytes exposed to UMTS signal (1900-2200 MHz) using a cell phone for 15 min	Chromatid-type aberrations (gaps and breaks) observed.
Panagopoulos (2020)	Human lymphocytes (in G2/M phase) exposed to UMTS (3G) 1920-1960 MHz RFR emitted from a smart phone on talk mode for 15 min; peak power density $92 \pm 27 \mu\text{W}/\text{cm}^2$; averaged over 6 min $29 \pm 14 \mu\text{W}/\text{cm}^2$	Chromatid-type aberrations were observed. Effect synergistic with caffeine.
Pandey et al. (2017)	Swiss albino mice exposed to 900-MHz RFR for 4 or 8 h per day for 35 days; SAR 0.0054-0.0516 W/kg	RFR exposure-induced oxidative stress causes DNA single-strand breaks (Comet assay) in germ cells, with altered cell cycle progression leading to low sperm count in mice (depolarization of mitochondrial membranes resulting in destabilized cellular redox homeostasis). Larger effect with longer exposure time, and recovery at 35 days post-exposure.
Pandey and Giri (2018)	Swiss albino mice exposed to GSM 900-MHz RFR 3h twice/day for 35 days, SAR 0.0516-0.0054W/kg	Increased DNA single strand breaks (Comet assay) and free radicals in testis and germ cells, effects attenuated by melatonin.
*Paparini et al. (2008)	Mice exposed to GSM 1800-MHz signal for 1 h; SAR whole body average 1.1 W/kg, brain 0.2 W/kg	No significant modulation in gene expression in whole brain.
Paulraj and Behari (2006)	35-day old male Wistar rats exposed 2 h/day for 35 days to 2450 MHz or 16.6 GHz RFR; SAR 1.0 and 2.01 W/kg,	Increased in DNA single strand breaks (Comet assay) in brain cells for both frequencies.

	respectively.	
Pesnya and Romanovsky (2013)	Onion (<i>Allium cepa</i>) exposed to GSM 900-MHz RFR from a cell phone for 1 h/day or 9 h/day for 3 days; incident power density 0.05 $\mu\text{W}/\text{cm}^2$	Increased the mitotic index, the frequency of mitotic and chromosome abnormalities, and the micronucleus frequency in an exposure-duration manner.
Phillips et al. (1998)	Human Molt-4 T-lymphoblastoid cells exposed to pulsed signals at cellular telephone frequencies of 813.5625 MHz (iDEN signal) and 836.55 MHz (TDMA signal) for 2 or 21 h. SAR 0.0024 and 0.024 W/Kg for iDEN and 0.0026 and 0.026 W/kg for TDMA)	Changes in DNA single strand breaks (increase and decrease depending on exposure parameters) (Comet assay) were observed.
*Port et al. (2003)	Human leukaemia cells (HL-60) exposed to pulsed (1 Hz) 400 MHz RFR for 6 min; 50 kV/m-25 times higher than the ICNIRP reference levels for occupational exposure	No significant effects on apoptosis, micronucleation, abnormal morphologies and gene expression assayed at 9, 24, 48, and 72 h post-exposure.
Qin et al. (2018)	Male mice exposed to 1800-MHz RFR 2 h/day for 32 days, SAR 0.0553 W/kg	Inhibition of testosterone synthesis might be mediated through CaMKI/ROR α signaling pathway.
Qin et al. (2019)	Mouse Leydig cells exposed to a 1800-MHz RFR for 1, 2 or 4 h, SAR 0.116 W/kg	Cells showed downregulated of testosterone synthase genes (<i>Star</i> , <i>Cyp11a1</i> , and <i>Hsd-3β</i>) and clock genes (<i>Clock</i> , <i>Bmal1</i> , and <i>Rora</i>), also reduced level of testosterone and increased oxidative stress.
*Qutob et al. (2006)	Human U87MG glioblastoma cells exposed to pulse-modulated 1900 MHz RFR for 4 h; SAR 0.1, 1.0, and 10 W/kg	No significant effect on gene expression.
Racuciu (2009)	Zea mays root tips exposed to continuous-	Increased mitotic index and chromosomal aberration frequency linear with increased

	wave 900 MHz RFR for 1 – 36 h; SAR < 1 W/kg)	exposure time.
Rago et al. (2013)	Human subjects with different daily durations of cell phone use (no use, < 2 h, 2-4 h, > 4 h) and “trouser users” and “shirt users”	>4 h daily use and “trouser users” had higher sperm DNA fragmentations.
Rammal et al. (2014)	Lycopersicon esculentum (tomato) exposed to 1250 MHz RFR for 10 days at 0.0095 mW/cm ²	Increased expression of proteinase inhibitor (Pin II) and Lycopersicon esculentum basic leucine Zipper1 (lebZIP1), two wound-plants genes.
*Regalbuto et al. (2020)	Human fibroblasts exposed to 2450 MHz continuous-wave or pulsed (1 ms square pulses, 50% duty cycle) RFR; SAR 0.7W/kg	No significant effect on γ -H2AX/53BP1 foci, differential gene expression, micronucleus formation, and cell cycle.
Remondini et al. (2006)	Six human cell types exposed to 900 and 1800 MHz RFR; three exposure systems were used, exposure time 1, 24, or 44 h, SAR 1 - 2.5 W/kg (Details in Table 1 of paper.)	Some but not all human cells reacted to RFR with an increase in expression of genes encoding ribosomal proteins and therefore up-regulating the cellular metabolism.
Romano-Spica et al. (2000)	Human hemopoietic and testicular cell types exposed to 50 MHz RFR modulated (80%) with a 16-Hz frequency for 0.5-24 h; the exposure system generates a 0.2 microT magnetic field parallel to the ground and a 60 V/m electric field orthogonal to the earth's magnetic field.	Overexpression of the proto-oncogene ets1 mRNA in Jurkat T-lymphoblastoid and Leydig TM3 cell lines only in the presence of the 16-Hz modulation.
*Ros-Lior et al. (2012)	Cells collected from cheeks of human subjects	Comparing control area with the side cell phone was placed; no significant genotoxic effect was found (DNA damage and cytokinetic defects, proliferative potential, and cell death).

*Roti-Roti et al (2001)	C3H 10T(1/2) cells exposed to 835.62 MHz FDMA or 847.74 MHz CDMA for 7 days and then one-dose X-ray followed by RFR for 42 days; SAR 0.6 W/kg	No significant effect of RFR on neoplastic transformation (induced by X-ray) was observed.
Roux et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 2-10 min at 0.0066 mW/cm ²	Increased stress-related transcripts (calmodulin, protease inhibitor and chloroplast mRNA-binding protein) in leaves. (Increased at 15 min after the end of electromagnetic stimulation, dropped to close to initial levels by 30 min, and then increased again at 60 min.)
Roux et al. (2008)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm ²	Induction of stress gene expression; similar to wound responses suggesting that the radiation is perceived by plants as an injurious stimulus.
Sagripanti and Swicord (1986)	Purified DNA solution exposed to 2.55-GHz RFR for 20min; SAR _{min} and SAR _{max} ranges: 0, 2-8-5 and 21-85 W/kg,	Structural changes in DNA suggested that exposure to RFR can cause single as well as double-strand breaks in DNA in solution.
Sagripanti et al. (1987)	Purified plasmid DNA exposed to RFR in the frequency range from 2.00 to 8.75 GHz for 20 min; SAR 0, 8.5, or 85 W/kg	Induced dose- and exposure-duration-dependent DNA single and double strand breaks depends on the presence of small amounts of cuprous ions.
Sahin et al. (2016)	Rats exposed to 3-G 2100 MH RFR 6 h/day for 10 or 40 days	Oxidative DNA damage (8-hydroxy-2'deoxyguanosine) in brain increased after 10-day exposure but decreased after 40 day exposure.
Said-Salman et al. (2019)	Escherichia coli K-12 DH5 α exposed to 2.4 GHz RFR for 5 h	Expression of 101 genes was differentially affects (up- and down-regulation).
*Sakuma et al. (2006)	Human glioblastoma A172 cells exposed to W-CDMA 2.1426 GHz radiation at SARs of 80, 250, and 800 mW/kg and CW radiation at 0.08 W/kg for 2 and 24 h; normal human IMR-90 fibroblasts from fetal lungs exposed to W-	No significant effect on DNA single strand breaks (Comet assay).

	CDMA and CW radiations at a SAR of 0.08 W/kg for 2 and 24 h.	
*Sakurai et al. (2011)	Human glial cell line, SVGp12, exposed to continuous-wave 2450 MHz RFR for 1, 4, and 24 h; SAR 1, 5, and 10 W/kg	No evidence of effect on gene expression.
*Salmen et al. (2018)	<i>S. aureus</i> , <i>S. epidermidis</i> , and <i>P. aeruginosa</i> . Exposed to exposed to 900 and 1800 MHz RFR for 2 h using a cell phone	No significant effects on DNA, growth rate and antibiotic susceptibility.
*Sannino et al. (2006)	Human blood leukocytes exposed to UMTS-1950 MHz signal for 24 h; SAR 0.5 or 2 W/kg	No effect on DNA single strand breaks (Comet assay) and cell viability.
*Sannino et al. (2009a)	Human dermal fibroblasts from a healthy subject and from a subject affected by Turner's syndrome exposed to GSM 900 MHz.RFR for 24 h; SAR 1 W/kg	No significant effect on DNA single strand breaks (Comet assay)
*Sannino et al. (2009b)	Human dermal fibroblasts from one subject exposed to 900 MHz RFR for 24 h; SAR 1 W/kg	No significant effect on DNA single strand breaks (Comet assay) and micronucleus frequency.
Sannino et al. (2011)	Phytohemagglutinin activated human blood lymphocytes exposed to a 900-MHz RFR for 20 h; SAR 1.25 W/kg, and then to mitomycin C	RFR attenuated micronucleus induced by mitomycin c at S-phase, and not at G(0)- and G(1)-phases of the cell cycle. (Adaptive response)
Sannino et al. (2014)	Phytohemagglutinin activated human blood lymphocytes exposed to a 900-MHz RFR for 20 h; SAR 0.3 W/kg, and then to x-ray	RFR attenuated micronucleus induced by x-ray.
Sannino et al. (2017)	Chinese hamster lung fibroblasts exposed to 1950 MHz, Universal	Increased micronucleus frequency at 0.15 and 0.3 W/kg, no effect at 0.6 and 1.25 W/kg; attenuated micronucleus induced by

	Mobile Telecommunication System signal for 20 h; SAR 0.15 – 1.25 W/kg	mitomycin-C at 1.25 W/kg.
Sarimov et al. (2004)	Human lymphocytes exposed to GSM 895-915 MHz signals for 30 min; SAR 0.0054 W/kg	Condensation of chromatin was observed. (Stronger effect at 1 h exposure.)
Sarkar et al. (1994)	Mice exposed to 2450 MHz RFR 2 h/day for 120, 150, and 200 days; SAR 1.18 W/kg	Rearrangements of DNA segments were observed in brain and testis.
Scarfi et al (1996)	Bovine lymphocytes exposed to 9 GHz RFR for 10 min, SAR 70 W/kg	Increased micronucleus frequency.
*Scarfi et al. (2003)	Human peripheral blood lymphocytes exposed to pulsed 120-130 GHz (pulse rate 2 Hz, pulsed duration 4 μ s) field for 20 min; delivered energy 1.2 and 0.72 J for the two frequencies, respectively.	No effect on micronucleus frequency and cell proliferation.
*Scarfi et al (2006)	Human lymphocytes exposed to GSM 900 MHz RFR for 24 h, SAR 1, 5, and 10 W/kg).	The results provided no evidence for the existence of genotoxic (micronucleus) or cytotoxic effects
* Schuermann et al. (2020)	Human MRC-5 lung fibroblasts, human osteosarcoma cells, HTR-8/SVneo human trophoblasts, and GFP-tagged XRcc1 cells exposed to intermittent (5/10 min ON/FF) or continuous 1950 MHz, 2450 MHz (GSM or unmodulated) RFR for 1-24 h; SAR 0.5-4.9 W/kg.	No significant effect on DNA single strand breaks (Comet assay).
Schwarz et al. (2008)	Human fibroblasts and lymphocytes exposed to UMTS 1950 MHz RFR for 4-48 h; SAR 0.05 to 2.0 W/kg	Increased DNA single strand breaks (comet assay) and micronucleus were observed in fibroblasts but not in lymphocytes either unstimulated or stimulated with phytohemagglutinin.

Sekeroğlu et al. (2012)	Immature (2 week old) and mature (10 weeks old) Wistar rats exposed to continuous-wave 1800 MHz RFR for 2 h/days for 45 days; SAR 0.38-0.78 W/kg (immature rats), 0.31-0.52 W/kg (mature rats)	Bone marrow cells showed chromosome aberrations, micronucleus frequency, mitotic index and ratio of polychromatic erythrocytes (PCEs) in all exposed groups. Immature group showed more effect and less recovery at day 15 post-exposure. The cytogenotoxic damage in immature rats was statistically higher than the mature rats.
Sekeroglu et al. (2013)	Immature and mature rats exposed to 900 MHz RFR for 2 h/days for 45 days; SAR immature rats, 0.38-0.78 W/kg; mature rats 0.31-0.52 W/kg	Bone marrow cells showed chromosome aberrations, increases in micronucleus frequency, mitotic index, and ratio of polychromatic erythrocytes. Effects persisted for 15 days after exposure.
*Sekijima et al. (2010)	Human A172 (glioblastoma), H4 (neuroglioma), and IMR-90 (fibroblasts from normal fetal lung) cells exposed to continuous-wave and W-CDMA 2.1425 GHz RFR up to 96 h; SAR 0.08, 0.25, 0.8 W/kg	No significant effects on gene expression and cell proliferation.
Semin et al. (1995)	DNA in glycine and formaldehyde exposed to 10 different 4 to 8 GHz RFR 25 ms pulses, 1-6-Hz repetition rate, 0.4 to 0.7 mW/cm ² peak power density	3 or 4 Hz pulses and 0.6 mW/cm ² peak power increased the accumulated damage to the DNA secondary structure. However, changing the pulse repetition rate to 1, 5, 6 Hz, as well as changing the peak power to 0.4 or 0.7 mW/cm ² had no effect (“window effect”).
*Senturk et al. (2019)	Lymphocytes from patients received radiofrequency treatment on inferior turbinate as they were diagnosed with inferior turbinate hypertrophy	No significant effect on DNA single strand breaks (Comet assay) on Day 15 post-treatment. Increase in oxidative stress was observed.
Shah et al. (2015)	Human blood samples exposed to 916-MHz RFR at two power densities and 1-8 hr using an antenna	Chromosomal damage observed in lymphocytes at higher power density and longer exposure duration.

Shahin et al. (2013)	Female mice (<i>Mus musculus</i>) exposed to continuous-wave 2.45 GHz RFR 2 h/day for 45v days; SAR 0.023 W/kg	Increased DNA strand breaks (Comet assay) observed in the brain. Changes in oxidative mechanisms and oxidative stress were observed in liver, kidney and ovary. Increased embryo implantation/resorption and abnormal pregnancy were observed.
Shahin et al. (2019)	Male Wistar rats exposed to 900 MHz RFR for 2 h/day for 8 weeks, SAR 1.075 W/kg	Increased DNA single strand breaks (Comet assay) in testis and increased oxidative stress.
Sharma ad Shukla (2020)	Male Wistar rats exposed to 900 MHz RFR for 1, 2, or 4 h/day for 90 days; SAR brain 0.231 W/kg	Increased DNA single strand breaks (Comet assay) and increased oxidative stress in brain.
Shckorbatov et al. (2009)	Human buccal epithelium cells exposed to 35 GHz RFR for 10 sec; SAR 0.75 W/kg	Caused condensation of chromatin. Left circularly polarised radiation induced less effect than linearly polarised radiation. Cell membrane damage observed.
Shckorbatov et al. (2010)	Human fibroblasts exposed to 36.65 GHz RFR at incident power densities of 1, 10, 30 and 100 microW/cm ² for 10 sec	Chromosome condensation observed at 10 and 100 μW/cm ² exposure. Right-handed elliptically polarized radiation was more biological activity than the left-handed polarized one.
*Shi et al (2014)	Cultured human lens epithelial cells (HLECs) exposed to 90 kHz magnetic field for 2 and 4 h; 93.36 μT	No significant effects on DNA single strand break (comet assay) and double strand breaks.
*Silva et al. (2016)	Human primary thyroid cells exposed to 895 and 900 MHz RFR for 3-65 h, SAR 0.082-0.170 W/kg	No effect on expressions of Ki-67 (involved in cell proliferation) p53 (tumor suppression) HSP-70 (stress biomarker), and reactive oxygen species.
Smith-Roe et al. (2020)	Male and female Hsd:Sprague Dawley rats and B6C3F1/N mice exposed from Gestation day 5 or Postnatal day 35, respectively, to code division multiple access (CDMA) or global system for mobile modulations over 18 hr/day, at 10-min	Significant increases in DNA single strand breaks (Comet assay) observed in the frontal cortex of male mice (both modulations), leukocytes of female mice (CDMA only), and hippocampus of male rats (CDMA only). No significant increases in micronucleated red blood cells were observed in rats or mice.

	intervals for 19 (rats) or 14 (mice) weeks; SAR 1.5, 3, or 6 W/kg (rats, 900 MHz) or 2.5, 5, or 10 W/kg (mice, 1,900 MHz).	
Sokolovic et al. (2015)	Wistar rats exposed to RFR (4 h/day, for 20, 40, and 60 days) from a Nokia 3110 cell phone: SAR 0.043-0.135 W/kg; some rats treated with melatonin (2 mg/kg, ip)	Melatonin reduced DNA fragmentation in testicular tissues also reversed oxidative changes caused by RFR (malondialdehyde, xanthine oxidase, and acid-DNase)
Soubere Mahamoud et al. (2016)	Human keratinocyte exposed to a 60.4-GHz RFR at an incident power density of 20 mW/cm ² for 3 hours	No keratinocyte transcriptome modifications were observed. Co-treatment with a glycolysis inhibitor slightly alter the transcriptome of 6 genes encoding transcription factors or inhibitors of cytokine pathways. Thus, the RFR exposure may affect metabolically stressed cells
Souza et al. (2014)	Exfoliated cells from the oral epithelium from human subjects who spent different time using cell phones (group I, t > 5 h; group II, t > 1 h and ≤ 5 h; and group III, t ≤ 1 h).	Structures that may be associated with gene amplification were significantly greater in the individuals in group I. No significant effects on micronucleus frequency and apoptosis and necrosis were observed.
*Speit et al. (2007)	Human fibroblasts (ES1 cells) and Chinese hamster cells (V79) exposed to intermittent (5 min ON/10 min OFF) 1800-MHz for 1, 4, 24 h; RFR; SAR 2 W/kg	No significant effects on DNA single strand break (Comet assay) and micronucleus frequency.
*Speit et al. (2013)	Human HL-60 exposed to intermittent (5 min ON/10 min OFF) 1800 MHz RFR for 24 r; SAR 1.3 W.kg	No significant effects on DNA single strand break (Comet assay) and micronucleus frequency.
*Stronati et al. (2006)	Human blood samples exposed to GSM 935-MHz signal for 24h; SAR 1 and 2 W/kg	Lymphocytes showed no changes in DNA single strand breaks (Comet assay), chromosomal aberrations, sister chromatid exchanges, micronuclei frequency and cell cycle. No significant interaction with x-ray.
*Su et al (2017)	Neurogenic A172, U251, and SH-SY5Y cells	No significant DNA damage (γH2AX foci)

	exposed to an intermittently (5 min ON/10 min OFF) 1800 MHz RFR at SAR of 4.0 W/kg for 1, 6, or 24 h.	
*Su et al. (2018)	Primary cultured astrocytes, microglia and cortical neurons were exposed to intermittent (5 min ON/10 min OFF) GSM 1800 MHz RFR for 1, 6 or 24 h; SAR 4.0 W/kg.	The RFR did not elicit DNA double strand breaks (γ H2AX foci) but inhibited the phagocytic ability of microglia and the axon branch length and branch number of cortical neurons.
Sun C. et al. (2016)	Mouse embryonic fibroblasts (MEFs) with proficient ($Atm^{+/+}$) or deficient ($Atm^{-/-}$) ataxia telangiectasia mutated, which is critical to initiation of DNA repair, to GSM 1800-MHz RFR for 1, 12, 24, or 36 h; SAR 4 W/kg.	Increased DNA single-strand breaks (SSBs) (Comet assay) and activated the SSB repair mechanism. This effect reduced the DNA damage to less than that of the background level after 36 hours of exposure. In the $Atm^{-/-}$ MEFs, the same RF-EMF exposure for 12 h induced both DNA single and double-strand breaks (Comet assay) and activated the two repair processes, which also reduced the DNA damage to less than the control level after prolonged exposure. (compensatory effects) (Conclusion from interpretation of different results from ($Atm^{+/+}$) and ($Atm^{-/-}$) cells.
Sun, LX et al. (2006a)	Human lens epithelial cells exposed to 217 Hz-modulated 1800 MHz RFR for 2 h; SAR 1, 2, 3, 4 W/kg	No or repairable DNA single strand breaks (Comet assay) was observed after 2 hour irradiation of 1.8 GHz microwave on LECs when SAR \leq 3 W/kg. The DNA damages caused by 4 W/kg irradiation were irreversible.
Sun, LX et al. (2006b)	Human lens epithelial cells exposed to 217 Hz-modulated 1800 MHz RFR for 2 h; SAR 1, 2, 3, 4 W/kg	No DNA single strand breaks (comet assay) was induced using comet assay after 2 hours irradiation of 1.8 GHz microwave on hLECs at the dose SAR \leq 3.0 W/kg. 4.0 W/kg irradiation caused significantly DNA damage and inhibition of hLECs proliferation.
Sun Y. et al. (2017)	HL-60 cells from human leukemia exposed to a 900-MHz RFR for 4 h/day for 5 days, Peak and average SAR 4.1x	Increased oxidative DNA damage, decreased mitochondrial transcription, and increased oxidative stress.

	10^{-4} and 2.5×10^{-4} W/kg	
Sykes et al. (2001)	pKZ1 mice exposed daily for 30 min to 217-Hz modulated 900 MHz RFR 1, 5, or 25 days; SAR 4 W/kg	After 25 days of exposure, RFR could lead to a perturbation in recombination frequency which may have implications for recombination repair of DNA.
*Takahashi et al. (2002)	Male Big Blue mice (BBM) exposed to 1.5 GHz RFR in the head region for 90 min/day, 5 days/week, for 4 weeks; SAR 0.67 and 2 W/kg	There was no significant variation in the frequency of independent mutations of the lacItrans gene and deletion mutation in the brain.
Tice et al. (2002)	Human blood leukocytes and lymphocytes exposed to voice modulated 837 MHz produced by an analog signal generator or by a time division multiple access (TDMA) cellular telephone, 837 MHz generated by a code division multiple access (CDMA) cellular telephone (not voice modulated), and voice modulated 1909.8 MHz generated by a global system of mobile communication (GSM)-type personal communication systems (PCS) cellular telephone for 3 or 24 h, SAR 1-10 W/kg	No significant effect on DNA single strand break (Comet assay). Exposure to each of the four RF signal technologies for 24 h at an average SAR of 5.0 or 10.0 W/kg resulted in a significant and reproducible increase in the frequency of micronucleated lymphocytes.
Tiwari et al. (2008)	Blood samples from male human subjects exposed to a CDMA cell phone for 1 h	In vitro exposure to RFR induces reversible DNA single strand breaks (Comet assay) in synergism with aphidicolin, a DNA repair inhibitor,
Tkalec et al. (2009)	Allium cepa L root meristematic cells from seeds exposed to 400 and 900 MHz RFR for 2 h, power density 10, 23, 41 and 120 V/m).	Lagging chromosomes, vagrants, disturbed anaphases and chromosome stickiness were observed.

Tkalec et al. (2013)	Earthworm (<i>Eisenia fetida</i>) exposed to continuous-wave and AM-modulated 900-MHz RFR for 2 - 4 h; SAR 0.00013, 0.00035, 0.0011, and 0.00933 W/kg	Increased DNA single strand breaks (Comet assay) in earthworms coelomocytes and oxidative stress (lipid and protein oxidation)
Tohidi et al. (2020)	Male BALB/c mice exposed to RFR from a cell phone jammer that emits 900- and 1800 MHz CDMA and GSM signals) for 0.5, 1, 2, or 4 h twice a day for 30 days.	Apoptotic genes Bax and Bcl2 expression in the hippocampus were upregulated for 1- and 2-h exposures and down-regulated with longer exposure.
*Tomruk et al. (2010)	Nonpregnant and pregnant New Zealand White rabbits exposed to GSM 1800 MHz RFR 15 min/day for a week	No oxidative damage in liver of exposed adult and offspring, increased lipid peroxidation.
Trivino Pardo et al (2012)	T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2 or 48 h; SAR 9.0035 W/kg	Changes in gene expressions (e.g., an early activation of genes involved in DNA double- and single-strand breaks repair).
Trosic (2001)	Rats exposed to 2450 MHz RFR for 2, 8, 13 and 22 irradiation treatments of two hours each; power density 5-15 mW/cm ² , SAR 20 W/kg	Increased multinucleated alveolar macrophages- the elevation of the number of nuclei per cell was exposure time- and dose-dependent.
Trosic and Busljeta (2005)	Wistar rats exposed to continuous-wave 2450 MHz RFR 2 h/day 7 days /week for a total of 4, 16, 30, and 60 h. power density 5-10 mW/cm ² SAR 1-2 W/kg	The frequency of micronucleated bone marrow erythrocytes was significantly increased after 15 irradiation treatments. No effect after 2, 8, and 30 exposure treatments.
Trosic and Busljeta (2006)	Rats exposed to 2450 MHz RFR 2 h/day, 7 days/week; SAR 1.24 W/kg	Bone marrow cell micronucleus frequency increased on experimental day 15, and micronucleated polychromatic erythrocytes in peripheral blood increased on day 8.
Trosic et al. (2002)	Male Wistar rats exposed for 2 h/day, 7 days a week for up to 30 days to continuous-wave 2450	Increased micronuclei in peripheral blood polychromatic erythrocytes on the 2nd, 8 th , and 15 th day of exposure. It is likely that an adaptive mechanism, both in

	MHz RFR; power density 5-10 mW/cm ² SAR 1-2 W/kg	erythrocytopoiesis and genotoxicity occurred.
Trosic et al. (2004)	Male Wistar rats exposed for 2 h/day, 7 days/week for 4, 16, 30, and 60 h to continuous-wave 2450 MHz RFR; power density 5-10 mW/cm ² SAR 1.25 W/kg	The frequency of micronucleated polychromatic erythrocytes in bone marrow was significantly increased on experimental day 15, but not on 2, 8, and 30 days.
Trosic et al. (2011)	Male Wistar rats exposed to GSM 915 MHz RFR for 1 h /day 7 days/week for 2 weeks; SAR 0.6 W/kg	Increased DNA single strand breaks (Comet assay) in brain, renal, and liver cells.
Tsybulin et al. (2013)	Japanese Quail embryos exposed in ovo to GSM 900 MHz signal from a cell phone intermittently (48 sec ON/12 sec OFF) during initial 38 h of brooding or for 158 h (120 h before brooding plus initial 38 h of brooding): SAR 0.000003 W/kg	The lower duration of exposure led to a significant decrease in DNA single strand breaks (Comet assay) in cells of 38-h embryos, while the higher duration of exposure resulted in a significant increase in DNA damage.
Usikalu et al., (2013)	Sprague-Dawley rats exposed to 2450 MHz RFR for 10 min: SAR 0-4.3 W/kg	Increased DNA single strand breaks (Comet assay) found in ovary and testis.
Vafaei et al. (2020)	Pregnant mice exposed to 2400 MHz RFR from a D-link Wi-Fi router from 5 days after mating to 1 day before delivery for 2-4 h/day, head SAR at 30 cm from router 0.09 W/kg	Placenta tissue showed increased superoxide dismutase mRNA, CDKN1A, and Gadd 45a expression. (CDKN1A, and Gadd 45a are involved in DNA repair, cell cycle arrest, apoptosis, and cellular responses to environmental stressors.) Also, increased BAX mRNA and decreased Bcl-2 mRNA leads to apoptosis.
*Valbonesi et al. (2008)	Human trophoblast cell line HTR-8/SVneo exposed to pulsed 1817 MHz RFR or 1 h; SAR 2 W/kg	No significant change in either HSP70 or HSC70 protein or gene expression, or DNA single strand breaks (Comet assay).
Valbonesi et al. (2014)	Rat PC12 cells exposed to continuous-wave 1.8 GHz RFR or GSM-	After PC12 cells exposure to the GSM-217 Hz signal for 16 or 24 h, HSP70 mRNA transcription significantly increased, whereas

	217Hz and GSM-Talk signals for 4, 6, or 24 h, SAR 2W/kg	no effect was observed in cells exposed to the CW or GSM-Talk signals.
*Valbonesi et al. (2016)	Rat PC12 cells exposed to 1.8 GHz 217-GSM signal for 24 h. SAR 2 W/kg	Acetylcholine esterase transcriptional or translational pathways not affected, whereas acetylcholine esterase enzymatic activity increased.
Vanishree et al. (2018)	Buccal cells from low and high cellular phone users	There was a significant increase in micronucleus counts in subject who use the phone longer. There was highly significant difference in the mean micronucleus count of participants using (code division multiple access) CDMA than (global system for mobiles) GSM cellular phones.
Varghese et al. (2018)	Female Sprague-Dawley rats exposure 2450 MHz RFR, 4/day. For 45 days; SAR 0.23W/kg	Increased caspase-3 gene expression in brain tissues; decreased antioxidant enzymes and increased lipid preoxidation. Rat showed lowering of learning and memory and expression of anxiety behavior.
Veerachari and Vasani (2012)	Human elected semen exposed to a 900-GSM cellular phone in talk mode for 1 h; power density 1-40 $\mu\text{W}/\text{cm}^2$ at 2.5 cm from antenna.	Increased DNA fragmentation index and reactive oxygen species, and decreased sperm motility and viability.
*Verschaeve et al. (2006)	Female rats exposed to RF fields for 2 h per day, 5 days per week for 2 years; SAR 0.3 or 0.9 W/kg. the mutagen and carcinogen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) was given in the drinking water. at a concentration of 19 $\mu\text{g}/\text{ml}$.	No significant genotoxic activity of MX in blood and liver cells measured by micronucleus and DNA single strand breaks (comet assay). However, MX induced DNA damage in rat brain. Co-exposures to MX and RF radiation did not significantly increase the response of blood, liver and brain cells. (no data on RFR alone.)
Vian et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of mRNA encoding the stress-related bZIP transcription factor.(3.5 folds at 5-15 min post-exposure)
Vijayalaxmi et al. (1997a)	C3H/HeJ mice exposed to for 20 h/day, 7 day to continuous-wave 2450 MHz RFR MHz for 20 h/day. 7 days/week, over	Significant increases in micronucleus formation in peripheral blood and bone marrow cells were observed.

	18 months: SAR 1.0 W/kg	
Vijayalaxmi et al. (1997b)	Human peripheral blood exposed to 2450 MHz RFR either continuously for 90 min or intermittently (30 min on and 30 min off, repeated three times); SAR 12.46 W/kg	No effect on several genotoxic indexes including chromosome damage, exchange aberrations, and micronucleus frequency.
*Vijayalaxmi et al. (1999)	CF-1 male mice exposed to ultra-wideband electromagnetic radiation (UWBR) for 15 min; SAR 0.037 W/kg	No significant effects on micronucleus frequency and polychromatic erythrocytes in peripheral blood and bone marrow cells at 16 and 24 h post-exposure.
*Vijayalaxmi et al. (2000)	3 human peripheral blood samples exposed to pulsed 2450-MHz RFR for 2 h; SAR 2.135 W/kg	No significant effect on DNA single strand breaks (Comet assay) was observed in lymphocytes immediately and at 4 h post-exposure.
*Vijayalaxmi et al. (2001a)	4 human peripheral blood samples exposed to 835.62 MHz (FDMA) RFR for 24 h, SAR 4.4 or 5.0 W/kg	Lymphocytes were stimulated with a mitogen, phytohemagglutinin. No significant effects at 48 and 72 h post-exposure in mitotic indices, incidence of exchange aberrations, excess fragments, binucleate cells, and micronucleus frequency.
*Vijayalaxmi et al. (2001b)	Male Sprague-Dawley rats exposed to continuous-wave 2450 MHz RFR for 24 h; SAR 12 W/kg	Peripheral blood and bone marrow smears showed no effects on frequency of micronuclei in polychromatic erythrocytes at 24 h post-exposure.
*Vijayalaxmi et al. (2001c)	4 human peripheral blood samples exposed to continuous-wave 847.74 MHz (CDMA) RFR for 24 h; SAR 4.9 or 5.5 W/kg	No significant effects on mitotic indices, frequencies of exchange aberrations, excess fragments, binucleate cells, and micronuclei in lymphocytes at 48 and 72 h post-exposure.
*Vijayalaxmi et al. (2003)	Timed-pregnant Fischer 344 rats (from nineteenth day of gestation) and their nursing offspring (until weaning) exposed to a far-field 1.6 GHz Iridium wireless communication signal for 2 h/day, 7 days/week	No significant effects on micronuclei in polychromatic erythrocytes in bone marrow.

	for 2 years; SAR 0.036 to 0.077 W/kg	
*Vijayalaxmi et al. (2004)	Mice exposed to 42.2 GHz RFR applied to the nasal region 30 min/day for 3 days; peak SAR 622 W/kg	No effect on micronucleus frequency in polychromatic erythrocytes of peripheral blood and bone marrow cells collected 24 h after exposure.
*Vijayalaxmi et al. (2006)	Human peripheral blood samples exposed to 2.45 GHz or 8.2 GHz pulsed-wave RFR for 2 h; SAR 2.13 W/kg (245 MHz) or 20.71 W/kg (8.2 GHz),	No significant effects on chromosomal aberrations and micronuclei in lymphocytes.
Vilic et al. (2017)	Honey bee (<i>Apis mellifera</i>) larvae exposed to 900 MHz at 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.	DNA single strand break (Comet assay) increased significantly in honey bee larvae exposed to modulated (80% AM 1 kHz sinus) field at 23 V m ⁻¹ . Oxidative changes also observed. Modulated RF-EMF produced more negative effects than the corresponding unmodulated field.
*Waldmann et al. (2013)	Human peripheral blood samples exposed to GSM 1800 MHz RFR for 28 h; SAR 0.2, 2, and 10 W/kg	No significant effects on lymphocytes on chromosome aberration, micronucleus frequency, sister chromatid exchange and DNA single strand break (comet assay).
Wang et al. (2015)	Neuro-2a (mouse neuroblastoma) cells exposed to GSM 900 MHz RFR for 24 h; SAR 0.5, 1 or 2 W/kg	Increased DNA oxidative damage (comet assay) and reactive oxygen species. OGG1(a base excision DNA repair enzyme) may be involved.
Wu et al. (2008)	Human lens epithelial cells exposed to 1800 MHz mobile phone radiation for 24 h; SAR 4 W/kg	Increased DNA single strand breaks (Comet assay) and reactive oxygen species.
Xu et al. (2010)	Sprague-Dawley rat primary cultured cortical neurons exposed to intermittent (5 min ON/10 min OFF) 217-Hz pulsed 1800 MHz RFR for 24 h; SAR 2 W/kg	Increased in the levels of 8-hydroxyguanine, a common biomarker of DNA oxidative damage, in the mitochondria of neurons, levels of mitochondrial RNA (mtRNA) transcripts showed a reduction.
Xu et al. (2013)	Six different types of cells intermittently (5	RFR induced DNA damage (γ H2AX foci and alkaline and neutral comet assay) in a

	min ON/10 min OFF) exposed to pulsed GSM 1800 MHz RFR for 1 or 24 h: SAR 3.0 W/kg	cell type-dependent manner.
Yadav and Shama (2008)	Buccal-mucosa cells from 85 regular cell phone users (exposed) and 24 non-users (controls)	A positive correlation between 0-1, 1-2, 2-3 and 3-4 years of exposure and the frequency of micronucleated cells and total micronuclei.
Yakymenko et al. (2018)	Quail embryos exposed to GSM 1800 GHz signal from a smart phone (48 s ON/12 s OFF) for 5 days before and 14 days during incubation, power density 0.00032 mW/cm ²	Increased DNA single strand breaks (comet assay), oxidative DNA damage, reactive oxygen species, and mortality.
Yan et al. (2008)	Adult Sprague-Dawley rats exposed to a cell phones 1.9 GHz (PCE CDMA) for 6 h per day for 126 days (18 weeks).	Significant mRNA up-regulation of injury- related proteins in the brain of rats exposed to cell phone radiation
Yao et al. (2004)	Rabbit lens epithelial cells exposed to continuous-wave 2450- MHz RFR for 8 h, power densities 0.10, 0.25, 0.50, 1.00, and 2.00 mW/cm ²	The RFR higher than 0.50 mW/cm ² can inhibit lens epithelial cell proliferation, and increase the expression of P27Kip1.
Yao et al. (2008)	Human lens epithelial cells intermittently (5 min ON/10 min OFF) exposed to GSM 1.8 GHz RFR for 2 h; SAR 1, 2, 3, and 4 W/kg	Increased DNA single strand breaks (Comet assay), no change in double strand breaks (γ H2AX foci), and increased reactive oxygen species.
Ye et al. (2016)	Chicken embryos exposed to GSM 900 MHz RFR from cell phones 3 h/day from day 2 to day 21 of incubation	Increased DNA single strand breaks (Comet assay) from blood cells and mortality.
*Yildirim et al. (2010)	People who lived around cell phone base stations and healthy controls	There was no significant difference in micronucleus frequency and chromosomal aberrations in blood lymphocytes between the two study groups
Zalata et al. (2015)	Human semen samples exposed to 850-MHz	Significant increase in sperm DNA fragmentation percent, clusterin gene

	RFR from a cell phone for 1 h; SAR 1.46 W/kg at 10 cm	expression and clusterin protein (associated with clearance of cellular debris and apoptosis) levels in the exposed semen samples.
*Zeni et al. (2003)	Human peripheral blood exposed to continuous wave 925 MHz RFR or GSM 925 MHz (6 min ON/ 3 h OFF for 44h (SAR 1.6 W/kg); or GSM signal 1 h/day for 3 days (SAR 0.2 W/kg).	No statistically significant differences were detected in micronucleus frequency in lymphocytes.
*Zeni et al. (2005)	Human peripheral blood lymphocytes exposed to GSM 900 MHz signal for 2 h; SAR 0.3 and 1 W/kg	No significant effects on DNA single strand breaks (Comet assay), chromosome aberration, or sister chromatid exchange.
*Zeni et al. (2007)	Human whole blood samples exposed to 120 GHz (SAR 0.4 W/kg) and 130 GHz (SAR 0.24, 1.4, or 2 W/kg) RFR for 20 min.	No effects in leukocytes on micronucleus frequency and DNA single strand breaks (comet assay).
*Zeni et al. (2008)	Human peripheral blood exposed intermittently (6 min ON/2 h OFF) to 1945 MHz RFR for 24 – 68 h; SAR 2.2 W/kg	No significant effects on DNA single strand breaks (Comet assay) and micronucleus frequency in leukocytes.
Zeni et al. (2012a)	Human peripheral blood lymphocytes exposed to 1950-MHz RFR UMTS (universal mobile telecommunication system) signal for 20 h; SAR 1.25, 0.6, 0.3, or 0.15 W/kg. and then tomitomycin C	Cells pre-exposed to RFR at 0.3W/kg (less consistent at the other SARs) and then treated with MMC showed a significant reduction in the frequency of micronucleus, compared with the cells treated with MMC alone
*Zeni et al. (2012b)	Rat neuron-like pheochromocytoma (PC12) cells exposed to 1950-MHz 3G Universal Mobile Telecommunications System (UMTS) signal for 24 h; SAR 10 W/kg	No effect on DNA single strand break (Comet assay), cell viability, and apoptosis.

Zhang et al. (2006)	Chinese hamster lung cells exposed intermittently (5 min ON/10 min OFF) to GSM 1800 MHz RFR for 1 or 24 h; SAR 3 W/kg	Cells exposed for 24 h showed increased DNA double strand breaks (γ H2AX foci).
Zhang et al. (2002)	Human whole blood exposed to 2450 MHz RFR for 2 h; Power density 5 mW/cm ²	2450-MHz RFR cannot induce DNA and chromosome damage, but can increase DNA single strand breaks (Comet assay) induced by mitomycin C .
Zhang et al. (2008)	Primary culture of rat neurons exposed to a 1.8 GHz RFR for 24 h; SAR 2 W/kg.	Changes (up- and down-regulation) of many genes transcription (involving cytoskeleton, signal transduction pathway, metabolism, etc.) were observed.
Zhao J. et al. (2020)	Escherichia coli exposed to 3.1 THz RFR for 8 h at 33 mW/cm ² and 10 Hz repetition frequency	Plasmid copy number, protein expression and fluorescence intensity of bacteria from the irradiated area were 3.8-, 2.7-, and 3.3 times higher than in bacteria from the un-irradiated area, respectively.
Zhao R. et al. (2007)	Rat neurons exposed to pulsed 217-Hz modulated 1800 MHz RFR for 24 h; SAR 2 W/kg	up- and down-regulation of genes transcriptions were observed.
Zhao TY. et al. (2007)	Primary cultured neurons and astrocytes exposed to a GSM 1900 MHz cell phone for 2 h;	Up-regulation of caspase-2, caspase-6 and Asc (apoptosis associated speck-like protein containing a card) gene expression in neurons and astrocytes. Additionally, astrocytes showed up-regulation of the Bax gene. Neurons appeared to be more sensitive to this effect than astrocytes.
*Zhijian et al. (2009)	Leukocytes from four young healthy donors exposed intermittent (5 min ON/10 min OFF) to 1800 MHz RFR for 24 h; SAR 2 W/kg; Cell also exposed x-ray	No significant effect on DNA single strand breaks (Comet assay) and no synergistic effect with x-ray.
*Zhijian et al. (2010)	Human B-cell lymphoblastoid cells exposed to 1800 GHz RFR for 2 h; SAR 2 W/kg	RFR did not directly induce DNA single strand breaks (Comet assay)
*Ziemann et al. (2009)	Peripheral blood erythrocytes of B6C3F1	No significant effect on micronucleus frequency.

	mice exposed to GSM 900 or DCS 1747 MHz RFR 2 h/day, 5 days /week for 2 years; SAR 0.4, 1.3 and 4 W/kg	
Zong et al. (2015)	Mice exposed to 900 MHz RFR 4 h/day for 7 days; SAR 0.05 W/kg	RFR alone had no effect on DNA single strand breaks (Comet assay) and oxidative damage in blood leukocytes. It attenuated bleomycin-induced DNA breaks and repair, and oxidative damage.
Zothansiamama et al. (2017)	Blood samples from people lived closed to cell phone base station	The exposed group, residing within a perimeter of 80 m of mobile base stations, showed significantly higher frequency of micronuclei in lymphocytes when compared to the control group, residing 300 m away from the mobile base stations.
Zotti-Martelli et al. (2000)	Human peripheral blood lymphocytes exposed to 2.45 and 7.7 GHz RFR for 15, 30, or 60 min; power density 10, 20, or 30 mW/cm ²	Increased micronucleus frequency at a power density of 30mW/cm ² and after an exposure of 30 and 60 min.
Zotti-Martelli et al. (2005)	Human whole blood samples exposed to continuous-wave 1800 MHz RFR for 60, 120 and 180 min; power density 5, 10, or 20 mW/cm ²	A statistically significant increase of micronucleus was observed in lymphocytes dependent on exposure time and applied power density.
*Zuo et al. (2015)	Sprague-Dawley rat spiral ganglion neurons exposed intermittently (5 min ON/10 min OFF) to GSM 1800 MHz RFR for 24 h; SAR 2 and 4 W/kg	The RFR could not directly induce DNA single strand breaks (Comet assay) in normal spiral ganglion neurons, but it could cause the changes of cellular ultrastructure at SAR 4.0 W/kg when cells are in fragile or micro-damaged condition.

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Supplement 2

Genetic effects of static and ELF EMF (*study with no effect observed) Study reported effect =168 (79%); study reported no effect = 45 (21%). (literature up to January 2021)

	Exposure conditions	Results
Agliassa et al. (2018)	Arabidopsis thaliana (thale cress) exposed to 0.00004 mT static magnetic field for 38 days after sowing.	Changes in gene expression in leaf and floral meristem (cryptochrome-related gene involved); delayed flowering time and a significant reduction of leaf area index and flowering stem length, with respect to controls under geomagnetic field.
Ahuja et al. (1999)	Human peripheral blood samples exposed to 50 Hz EMF at 2, 3, 5, 7, or 10 mT	Increased DNA single strand breaks (Comet assay) in lymphocytes.(Damage levels higher in female than in male subjects.)
*Albert et al. (2009)	Human subjects exposed to exposed to 60-Hz magnetic field at 0.2 mT for 4 h	No significant effect on DNA single strand breaks (Comet assay) and micronucleus frequency in lymphocytes.
Alcaraz et al. (2013)	Swiss mice exposed to 50-Hz magnetic field at 0.2 mT for 7, 14, 21, or 28 days	Increased micronucleus frequency in bone marrow. Effect not affected by antioxidants.
Al-Huqail and Abdelhaliem (2015)	Maize seedlings exposed to 50-Hz electric field at 6 kV/m for 1, 3, or 5 days	Increased DNA single strand breaks (comet assay)
Amara et al. (2006)	Male rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days	Increased 8-oxo-dG concentration and oxidative damage in testis.
Amara et al. (2007a)	Human monocytic leukemia THP-1 cells exposed to static magnetic field at 250 mT for 1, 2, or 3 h	Lower level of DNA single strand breaks (Comet assay) at 3 h of exposure, no effect on oxidative damages and enzymes and oxidative DNA damage.
Amara et al. (2007b)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days	Increased 8-oxo-7,8-dihydro-2'-desoxyguanosine in kidney but not in liver. Also decreased anti-oxidative enzymes and increased lipid peroxidation. Zinc supplementation attenuated DNA oxidation induced by static magnetic field in kidney to the control level.

*Amara et al. (2009)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days	No significant effect on 8-oxo-7,8-dihydro-2'-deoxyguanosine in frontal cortex and oxidative stress induced. However, there was an increase in metallothioneins level which might have protected DNA from oxidative damage.
*Amara et al. (2011)	Rats exposed to a static magnetic field at 128 mT, 1 h/day for 30 days, also treated with cadmium (Cd)	Magnetic field had no interaction on Cd-induced increase in 8-oxo-7,8-dihydro-2'-deoxyguanosine in the frontal cortex and hippocampus. However, static magnetic field enhanced Cd-induced increase in oxidative damage in the rat brain.
Arruda-Neto et al. (2009)	<i>Microcystis panniformis</i> , the eukaryote <i>Candida albicans</i> and human MRC5 lung cells exposed to gamma radiation and then to static electric field for 2-20 h at 20- 1250 V/cm	Static electric field caused suppression of DNA repair in <i>C. albicans</i> . It decreased cell growth in <i>M. panniformis</i> when compared with gamma radiation alone. The electric field increased number of nuclei with γ -H2AX foci in the irradiated MRC5 cells. Electric field interferes mostly in the DNA repair mechanisms.
Ashta et al. (2020)	Human glioblastoma cells (A172) exposed to 10 Hz or static magnetic field at 5 mT, up to 96 h	Increased p52 gene expression, cytotoxicity and free radical formation; effects enhanced by Temozolomide.
Back et al. (2019)	Mouse embryonic stem cells exposed to hypomagnetic field (<0.005 mT) up to 12 days	Induced abnormal DNA methylation through the dysregulation of DNA methyltransferase3b (Dnmt3b) expression, eventually resulting in incomplete DNA methylation during differentiation.
Bagheri Hosseinabadi et al. (2019)	Blood samples from 102 thermal power plant workers as the exposure group and 136 subjects as the unexposed group.	Increased DNA single strand breaks (Comet assay) in lymphocytes of exposed subjects.
Bagheri Hosseinabadi et al. (2020)	Blood samples from thermal power plant workers; mean levels of exposure to ELF magnetic and electric fields were .0165 mT (± 6.46) and 22.5 V/m (± 5.38), respectively,	DNA single strand breaks (Comet assay) in lymphocytes decreased by antioxidants.

Balamuralikrishnan et al. (2012)	Blood from electrical workers exposed to ELF EMF occupationally	Increased chromosome aberrations and micronucleus in lymphocytes.
Baraúna et al. (2015)	Chromobacterium violaceum bacteria cultures exposed to ELF-EMF for 7 h at 0.00066 mT	Five differentially expressed proteins detected including the DNA-binding stress protein, which may help to prevent physical damage to DNA.
Belyaev et al. (2005)	Human lymphocytes exposed to 50 Hz magnetic field at 0.015 mT (peak) for 2 h (measurements made at 24 and 48 h after exposure).	Induced chromatin conformation changes and decreased background 53BP1 (protein co-localized with DNA double strand breaks and involves in DNA damage signaling pathway.)
Bertea et al. (2015)	Arabidopsis thaliana (thale cress) exposed to artificially reversed geomagnetic field conditions for 10 days at .0419 mT	Significant effects on plant growth and gene expression observed. This supports the hypothesis that GMF reversal contributes to inducing changes in plant development that might justify a higher selective pressure, eventually leading to plant evolution.
Borhani et al. (2011)	Female NMRI mice exposed to a 50-Hz EMF at 0.5 mT for 4 h/day, 6 days/week for 2 weeks. Mated on day 8 after exposure, on day 4, blastocysts were obtained by flushing the uterus horns.	DNA fragmentation index increased and decrease in blastocytes in exposed group.
*Brix t al. (2020)	Young volunteers allocated to three study arms were exposed to [¹⁸ F] fluoro-D-glucose alone, to a 3-T SMF alone or to both combined over 60 min at a PET/CT or a PET/MRI system.	No significant change in lymphocyte DNA double strand breaks (γH2AX) to static magnetic field or interaction with [¹⁸ F] fluoro-D-glucose.
Buddak et al. (2012)	Murine AT478 carcinoma cells cultured with cisplatin exposed to 50-Hz EMF for 16 min at 1 mT	Exposure to ELF-EMF alone resulted in an increase in DNA single strand breaks (Comet assay) compared to control cells. ELF-EMF lessened the effects of oxidative stress and DNA damage that were induced by cisplatin;

		however, ELF-EMF alone was a mild oxidative stressor and DNA damage inducer. The addition of ELF-EMF exposure to cisplatin treatment resulted in decreased ROS levels and antioxidant enzyme activity.
Burgos-Molina et al (2020)	DNA double strand breaks were induced in <i>Saccharomyces cerevisiae</i> yeast and exposed to a 50-Hz magnetic field for 21 days at 2.45 mT	Long-term magnetic field exposure increased the DNA repair activity.
Calabro et al. (2011)	Human neuronal-like cells exposed to static (2 mT) and 50 Hz (1 mT) for 3 h.	Fourier self deconvolution spectroscopic analysis showed alteration in DNA/RNA and increased beta-sheet.
Calabro et al. (2020)	Human Neuronal-like cells and roots of <i>Allium sativum</i> and <i>Vicia faba</i> exposed to a static and 50 Hz magnetic fields at intensities ranging from 1 mT to 0.8 T	Exposure to both low- and high-intensity magnetic fields in typical human and plant cells induces uncoiling and unpackaging of chromatin constituents, followed by chromosome alignment towards the direction of applied magnetic field, providing further demonstration that magnetic fields can induce the orientation of organic macromolecules even at low-intensity values.
*Cantoni et al.(1996)	Cultured mammalian cells exposed to 50 Hz electric (0.2 - 20 kV/m), magnetic (0.0002-0.2 mT), or combined electric and magnetic fields.	Repair of DNA single strand breaks (Comet assay) induced by the carcinogens methylmethane sulphonate (MMS), chromate, and 254 U.V. radiation not affected by ELF EMF exposure.
Celikler et al. (2009)	Workers from transformer and distribution line stations. The electric field was in the range from 130–8310 V/m and from 300–15,000 V/m, the magnetic field was between 0.5 and 1.7 A/m and 0.25–17 A/m around and inside transformer buildings. Average time of exposure was 19 years.	Increased chromosomal aberrations and micronucleus in peripheral lymphocytes. The frequency of chromosomal aberration in exposed groups correlated with the years of exposure.

*Cellini et al. (2008)	Escherichia coli ATCC 700926 exposed to 50-Hz EMF (0.1, 0.5, 1.0 mT); 20-120 min	No changes among DNA finger-printings. Other measurements indicates 50 Hz EMF acts as a stressing factor on bacteria
*Chahal et al. (1993)	Escherichia coli strain AB1157 exposed to a frequency of 1 Hz with field strengths of 1 or 3 kV m ⁻¹	Low frequency electromagnetic fields do not increase spontaneous mutation, induce DNA repair or increase the mutagenic effects of UV or mitomycin C.
Chen GD et al. (2008)	Human MCF-7 breast cancer cells exposed to a 50-Hz magnetic fields for 24 h at 0.4 mT	Identified three 50 Hz MF responsive genes in MCF-7 cells.
*Chen G et al. (2012)	<i>Saccharomyces cerevisiae</i> yeast cells exposed to a 50-Hz magnetic field at 0.4 mT for 6 h	Yeast cells did not alter gene expression in response to 50 Hz magnetic field.
Chan J. et al. (2020)	Human choriocarcinoma cells exposed to DC electric field (150 mV/mm) for 8 h	Increased gene expressions of ErbB and HIF-1 signaling pathways involved in cell migration/motility, cell cycle progression and proliferation.
Chen WF et al. (2010)	Human myelogenous leukemia K562 cells exposed to static magnetic field at 8.8 mT with or without cisplatin	Static magnetic field exposure induced DNA to become thicker than controls, and enhanced DNA breakage (Comet assay) induced by cisplatin.
Cho S et al. (2014)	Human lymphocytes exposed to 60-Hz EMF at 0.8 mT for 12-72 h with or without gadolinium.	ELF-EMF increased cell death, micronucleus frequency, DNA single strand break (Comet assay), and apoptosis induced by gadolinium.
Cho YH et al. (2007)	Human fibroblasts exposed to 60-Hz EMF at 0.8 mT plus bleomycin for 28, 88, and 240 h	The co-exposure of cells to bleomycin and EMF led to a significant increase in the frequencies of micronucleus and aneuploidy compared to the cells treated with bleomycin alone.
Chow and Tung (2000a)	Escherichia coli strain XL-1 Blue exposed a 50-Hz magnetic field at 0.1-1.2 mT for 1 h	This result was indicative that the efficiency of DNA repair had been improved. The improvement was found to be mediated by the induced overproduction of heat shock proteins DnaK/J (Hsp70/40).
Chow and Tung (2000b)	Escherichia coli strain XL-1 Blue (transformed by plasmid pUC8 that had been mutagenized by	Improved efficiency of DNA repair mediated by the induced overproduction of heat shock proteins DnaK/J (Hsp70/40).

	hydroxylamine exposed a 50-HZ magnetic field at 0.1-1.2 mT for 1 h	
Collard et al. (2013)	Epidermis cultures harvested from human abdominoplasty exposed to ELF electric fields (a biphasic, asymmetric, charge-balanced current stimuli, with a repetition frequency of 40 Hz modulated by a fundamental frequency of 0.125 Hz. The exposure was repeated during 4 s followed by a 4 s break for 40 min/day for 11 days	Observed a significant change in genes expression after 4 days and change in expression in another group of genes at day 4 and 7. Genes are involved in cell proliferation or differentiation, mitosis, cell cycle or in the DNA replication transcription and translation.
Consales et al. (2018)	Human SH-SY5Y neuroblastoma cells and mouse primary cortical neurons exposed to a 50-Hz magnetic field at 1 mT for 4-72 h	Expressions of microRNA miR-34b/c that caused mitochondrial oxidative stress, also altered α -synuclein expression involved in synaptic functions. These effects may be related to neuro-degeneration.
Cuccurazzu et al. (2010)	Mice exposed to 50 Hz EMF at 1 mT for 1-7 h/day for 7 days	Induced increases in the transcription of pro-neuronal genes (Mash1, NeuroD2, Hes1) and genes encoding Ca(v)1.2 channel α (1C) subunits in the hippocampus. Generation of new granule cells in the dentate gyrus.
Del Re et al. (2006)	Escherichia coli exposed to sinusoidal or pulsed square wave 50-Hz magnetic field at 1 mT for 40 min	Sinusoidal magnetic field exposure induced a significantly higher level of DnaK and GroEL, whereas a lower level was observed after pulsed magnetic field exposure. When bacterial cells were exposed to heat shock (HS) after ELF-magnetic field exposure: again sinusoidal and pulsed fields resulted in an increase and in a reduction of HSP amount.
Delimaris et al. (2006)	Human lymphocytes exposed to 50-Hz pulsed electric fields (10-Hz carrier frequency) at 4×10^5 V/m for 120 min	Increased in DNA single strand breaks (Comet assay).
Di Campli et al. (2010)	Helicobacter pylori biofilm exposed to 50-	No changes in DNA patterns were recorded, whereas a modulation in amiA gene

	Hz EMF at 1 mT for 2 days	expression was detected; phenotypic changes induced.
Dominici et al. (2011)	Lymphocytes from welders (average magnetic field exposure from personal dosimeters 0.00781 mT (general environmental level 0.00003 mT)	Higher micronucleus frequency correlated with EMF exposure levels; decreased in sister chromatid exchange frequency.
Dong et al. (2019)	Human pre-osteoclast RAW264.7 cells exposed to a 16 T static magnetic field for 2-4 days	HiSMF markedly blocked the expression of osteoclast-associated transcription factors and osteoclast marker genes and inhibited iron absorption and iron storage-related protein expression. Mitochondrial concentration and oxidative stress levels in osteoclasts were decreased under magnetic field exposure.
Du et al. (2008)	Cultured human lens epithelial cells exposed 50-Hz magnetic field at 0.4 mT for 2 h, 6 h, 12 h, 24 h and 48 h	Increased DNA doubled strand breaks (γ -H2AX foci) after 24 h exposure.
Duan et al. (2015)	A mouse spermatocyte-derived GC-2 cell line intermittently (5 min on and 10 min off) exposed to a 50 Hz EMF at 1, 2 or 3 mT for 24 h	Increased DNA strand breaks (Comet assay and γ -H2AX foci) at 3 mT exposure.
El-Bialy and Rageh (2013)	Mice with Ehrlich tumors exposed to a 50-Hz magnetic field 1 h/day for 2 weeks at 10 mT	Exposure cause DNA single strand breaks (Comet assay) in tumor cells and increased micronucleus frequency in bone marrow cells. ELF-MF enhanced the effects of cisplatin.
Erdal et al. (2007)	Wistar rats exposed to 50 Hz magnetic field at 1 mT for 4 h or 4h/day for 45 days	Micronucleus frequency higher in bone marrow cells of long-term exposed rat. Mitotic index decreased in both exposed groups.
*Fairbairn and O'Neill (1994)	Human cells exposed to ELF-EMF	No significant effect on DNA single strand breaks (Comet assay)
Fan et al. (2005)	Rat bone marrow derived-mesenchymal stem cells exposed to a 50-Hz EMF at 1 mT for 4 h/day for 3 days	Increased cell viability, DNA synthesis and proportion of cells in S phase and up-regulated the expressions of hematopoietic growth factors.

Fan et al. (2018)	<i>Enterococcus faecalis</i> (isolated from dental infection) exposed to a static magnetic field at 170 mT for 24 or 72 h.	Static magnetic field up-regulated the expression of stress gene (dnaK) and virulence genes (efaA and ace). Synergistic with alkaline pH induced by calcium hydroxide (a major dental antimicrobial) in antimicrobial action and up-regulation of stress and virulence genes.
Fatigoni et al. (2005)	Tradescantia (a perennial wildflower) exposed to a 50-Hz magnetic field at 1 mT for 6 or 24 h	Caused a time-dependent increase in micronucleus frequency.
Fedrowitz and Loscher (2012)	Female F344 and Lewis rats exposed to a 50-Hz magnetic field at 0.1 mT 24 h/day for two weeks	F344 breast tissue showed alterations in gene expression, which were absent in Lewis rats, particularly, α -amylase, a stress marker.
*Fiorani et al. (1992)	Human immortalized myelogenous leukemia K562 cells exposed to 50-Hz electric (0.2-20 kV/m) or magnetic (0.0002-.2 mT) or combination of electric and magnetic fields, for 24 h	No detectable DNA lesions (measured by filter elution technique).
Focke et al. (2010)	Human fibroblasts exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 15 h	Increased DNA single strand breaks (Comet assay) caused by magnetic and not electric field, No oxidative DNA damage. Could be caused by minor disturbances in S-phase processes and occasional triggering of apoptosis rather than by the generation of DNA damage.
*Frahm et al. (2006)	Mouse macrophages exposed to a 50-Hz magnetic field for 45 min, 12, 24, or 48 h; 0.05 – 1 mT	No genotoxic effect (micronucleus formation); increased phagocytic activity, free radicals, and IL-1 beta production.
*Frazier et al. (1990)	Human lymphocytes induced with DNA damage with ionizing radiation were exposed to 60-Hz magnetic field at 1 mT, electric field at 1 or 20V/m, or combinations of magnetic and electric	EMF exposure did not affect repair of DNA single strand breaks (Comet assay).

	fields (0.2 V/m and 0.05 mT, 6 V/m and 0.6 mT, or 20 V/m and 1 mT) up to 180 min	
Frisch et al. (2013)	Transfected rat primary fibroblast (RAT1) cells exposed to 10 Hz electric fields at 20-500 V/m for 2 h	Induced HSP70 heat shock expression, with peak responses obtained at 8 h following exposure.
Giorgi et al. (2011)	Two Escherichia coli model systems were exposed to sinusoidal or pulsed-square wave magnetic fields of various frequencies (20, 50, 75 Hz) and for different exposure times (15 and 90 min). at 1 mT	ELF-MF exposure affected transposition activity (transposon (Tn) mobility) and the effects critically depended on the wave shape of the field, but not on the frequency and the exposure time.
*Giorgi et al. (2014)	Human neuroblastoma BE(2)C cells treated with hydrogen peroxide exposed to 50-Hz pulsed magnetic field at 1 mT for 1-72 h	Pulsed magnetic field exposure did not interfere with genotoxicity (DNA double strand breaks measured by γ -H2AX foci) and cytotoxicity induced by oxidative stress.
Giorgi et al. (2017)	Human neural cells (BE(2)C) exposed to pulsed 50-Hz magnetic field at 1 mT for 24 and 48 h in combination with oxidative stress (hydrogen peroxide)	Pulsed magnetic field and oxidative stress induced weak decreases and increases of DNA methylation levels; combined exposure led to significant transient decrease of DNA methylation levels at different genome loci.
Heredia-Rojas et al. (2010)	Human non-small cell lung cancer cells (INER-37) and mouse lymphoma cells (RMA E7) (transfected with a plasmid with hsp70 expression when exposed to magnetic field and contains the reporter for the luciferases gene) exposed to a 60-Hz magnetic field at 0.008 and 0.00008 mT for 20 min.	An increased in luciferase gene expression was observed in INER-37 cells exposed to magnetic field, but similar exposure had no effect on the RMA E7 cell line.

Hong et al. (2005)	Mice exposed to a 50-Hz EMF at 0.2 or 6.4 mT for 4 weeks	EMF induced DNA single strand breaks (Comet assay) in testicular cells and chromatin condensation in spermatozoa.
*Huwiter et al. (2012)	Escherichia coli K-12 MG1655 exposed at 50-Hz magnetic fields generated by three signal types (sinusoidal continuous, sinusoidal intermittent, and power line intermittent) at 1 mT for 8 min, 2.5 h, or 15 h	No effect on transcription of 4358 gene studied.
Ivancsits et al. (2002)	Human diploid fibroblasts exposed to continuous or intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 24 h	Intermittent exposure induced DNA single and double strand breaks (Comet assay).
Ivancsits et al. (2003a)	Human diploid fibroblasts exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 0.02- 1 mT for 1-24 h	DNA Single and double strand breaks (Comet assay) observed at 0.035 mT at 15 h; recovered within 9 h.
Ivancsits et al.(2003b)	Fibroblasts from human subjects of different ages exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 1-24 h	Increased DNA Single and double strand breaks (Comet assay) at 15 h; more pronounced in cells from older donors
Ivancsits et al. (2005)	Various cell types exposed to intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 1-24 h	Effects on DNA Single and double strand breaks (Comet assay) showed three responder (human fibroblasts, human melanocytes, rat granulosa cells) and three non-responder cell types (human lymphocytes, human monocytes, human skeletal muscle cells).
Jajte et al. (2001)	Rat peripheral blood lymphocytes exposed to a 50-Hz magnetic field at 7 mT for 3 h	Increased DNA single strand breaks (Comet assay) in cells treated with ferrous chloride; melatonin attenuated the effect.
*Jin H. et al. (2015)	Non-tumorigenic human lung epithelial L132 cells exposed to a 60-Hz magnetic field at 1 or 2 mT for 9 h	No G2/M arrest or aneuploidy nor interaction with gamma radiation and H ₂ O ₂

*Jin et al, (2012)	Mouse embryonic fibroblast NIH3T3 cells and human lung fibroblast WI-38 cells exposed to a 60 Hz magnetic field at 1 mT for 4 h	No significant effect on micronucleus frequency and interaction with ionizing radiation, H ₂ O ₂ , or c-Myc activation.
*Jin et al, (2014)	NIH3T3 mouse fibroblast cells, WI-38 human lung fibroblast cells, L132 human lung epithelial cells, and MCF10A human mammary gland epithelial cells exposed to a 60-Hz magnetic field at 1 mT for 4 or 16 h	No significant effect on DMA single strand breaks (Comet assay), and interaction with ionizing radiation, H ₂ O ₂ , or c-Myc activation.
Jin et al. (2019)	Arabidopsis young seedlings exposed to a static magnetic field at 600 mT	Increased auxin (a plant growth hormone) from expression of PIN3 and AUX1 genes in root tips; cryptochromes (cry1 and cry 2) are also involved. Root growth enhanced. Effects occurred when static magnetic field was parallel and perpendicular not opposite, to geomagnetic field.
Jouni et al. (2012)	<i>Vicia faba</i> (broad bean) culture in soil with high background radioactivity and exposed to static magnetic field at 15 mT for 8h/day for 8 days	Increased chromosomal aberration and DNA damage in root tip cells with lowering of antioxidant defense; soil radioactivity enhanced the effects.
Kesari et al. (2015)	Human neuroblastoma SH-SY5Y cells exposed to a 50-Hz 100 μ T magnetic field for 24 h.	Micronucleus formation was observed at 15 and 30 days postexposure. Effect not related to oxidative changes.
Kesari et al. (2016)	Human glioblastoma SH-SY5Y and rat glioma C6 cells exposed to a 50-Hz magnetic field at 0.01 and 0.03 mT for 24 h with menadione as a cofactor	Micronuclei were significantly increased in SH-SY5Y cells at 0.03 mT Increased cytosolic and mitochondrial superoxide levels were observed in C6 cells. The results indicate that the threshold for biological effects of ELF magnetic field is 0.01 mT or less.
Khalil and Qassem (1991)	Human lymphocytes exposed to a pulsing 50-Hz EMF at 1.05 mT for 24, 48 and 72 h	Suppression of mitotic activity and a higher incidence of chromosomal aberrations. Delay in cell proliferation index and an increase in the baseline frequency of sister-chromatid

		exchanges occurred only after 72 h f exposure.
Ki et al. (2020)	Human hair follicle dermal papilla cells, a type of cells involved in hair growth, exposed to a 70 Hz EMF at intensities ranging from 0.5 to 10 mT over four days	Increased the expression of anagen-related molecules, including collagen IV, laminin, ALP, and versican, and increased β -catenin and Wnt3 α expression and GSK-3 β /ERK/Akt phosphorylation. Cell proliferation enhanced.
Kim HJ. et al. (2013)	Bone marrow derived mesenchymal stem cells (BM-MSCs) were subjected to a 50-Hz EMF	Increased levels of neuronal differentiation marker (MAP2), while early neuronal marker (Nestin) was down-regulated; increased differentially expression of 8 proteins; notably, a significantly increased expression of the ferritin light chain.
Kim J. et al. (2010)	IMR90 (human lung fibroblast) primary cells and HeLa (human cervical carcinoma) cells exposed to a time-varying (rotating) 60-Hz magnetic field at 6 mT for 60 min or 30 min/day for 3 days	Repeated exposure showed DNA double strand breaks (γ -H2AX foci) and decreased cell viability and increased apoptosis through p38 activation.
Kim J. et al. (2012)	Human primary fibroblast and cervical cancer cells exposed to a time-varying 60-Hz magnetic field at 7 mT for 10-60 min	DNA double strand breaks (γ -H2AX foci and Comet assay) detected (intracellular reactive oxygen species not affected).
Kimsa-Dudek et al. (2018)	Normal human dermal fibroblasts exposed to static magnetic field at 0.65 T for 24 h and sodium fluoride	Static magnetic field attenuated expression of antioxidant defense genes (SOD1, PLK3, CLN8, XPA, HAO1) induced by sodium fluoride.
Kimsa-Dudek et al. (2020)	Normal human dermal fibroblasts exposed to static magnetic field at 0.45, 0.55 and 0.5 T for 24 h and sodium fluoride	The field reduced fluoride-induced apoptosis and affected apoptosis gene expression; reduced fluoride-induced increases in reactive oxygen species and lipid peroxidation and decrease in antioxidant enzymes.
Kimura et al. (2008)	<i>Caenorhabditis elegans</i> exposed to 2, 3, or 5 T static magnetic field for 4-24 h	Genes involved in motor activity, actin binding, cell adhesion, and cuticles are transiently and specifically induced; also hsp (heat shock protein) 12 and 16 family genes.

Kindzelskii and Petty (2000)	Human neutrophils exposed to pulsed square-wave (20 msec) DC electric field at 0.2 V/m for 30, 45, 60 min	Increased DNA single strand breaks (Comet assay).
*Kirschenlohr et al. (2012)	Male human subjects exposed to 50-Hz EMF at 0.062 mT for 2 h (Exposure repeated two more times.)	No genes or gene sets in blood samples showed consistent response profiles to repeated ELF-EMF exposures (including immediate early genes, stress response, cell proliferation and apoptotic genes).
Koyama et al. (2008)	Human glioma A172 cells exposed to a 60-Hz magnetic field at 5 mT for 2, 4, 8, 16, 24 h	The number of apurinic/apyrimidinic sites induced by genotoxic agents methyl methane sulfonate and H ₂ O ₂ was enhanced by exposure to ELF magnetic fields. (Apurinic/apyrimidinic sites are common DNA lesions arise from spontaneous depurination or by base excision repair of oxidized, deaminated or alkylated bases.)
Kubinyi et al. (2010)	Human lymphocytes exposed to an inhomogeneous static magnetic field with a lateral magnetic flux density gradient of 47.7, 1.2, or 0.3 T/m by 10 mm lateral periodicity, or a homogeneous SMF of 159.2 mT magnetic flux density for a time period of 0.5 min, 1, 2, 4, 6, 18, 20, or 24 h.	Increased DNA single strand breaks (Comet assay); affected DNA repair induced by gamma ray when exposure occurred after ionizing radiation treatment.
Kumari et al. (2017)	Mice exposed continuously for 5 weeks to 7.5 KHz MF at 120 μ T	Expression of the pro-inflammatory cytokine tumor necrosis factor alpha mRNA was significantly increased in the hippocampal region; impairment of memory observed.
*Lacy-Hulbert et al. (1995)	Human leukemic cells (HL60) exposed to a 60-Hz EMF for 20 min at 0.00057, 0.0057, or 0.057 mT	No change in MYC and beta-actin gene expression observed.
Lagroye and Poncy (1997)	Rat tracheal epithelial cell lines were first exposed to gamma rays and then cultured in a 50-Hz magnetic field at 0.1 mT for 24 h.	Increased binucleated cells with micronuclei in cells exposed to gamma rays and magnetic field, compared with gamma irradiation alone. Magnetic field alone had no significant effect on micronucleus frequency.

Lai and Singh (1997a)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.1, 0.25, or 0.5 mT for 2 h	Increased DNA single and double strand break (Comet assay) in brain cells.
Lai and Singh (1997b)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.5 mT for 2 h	Increased DNA single and double strand break (Comet assay) in brain cells. Effects blocked by melatonin and a spin-trap compound.
Lai and Singh (2004)	Male Sprague-Dawley rats exposed to a 60-Hz magnetic field at 0.01 mT for 24 or 48 h	Increased DNA single and double strand break (Comet assay) in brain cells. More effect with 48-h than 24-h exposure. Effects blocked by Trolox (a vitamin E analog) and 7-nitroindazole (a nitric oxide synthase inhibitor).
Laramee et al. (2014)	Transfected rat primary fibroblast (RAT1) cells exposed to static magnetic fields of 1 to 440 mT for 16, 24, or 48 h starting at 24 and 48 h post transfection	Induction of heat shock protein (HSP70) expression showed a dependency on flux density, exposure duration, and start time post transfection.
Lee et al. (2010)	<i>Caenorhabditis elegans</i> exposed to exposed to a static magnetic field at 200 mT	Expression of genes involved in development and aging. Accelerated development and shorten lifespan.
Lee et al. (2016)	MCF10A, MCF7, Jurkat, and NIH3T3 cells exposed to a 60 Hz magnetic field at 1 mT for 4 or 16 h	MCF10A and MCF7 cells showed consistent and significant decreases in cell number, cell viability, and DNA synthesis rates (cell cycle delay), whereas Jurkat and NIH3T3 cells showed no effect. MCF7 cells (2 mT for 16 h) showed up-regulation of PMAIP1 gene (involved in apoptosis).
Lee et al. (2011)	Human lymphocytes exposed to EMF generated during MRI scanning (clinical routine brain examination protocols: three-channel head coil) for 22, 45, 67, and 89 min	Significant increases in DNA single-strand breaks (Comet assay), and frequencies of both chromosome aberrations and micronuclei in a time-dependent manner.
Leone et al. (2014)	Neural stem cells isolated from hippocampi of newborn mice exposed to a 50-Hz EMF at 1 mT for 10 days	Histone acetylation-related chromatin remodeling leading to enhanced proliferation and neuronal differentiation.

Li and Chow (2001)	E. coli XL-1 Blue transformed with plasmid pUC18 and DNA samples exposed to a 50-Hz magnetic field at 1.2 mT for 1-5 h, with heat shock response suppressed	Without the protection of the heat shock response, magnetic field exposure induced DNA degradation, which could be attenuated by the presence of an antioxidant,
*Li L. et al (2015)	Workers from a power supply bureau (inspection workers vs. logistic staff); The average time-weighted average was 0.0073 mT (0.00156-0.02633 mT) and the subjects were subgrouped by cumulative ELF-magnetic field exposure dose: low (<0.0156 mT), middle (0.0156-0.073 mT) and high (> 0.073 mT)	No significant effect on the frequency of micronucleus lymphocytes or micronuclei frequency; no changes in antioxidant enzymes and cellular oxidative damage.
Li SS et al. (2013)	Male <i>Drosophila melanogaster</i> fruit flies exposed to a 50-HZ EMF at 3 mT for 72 or 312 h	Different sets of genes were up- and down-regulated after short- or long-term exposure. Short-term exposure may decrease the reproductive ability of males, whereas long-term exposures had no effect on reproductive ability.
Li Y. et al. (2014)	Fertilized embryos of zebra fish (<i>Danio rerio</i>) exposed to a 50-Hz magnetic field at 0.1 - 0.8 mT for 96 h	The transcription of apoptosis-related genes (caspase-3, caspase-9) was significantly up-regulated in exposed embryos. Delayed hatching and apoptosis observed.
Li, Y. et al. (2015)	Rat oligodendrocyte precursor cells exposed to DC electric field at 50, 100. Or 200 mV/mm for 1.5 h	Mitogen-activated protein kinase pathway that signals cell migration was significantly upregulated in cells treated with an EF of 200 mV/mm compared with control cells and downregulation of differentially expressed genes in chemotaxis.
Li Y. et al. (2019)	Dementia rats induced by streptozotocin (STZ) intracerebroventricular injection exposed to a 10 mT 20-Hz pulsed EMF,	Pulsed EMF increased expression of insulin growth factor 2 (IGF-2) in the hippocampus and improved the ability of learning and memory in STZ-treated rats.

	2 h/day, 10 days	
Lin et al. (2016)	Budding yeast exposed to a 50-Hz EMF at 6 mT for 96 h	The transcription levels of 28 genes were upregulated and those of four genes were downregulated. Exposure can upregulate the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway.
Liu et al. (2015)	Mouse spermatocyte-derived GC-2 cell line exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1, 2, or 3 mT for 72 h	Exposure decreased genome-wide methylation at 1 mT, but global methylation was higher at 3 mT. Expression of DNMT1 and DNMT3b (DNA methyltransferases) was decreased at 1 mT, and increased at 3 mT.
*Lopucki et al. (2005)	Cotyledons dissected from placentas obtained immediately after physiological labors exposed to a 50-Hz magnetic field at 2 or 5 mT for 3 h	No significant effect on level of 8-hydroxy-2'-deoxyguanosine in DNA (oxidative DNA damage).
Lourencini da Silva et al. (2000)	SnCl ₂ -treated pBR322 plasmids exposed to a 3400Hz square-wave EMF with peak power of 4V for 2 h	An EMF-dependent potentiation of DNA scission (i.e. the appearance of relaxed plasmids) was observed. The results indicate that the EMF, in the presence of a transition metal, is capable of causing DNA damage.
*Luceri et al. (2005)	Human peripheral blood lymphocytes and DBY747 <i>Saccharomyces cerevisiae</i> exposed to a 50-Hz magnetic field at 0.001, 0.01 or 0.1 mT for 18 h	No significant effects on DNA single strand breaks (Comet assay), oxidated DNA base, and gene expression.
Lupke et al (2006)	Human umbilical cord blood-derived monocytes exposed to a 50-Hz magnetic field at 1 mT for 45 min	Alteration of 986 genes involved in metabolism, cellular physiological processes, signal transduction and immune response.
Luukkonen et al. (2011)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by chemical (menadione) exposure for 3 h	Magnetic field enhanced menadione-induced DNA damage, DNA repair rate, and micronucleus formation. No effects were observed after magnetic field exposure alone.

Luukkonen et al. (2014)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by menadione exposure for 3 h	Persistently elevated levels of micronuclei were found in the progeny of magnetic field (alone)-exposed cells at 8 and 15 days after exposure, indicating induction of genomic instability. (No magnetic field x menadione interaction effect). Magnetic field disturbed oxidative balance immediately after the exposure, which might explain the previous findings on MF altered cellular responses to menadione-induced DNA damage.
Luukkonen et al. (2017)	Human SH-SY5Y neuroblastoma cells. Exposed to a 50-Hz magnetic field at 0.1 mT for 24 hours, followed by menadione exposure for 1 or 3 h	Decreased p21 protein (a DNA damage response-related proteins) level after 1-h menadione treatment, as well as increased proportion of cells in the G1 phase and decreased proportion of S phase cells after 3-h menadione treatment. Magnetic field exposure decreased DNA single strand breaks (Comet assay) caused by 1 h treatment with menadione.
Ma et al. (2014)	Mouse embryonic neural stem cells exposed to a 50-Hz EMF at 2 mT for 3 days	Expression of genes regulating neuronal differentiation was altered.
Mahaki et al. (2019)	Rats exposed to a 50-Hz EMF at 0.001-2 mT for 2 h/day for 60 days	In the spleen, gene expression levels of ROR α (retinoid-related orphan receptor alpha) and c-Maf (transcription factor Maf) were significantly down-regulated at 0.001 and 0.1 mT, while the expression of STAT6 (signal transducer and activator of transcription 6) was only significantly decreased at the density of 0.1 mT. No effect on thymus.
Mahmoudinasab and Saadat (2016)	Human MCF-7 cells exposed to a 50-Hz magnetic field at 0.25 and 0.5 mT (5 min ON/5 min OFF, 15 min ON/15 min OFF, or 30 min field-on continuously) for 30 min	Alterations in the <i>NQO1</i> and <i>NQO2</i> (NAD(P)H: quinone oxidoreductase) mRNA levels seen at the "5 min ON/5 min OFF" condition.
Mahmoudinasab and Saadat (2018a)	MCF-7 and SH-SY5Y cells exposed to 50-Hz EMF at 0.5 mT (15 min ON/ 15 min OFF), and treated with morphine and cisplatin.	EMF exposure could protect SH-SY5Y cells from the cytotoxicity of cisplatin and morphine, whereas it has no significant change in MCF-7 cells. Expression patterns of antioxidant genes are different in both cell lines.

Mahmoudinasab and Saadat (2018b)	SH-SY5Y cells exposed to 50-Hz EMF at 0.5 mT ("15 min ON/ 15 min OFF" and "30 min ON") for 30 min, and treated with morphine and beta-lapachone	NQO1 mRNA level decreased in the "15 min field-on/15 min field-off" condition, the expression level of NQO2 was increased. Morphine and EMF reduced the cytotoxicity of beta-lapachone.
Mahmoudinasab et al. (2016)	Human MCF-7 cells exposed to a 50-Hz magnetic field at 0.25 and 0.5 mT (5 min ON/5 min OFF, 15 min ON/15 min OFF, or 30 min field-on continuously) for 30 min	Significant changes in mRNA levels of seven antioxidant genes for "the 15 min field-on/15 min field-off condition".
Mairs et al. (2007)	UVW human glioma cells to a 50-Hz EMF at 1 mT for 12 h	Induced 0.011 mutations/locus/cell, which was equivalent to a 3.75-fold increase in mutation induction compared with unexposed controls. The field also potentiated the mutagenic capacity of gamma-irradiation.
Manzella et al. (2015)	Human dermal fibroblasts exposed to a 50 Hz magnetic field at 0.1 mT for 1 h	Changes in expression of clock genes.
Mariucci et al. (2010)	CD1 mice exposed to a 50-Hz magnetic field at 1 mT for 1 or 7 days (15 h/day)	Increased DNA single strand breaks (Comet assay) in brain areas detected immediately after 7-day exposure. No effect on HSP-70 expression.
Markkanen et al. (2008)	Murine L929 fibroblasts exposed to a 50-Hz magnetic field at 0.1 or 0.3 mT for 24 h, with or without ultraviolet B (UVB, wavelength 280-320 nm) radiation or menadione (MQ)	Pre-exposure to magnetic field can alter cellular responses to other agents, and indicate that magnetic field as low as 0.1 mT has measurable impacts on cancer-relevant cellular processes such as DNA-damage.
Mastrodonato et al. (2018)	Mice exposed to a 50 Hz, 1 mT EMF 3.5 h/day for 12 days	Increased Wnt3 (neurogenesis gene) mRNA expression and nuclear localization of its downstream target β -catenin in subventricular zone of the lateral ventricle. Mice showed enhanced olfactory memory at 30 days post-exposure.

*McNamee et al. (2002)	10-day-old mice exposed to a 60-Hz magnetic field at 1 mT for 2 h, cerebellum assayed at 0, 2, 4, and 24 h after exposure	DNA single strand breaks (Comet assay): “While increased DNA damage was detected by tail ratio at 2h after MF exposure, no supporting evidence of increased DNA damage was detected by the other parameters.” “Taken together, these results do not support the hypothesis that acute MF exposure causes DNA damage in the cerebellums of immature mice.” No change in apoptosis.
*McNamee et al. (2005)	Rodents (adult rats, adult mice, and immature mice) exposed to a 60-Hz magnetic field at 0.1, 1 or 2 mT for 2 h. Assayed at 0, 2 and 4 h after exposure	This study provided no evidence of magnetic-field-induced DNA single strand breaks (Comet assay) in the brain.
Mercado-Sáenz et al. (2019)	<i>Saccharomyces cerevisiae</i> wild type strain (WS8105-1C) exposed to sinusoidal magnetic field (2.45 mT, 50 Hz, continuous) or pulsed magnetic field (1.5 mT, 25 Hz, 8 h/day). Chronological aging was evaluated during 40 days	Decreased spontaneous frequency of mitochondrial mutation during aging was observed in pulsed magnetic field-treated samples.
*Miyakoshi et al. (1996a)	Chinese hamster ovary (CHO) cells exposed to a 60-Hz magnetic field at 5 mT for 130 h	No significant effect on c-myc expression and cell growth rate.
Miyakoshi et al. (1996b)	Human melanoma MeWo cells exposed to a 50-Hz magnetic field at 400 mT up to 20 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene, synergistic with X-ray. No significant increase in mutant frequency occurred when DNA replication was inhibited during magnetic field exposure. DNA replication error is suspected of causing the mutations produced by ELF-MF exposure.
Miyakoshi et al. (1997)	Human melanoma MeWo cells exposed to a 50-Hz magnetic field at 400 mT for 2 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene, DNA replication errors and/or disturbance of the mismatch repair systems caused by exposure to ELF-MF may be involved in the

		mutagenic effect.
Miyakoshi et al. (1998)	Human osteosarcoma cells (Saos-LP-12), with deleted 53 gene, exposed to a 50-Hz magnetic field at 400 mT for 4 h	Induced mutations in the hypoxanthine-guanine phosphoribosyl transferase gene. Introduction of the wild-type (wt) p53 expression plasmid (pOPRSVp53) suppressed the magnetic induced mutation. The findings suggest that wt p53 has a function in suppression of DNA replication errors and/or in maintenance of genomic stability after high-density magnetic field exposure.
Miyakoshi et al. (1999)	Chinese hamster ovary K1 (CHO-K1) cells exposed to a 60-Hz magnetic field at 5 mT for up to 6 weeks	No effect on mutant frequency of the hypoxanthine-guanine phosphoribosyl transferase but enhanced the effect of x-ray.
Miyakoshi et al. (2000)	Human glioma MO54 cells exposed to a 50-Hz magnetic field at 55, 50, or 400 mT at 4 ⁰ C or on ice, for 30 min	Exposure to magnetic field at more than 50 mT potentiated X-ray-induced DNA single strand breaks (Comet assay).
*Mizuno et al. (2014)	Human fibroblast WI38VA13 subcloned 2RA and XP2OS(SV) cells exposed to a 60-Hz magnetic field at 5 mT for 24 h	Magnetic field exposure did not have modification effect on cell survival after UV-B irradiation and on repair process of DNA damage induced by UV-B irradiation.
Moraveli et al. (2016)	dermal papilla mesenchymal cells exposed to 50-Hz EMF at 1 mT for 5-14 days	Increased expression of MAP gene with decreased cell proliferation (cell differentiation occurred.) (MAP2 protein involves in neuritogenesis to stabilize microtubules.)
Moretti et al. (2005)	Jurkat cells exposed to a 50-Hz magnetic field at 1 mT for 1 h with added xenobiotics	Magnetic field exposure enhanced genotoxic effects (DNA single strand breaks (Comet assay)) of xenobiotics.
Mouhoub et al. (2017)	<i>Salmonella hadar</i> grown under static magnetic field of 200 mT for 3, 6, or 9 h	Increased expression of gene involved in the production of acdiolipin and phosphatidylethanolamine (both components of bacteria cell membrane).
Nakayama et al. (2016)	Macrophages stimulated with the bacterial endotoxin, lipopolysaccharide and	Increased DNA single strand breaks (Comet assay) and decreased viability.

	posed to a 50-Hz magnetic field at 0.5 mT for 24 h	
Nasrabadi et al. (2018)	Neonatal human retinal pigment epithelial cells exposed to pulsed 50-Hz EMF at 1 mT for 8 h daily for 3 days	Both gene and protein expressions of retinal progenitor cell markers were reduced.
Nikolova et al. (2005)	Mouse embryonic stem (ES) cells exposed to an intermittent (5 min ON/30 min OFF) 50-Hz EMF at 2 mT for 6 or 48 h	Significantly affected transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes, No effect on DNA single and double strand breaks (Comet assay).
*Okudan et al. (2010)	Swiss mice exposed to a 50-Hz EMF at 0.001 - 0.005 mT for 40 days	The results suggest that ≤ 0.005 mT intensities of 50 Hz EMFs did not cause genotoxic effect in the mouse. (However, The number of micronucleus per peripheral blood lymphocytes in the 0.004 and 0.005 mT-exposure groups were significantly higher than those of the lower intensity exposure groups. The males in 0.004 mT-exposure group displayed the highest micronucleus number per lymphocyte).
Panagopoulos et al. (2013)	Newly eclosed <i>Drosophila melanogaster</i> exposed to 50-Hz magnetic field (0.1, 1.1, and 2.1 mT) continuously during the first 5 days of their adult lives	Severe DNA damage (DNA fragmentation by TUNEL assay) and consequent cell death induction in the reproductive cells.
Pesqueira et al. (2017)	Human tendon-derived cells exposed to a 2 Hz magnetic field at 350 mT for 4 or 8 h, or 8 h every 24 or 48 h up to 14 days	8-h exposure significantly upregulated the expression of tendon-associated genes SCX, COL1A1, TNC and DCN. 8 h every 24 h exposure significantly upregulated COL1A1, COL3A1 and TNC at day 14.
Pilger et al. (2004)	Human fibroblasts exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF at 1 mT for 15 h	Exposure resulted in an increase in DNA single strand breaks (Comet assay) unlikely to be caused by intracellular changes that affect intracellular $[Ca^{2+}]$ or mitochondrial membrane potential.
Potenza et al. (2004a)	<i>E. coli</i> XL-1Blue exposed to static	Increased cell proliferation and changes in gene expression observed. The field

	magnetic field at 300 mT up to 50 h	magnetic field may stimulate transposition activity.
Potenza et al. (2004b)	Escherichia coli DNA, plasmid, and amplification products of different lengths exposed to static magnetic field at 200-150 mT for 5 h	The in vitro assays displayed interactions between the magnetic field and DNA, revealing principally that magnetic field exposure induces DNA alterations in terms of point mutations.. This genotoxic effect of the magnetic field, however, is minimized in living organisms due to the presence of protective cellular responses.
Rageh et al. (2012)	Newborn rats (10 days after delivery) exposed continuously to a 50 Hz magnetic field at 0.5 mT for 30 days	Increased DNA single strand breaks (Comet assay) in brain cells and micronucleus frequency in bone cells. Changes in anti-oxidative enzymes and increased lipid peroxidation.
*Reese et al. (1998)	Chinese hamster ovary (CHO) cells exposed to 60-Hz magnetic fields (0.1 or 2 mT), electric fields (1 or 38 V/m), or combined magnetic and electric fields (2 mT and 38 V/m, respectively) for 1 h	No significant effect on DNA single strand breaks (Comet assay) from exposures.
Reyes-Guerrero et al. (2010)	Adult male and female Wistar rats exposed to a 60-Hz magnetic field at 1 mT for 2 h/day for 9 days	ELF EMF modulates estrogen receptor- beta gene expression in the olfactory bulb of female adult rats but not in males.
Robison et al. (2002)	HL-60, HL-60R, and Raji cell lines exposed to a 60-Hz EMG at 0.15 mT for 24 h	EMF exposure offers significant protection from apoptosis (DNA double strand breaks (Comet assay)) and significantly decreased DNA repair rates in HL-60 and HL-60R cell lines but not in the Raji cell line.
*Ross et al. (2018)	Human mesenchymal stromal cell exposed to a 5-Hz EMF at 0.4 mT for 20 min/day, 3 times a week for 2 weeks	No chromosome breaks, viability and proliferation rate detected.
*Ruiz-Gómez et al. (2010)	Wild type (wt) and radiation sensitive mutant yeast strains (Saccharomyces cerevisiae) exposed to a	The exposure did not induce alterations in cell cycle and cause DNA damage.

	50 Hz magnetic field at 2.45 mT for 96 h	
Sadri et al. (2017)	Human mesenchymal stem cells derived from human newborn cords exposed to a static magnetic field of 12, 18, or 24 mT for 2 h	Induced differentiation and decreased expression of Sox-2, Nanog, and Oct-4 genes (These genes are involved in embryonic organ development, maintenance of multipotency and self renewal of undifferentiated embryonic stem cell.)
Sanie-Jahromi et al. (2016)	Human breast adenocarcinoma MCF-7 and neuroblastoma SH-SY5Y cells exposed to 50-Hz EMF at 0.25 and 0.5 mT (5 min ON/5min OFF; 15 min ON/15min OFF, or 30 ON continuously) for 30 min	mRNA levels of seven genes involved in DNA repair pathways down regulated in MCF-7 cells. Synergistic with cisplatin in MCF-7 and SH-SY5Y cells.
Sanie-Jahromi and Saadat (2017)	MCF-7 and SH-SY5Y cells exposed to an intermittent (15 min ON/15-min OFF) 50-Hz EMF at 0.5 mT for 30 min. Cells were also treated with cisplatin and bleomycin	EMF exposed MCF-7 cells treated with cisplatin and bleomycin showed more effects on some DNA repair gene expression compared with "cisplatin and bleomycin" treatment alone, while SH-SY5Y susceptibility was not changed between the two treatments.
Sanie-Jahromi and Saadat (2018)	MCF-7 and SH-SY5Y cells were treated with 5.0 μ M morphine and exposed to an intermittent (15 min ON/15 min OFF) 50-Hz EMF at 0.50 mT for 30 min	Morphine treatment showed significant down-regulation of expression of genes involved in DNA repair pathways, while in "Morphine + EMF" treatment, the genes were not significantly changed.
Sarimov et al. (2011)	Human lymphocytes exposed to 50-Hz magnetic field at 0.005-0.02 mT for 15-180 min	Magnetic field condensed relaxed chromatin and relaxed condensed chromatin.
*Scarfi et al (2005)	Human diploid fibroblasts exposed to an intermittent (5 min ON/10 min OFF) 50-Hz EMF or a 50-Hz field plus its harmonics for 24 h (1,2,4-BT) also studied	No significant effects on DNA single strand breaks (Comet assay) and micronucleus frequency.

Scassellati Sforzolini et al. (2004)	Cells exposed to a 50-Hz magnetic field at 5 mT; co-genotoxic effects with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), 4-nitroquinoline N-oxide (4NQO), benzene, 1,4-benzenediol (1,4-BD), or 1,2,4-benzenetriol	Magnetic field showed genotoxic (micronucleus test) and co-genotoxic (comet assay) capabilities.
Schmitz et al. (2004)	Male adult mice exposed to a 50-Hz magnetic field at 1.5 mT for 8 weeks	A significant increase in both unscheduled DNA synthesis and in situ nick translation was only found for epithelial cells of the choroid plexus. Mitochondrial DNA synthesis was exclusively increased in renal epithelial cells of distal convoluted tubules.
Seong et al. (2014)	Human bone marrow-mesenchymal stem cells exposed to a 50 Hz EMF at 1 mT for 8 days	Increased expression of early growth response protein 1 (Egr1).
*Shen et al. (2016)	Chinese Hamster Lung cells exposed to a 50-Hz EMF at 0.4mT for 30 min or 24 h	Increase in LC3-II expression and increased autophagosome formation; no significant effect on γ H2AX foci.(EMF-induced autophagy may balance the cellular homeostasis to protect the cells from severe adverse biological consequences.)
Shokrollahi et al. (2018)	Soybean plants exposed to static magnetic field at 20 and 30 mT for 5 h/day for 5 days	Exposure to 20 mT decreased gene expression of Fe transporter, ferrous and H ₂ O ₂ contents and gene expression, content and activity of ferritin and catalase. Opposite responses were observed at 30 mT exposure. Tertiary structures of ferritin, apoferritin and catalase altered by static magnetic field.
Singh and Lai (1998)	Rats exposed to a 60-Hz magnetic field at 0.5 mT for 2 h	Data suggested that both DNA-protein and DNA-DNA crosslinks (Comet assay) were formed in brain cells.
Skyberg et al. (2001)	Blood samples from high voltage laboratory workers exposed to electromagnetic fields and mineral oil	In inhibited (hydroxyurea-inhibits DNA synthesis, and caffeine-inhibits DNA repair) lymphocyte cultures, there were indications that electromagnetic fields in combination with mineral oil exposure may produce chromosomal aberrations. No effect on uninhibited cells.

Solek et al. (2017)	Mouse spermatogenic cell lines (GC-1 spg and GC-2 spd) exposed to pulsed (1sec on/off) or continuous-wave 2, 50, 120 Hz EMF at 2.5- 8 mT for 2 h	EMF activated oxidative and nitrosative stress-mediated DNA damage pathways, resulting in p53/p21-dependent cell cycle arrest and apoptosis
*Song et al. (2018)	HeLa and primary IMR-90 fibroblasts exposed to a 60-Hz EMF at 1, 3, 6, or 10 or mT continuously for up to 168 h or 30 min every 24h for 3 days	No effect on DNA damage (gamma-H2AX foci).; promoted cell proliferation (probably due to decreased reactive oxygen species).
Stankevičiūtė et al. (2019)	Rainbow trout (<i>Oncorhynchus mykiss</i>) exposed to a 50-Hz EMF at 1 mT for 40days; and the common ragworm (<i>Hediste diversicolor</i>) and the Baltic clam (<i>Limecola balthica</i>) for 12 days	Trout and ragworm erythrocytes and clam gill cells showed elevated micronucleus frequency, nuclear buds, nuclear buds on filament cells, and cells with blebbed nuclei.
*Stronati et al. (2004)	Human whole blood exposed to a 50-Hz magnetic field at 1 mT for 2 h	No significant effects on DNA single strand breaks (Comet assay), sister chromatid exchanges, chromosome aberrations, and micronucleus frequency in lymphocytes. A slight decrease in cell proliferation observed.
*Sun C et al. (2018)	ATM-proficient (Atm ^{+/+}) and ATM-deficient (Atm ^{-/-}) mouse embryonic fibroblasts exposed to a 50-Hz magnetic field at 2 mT for 15 min.(Ataxia telangiectasia mutated (ATM) plays a central role in DNA damage repair.)	No effect on γ -H2AX foci in both types of cells.
Sun L et al. (2019)	Irpex lacteus, a white-rot fungus, exposed to a 50-Hz magnetic field ay 3.5 mT for 3 h/day for 4 days	Global gene expression changes were observed.
Sun RG et al.(2012)	K562 human leukemia cells exposed to	The potency of the combination of SMF and paclitaxel was greater than that of SMF or

	paclitaxel in the presence or absence of 8.8 mT static magnetic field for 24 h	paclitaxel alone on K562 cells, and these effects were correlated with DNA single strand breaks (Comet assay).
Suzuki et al. (2001)	Mouse exposed to high intensity static magnetic fields (3.0 T for 48 and 72 h and 4.7 T for 24, 48 and 72 h).	Increased micronucleus frequency in bone marrow cells.
Svedenstal et al. (1999)	Brain cells of CBA mice exposed to a 50 Hz magnetic field at 0.5 mT 2 h, 5 days or 14 days	DNA single strand breaks (Comet assay) increased after 14 days of exposure,
*Szerencsi et al. (2013)	Peripheral blood samples from men exposed to EMF produced by 3T magnetic resonance imaging equipment for 0, 22, 45, 67, and 89 min during the scanning procedure	No significant effect on DNA single strand breaks (Comet assay) and DNA integrity in lymphocytes.
Teodori et al. (2014)	Human glioblastoma cells exposed to static magnetic field at 80 mT for 6,12, or 24 h, also in combination with X-ray	Increased in DNA single strand breaks (Comet assay) after 24 h of exposure; x-ray induced DNA strand breaks significantly reduced by post-irradiation exposure to static magnetic field. Further data suggested that static magnetic field modulated DNA damage and/or repair, possibly through a mechanism that affects mitochondria.
*Testa et al. (2004)	Human blood samples exposed to a 50-Hz magnetic field at 1 mT for 48 h	No significant effect on micronucleus frequency and proliferation of lymphocytes. No interaction with x-ray.
*Tiwari et al. (2015)	Blood samples of human subjects occupationally exposed to 132 kV high-voltage substations (mean duration on job 9.27 years, range 2-30 years).	No significant effect on DNA single strand breaks (Comet assay) in lymphocytes, increased oxidative stress observed.
Udroiu et al. (2006)	Liver and peripheral blood sampled from newborn mice exposed to a 50-Hz magnetic field of 0.65 mT during the	Data obtained in newborn mice showed a significant increase in micronuclei frequencies. No significant effect was recorded on exposed adults.

	whole intra-uterine life (21 days), and on bone marrow and peripheral blood from adult mice exposed to the same magnetic field for the same period	
Udroiu et al. (2015)	Mice exposed to 50-Hz, 0.065 mT magnetic field, 24 hours/day, for a total of 30 days, starting from 12 days post-conception	Magnetic field induced a slight genotoxic damage (micronucleus formation) and no interaction with x ray in erythrocytes, but modulate the response of male germ cells to X-rays with an impact on proliferation/differentiation processes. Magnetic field exposure decreased DNA single and double strand breaks (Comet assay) in germ cells at 42 days after birth.
*Verschaeve et al. (2011)	<i>Salmonella typhimurium</i> exposed to a 50-Hz magnetic field at 0.1 or 0.5 mT for 1 or 2 h	The magnetic field did not induce mutagenicity in <i>S. typhimurium</i> bacteria and did not show any synergetic effect when combined with chemical mutagens.
*Verschaeve et al. (2016)	<i>Salmonella typhimurium</i> exposed to 50 Hz magnetic field at 0.1 mT for 1 h	The magnetic field did not damage DNA and had no influence on the DNA damaging capacity of several mutagens.
Villarini et al. (2006)	Human leukocytes exposed to a 50-Hz magnetic field at 3 mT for 30, 60, or 120 min and treated with mutagens	Magnetic field exposure increased N-methyl-N'-nitro-N-nitrosoguanidine and decreased 4-nitroquinoline N-oxide-induced DNA single strand breaks (Comet assay).
Villarini et al. (2013)	Male CD1 mice exposed to a 50-Hz magnetic field at 0.1, 0.2, 1 or 2 mT for 7 days (15 hours/day) and sacrificed either at the end of exposure or after 24 h	Magnetic field exposure induced DNA single strand breaks (Comet assay) and did not affect hsp70 expression in the brain.
Villarini et al. (2015)	Blood leukocytes from electric arc welders presumably exposed to 50-Hz EMF (mean 0.0078 mT; range: 0.00003-0.171 mT)	Decreased DNA single strand breaks (Comet assay), may be caused by DNA-protein crosslinks by metal exposure.
*Villarini et al. (2017)	SH-SY5Y and SK-N-BE-2 human neuroblastoma cells	or AlCl ₃ alone induced DNA single strand breaks (Comet assay), changes in GSH/GSSG ratio or variations in Hsp70

	exposed to a 50-Hz magnetic field at 0.01, 0.1, or 1 mT for 1 h continuously or 5 h intermittently (15 min ON/15 min OFF), and also aluminum	expression. Co-exposure to ELF-MF and AlCl ₃ did not have any synergic toxic effects.
Wahab et al. (2007)	Human peripheral blood lymphocytes exposed to 50 Hz sinusoidal (continuous or pulsed) or square (continuous or pulsed) magnetic fields at 0.001 or 1 mT for 72 h	A significant increase in the number of sister chromatid exchange /cell observed.
*Wang Y et al. (2019)	Human ventricular cardiomyocytes exposed to a 50-Hz magnetic field at 0.1 mT for 1 h continuously or 75 min intermittently (15 min ON/15 min OFF). Sprague-Dawley rats exposed to 50 Hz magnetic field at 0.1 mT for 15 h/day for 7 days	Magnetic field exposure did not cause DNA single strand breaks (Comet assay) in heart cells in both in vitro and in vivo experiments.
Wang Y. et al. (2020)	<i>Caenorhabditis elegans</i> exposed to 50-Hz, 3 mT EMF for 15 generations	Expression levels of the <i>r53.4</i> , <i>hpo-18</i> , <i>atp-5</i> , and <i>atp-3</i> genes encoding ATPase and <i>sod-1</i> , <i>sod-2</i> , and <i>sod-3</i> genes encoding superoxide dismutase (SOD) were significantly upregulated.
Wang Z et al. (2009)	Human embryoid body derived (hEBD) LVEC cell line exposed to 0.23-0.28 T static magnetic field for 24 h	Gene expression in cells showed nine signaling networks responded to static magnetic field
*Williams et al. (2006)	Salmonella bacteria cultures exposed to a 60-Hz intermittent magnetic field (5 min ON/10 min OFF) at 14.6 mT for 4 h	No significant increase in recombination events and DNA single and double strand breaks (assayed using a recombination event counter). However, magnetic field exposure induced protection from heat stress.
Wilson et al. (2015)	BALB/c×CBA/Ca F1 hybrid males exposed to 50Hz magnetic fields at 0.01, 0.1 or 0.3 mT for 2 or 15 h	There was a marginally significant increase in a non-dose-dependent mutation frequency in sperm, and not in blood cells.

Winker et al. (2005)	Human fibroblasts exposed to a 50-Hz intermittent (5 min ON/10 min OFF) EMF at 1 mT for 2-24 h	Increased micronucleus frequency and chromosomal aberration.
Wolf et al. (2005)	HL-60 leukemia cells, Rat-1 fibroblasts, and WI-38 diploid fibroblasts exposed to a 50-Hz EMF at 0.5-1 mT for 24-72 h	Dose-dependent increases in DNA single strand breaks (Comet assay) and formation of 8-hydroxy-2'-deoxyguanosine adducts were observed in all cell lines. There were increases in cell proliferation and reactive oxygen species.
Yagci and Kesim (2016)	Human gingival fibroblasts exposed in vitro to static magnetic fields produced by dental magnetic attachments for 10-12 days. (The maximum magnetic flux densities measured at the magnet centers of 4 types of attachment were 95.6-148.1 mT and became almost zero at 10 mm away)	Increased micronucleus frequency.
Yaguchi et al. (1999)	Mouse embryonic skin m55 cells exposed to a 60-Hz magnetic field at 5, 50, or 400 mT for 42 h	Increase in sister chromatid exchanges after 400 mT exposure.
Yaguchi et al. (2000)	Mouse embryonic skin m55 cells exposed to 60-Hz (5 or 50 mT) or 50-Hz (400 mT) magnetic fields for 40 h. Some cells also treated with mitomycin C or X-ray	Increased chromosomal aberration, synergistic with mitomycin C and X-ray.
Yao et al. (2015)	Rat Schwann cells exposed to DC electric field for 36-72 h at 50, 100, or 200 mV/mm	Differential expression of genes participate in multiple cellular signaling pathways involved in the regulation of cell migration, including pathways of regulation of actin cytoskeleton, focal adhesion, and PI3K-Akt cell cycle regulation).
Yin et al. (2016)	Primary cultured rat hippocampal neurons exposed to a 50-HZ EMF at 1 mT for 90 min	Increase in DNA single strand breaks (Comet assay); free radicals involved.

Yokus et al. (2005)	Female Wistar rats exposed to a 50-Hz magnetic field at 0.97 mT for 3 h/day for 50 and 100 days	Increased 8-hydroxy-2'-deoxyguanosine in blood cells.
Yokus et al. (2008)	Male Sprague-Dawley rats exposed to a 50-Hz magnetic field at 0.1 or 0.5 mT for 2 h/day for 10 months	Increased DNA base modifications in leucocytes [8-hydroxyguanine (8-OH-Gua), 2,6-diamino-4-hydroxy-5-formamidopyrimidine (FapyGua), and 4,6-diamino-5-formamidopyrimidine (FapyAde)]
Yoon et al. (2014)	Human lung fibroblast WI38 cells and human lung epithelial L132 cells exposed to a 60-Hz magnetic field at 2 mT for 6 h	2 mT field induced increased γ -H2AX expression, as well as γ -H2AX foci production. Interacted with gamma radiation but not H ₂ O ₂ .
Yuan et al. (2020)	Tumor cell lines including lung cancer, gastric cancer, pancreatic cancer and nephroblastoma exposed to a 50-Hz EMF modulated by static MF with time-average intensity of 5.1 mT, for 2 h/day for 3 days	Induced DNA single strand breaks (Comet assay), gamma-H2AX and activation of DNA repair pathways, increased reactive oxygen species and ferroptosis, and decreased proliferation.
Zendehdel et al. (2019)	Peripheral blood cells of male power line workers in a power plant. The median value of the magnetic field at the working sites was 0.00085 mT	Increased in DNA single strand breaks (Comet assay).
Zhang H et al. (2016)	ICR mice exposed to a 50-Hz EMF at 8 mT for 4 h/day for 28 days	Declined DNA content and increased expression of apoptosis genes in spleen. Free radical may be involved.
Zhang Y et al. (2016)	Workers with or without exposure to ELF-EMF (50 Hz) of 110-420kV power lines	Significant increased urinary 8-isoprostane and 8-OHdG were observed in workers with EMF exposure. Free radical may be involved.
Zheng et al. (2018)	dental pulp stem cells exposed to a static magnetic field of 1,2, 4 mT for 15 min, 30 min, 1 h or 24 h	Increased expression of several growth factors (FGF-2, TGF- β , and VEGF), migration genes (MMP-1 and MMP-2), and upregulated the two YAP/TAZ-regulated genes, CTGF and ANKRD1. (YAP/TAZ are transcriptional activators particularly

		involved in cancer cell proliferation, therapy resistance and metastasis. Increased cell proliferation, osteo/odontogenesis and mineralization observed in the stem cells.
*Zhu et al. (2016)	Human lens epithelial cells exposed to a 50-Hz magnetic field at 0.4 mT for 2, 6, 12, 24, or 48 h	No effect on DNA single strand breaks (Comet assay) and gamma-H2AX foci.
Zmyslony et al. (2000)	Rat exposed to a static or 50-Hz magnetic field at 7 mT for 3 h	In combination with FeCl ₂ , increases in DNA single strand breaks (Comet assay) observed for both static and 50-Hz field exposure in lymphocytes.
Zmyslony et al. (2004)	Rat lymphocytes exposed first to ultraviolet radiation and then to a 50-Hz magnetic field at 0.04 mT for 5 or 60 min	60-min magnetic field exposure (plus UVA) caused an increase in DNA single strand breaks (Comet assay). MF may affect the radical pairs generated during the oxidative or enzymatic processes of DNA repair.

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Supplement 3

Gene expressions after RFR and static/ELF EMF exposure (literature up to January 2021)

<i>RFR</i>	<i>Exposure effects</i>
Akhavan-Sigari et al. (2014)	Increased risk for the mutant type of p53 gene expression in the peripheral zone of the glioblastoma, and that this increase was significantly correlated with shorter overall survival time.
Beaubois et al. (2007)	Accumulation of basic leucine-zipper transcription factor (bZIP) mRNA in the exposed terminal leaf of tomato plant.
Belyaev et al. (2006)	Expression of genes encode proteins with diverse functions including neurotransmitter regulation, blood-brain barrier (BBB), and melatonin production in rat brain.
Buttiglione et al. (2007)	Affected both Egr-1 gene expression and cell regulatory functions, involving apoptosis inhibitors like Bcl-2 and surviving in human neuroblastoma cells.
Cervellati et al. (2013)	Induced 17- β -estradiol modulates connexins and Integrins as well as estrogen receptor (ER- β) expression in trophoblast cells, suggesting an influence on cell differentiation and migration.
Chen et al. (2012)	Expression was limited to only a very small number of genes in yeast. (Expressions of structural maintenance of chromosomes 3 (SMC3) and aquaporin 2 (AQY2 (m)) while halotolerance protein 9 (HAL9), a kinase 1 (YAK1) and one function-unknown gene showed opposite changes in expression.
Del Vecchio et al. (2009)	Increased expression of beta-thymosin gene, a cytoskeleton regulating factor in murine cortical neurons, correlated to reduced number of neurites generated.
Deshmukh et al. (2015)	Increased heat shock protein 70 (HSP70) in rat brain.
Eker et al. (2018)	Caspase-3 and p38 mitogen-activated protein kinase (p38MAPK) (a kinase responsive to stress stimuli, and involved in cell differentiation, apoptosis and autophagy) gene expressions were significantly up-regulated in the ocular tissues of rat.
Engelmann et al. (2008)	Significant changes in transcription of 10 genes in <i>Arabidopsis thaliana</i> (thale cress) cells.
Fragopoulou et al. (2018)	Expression of 178 genes changed significantly mouse hippocampus
Franzellitti et al. (2008)	Levels of the inducible HSP70C transcript were significantly enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals in human trophoblasts.
Ghatei et al. (2017)	No effect on expression level of bcl-2 and p53 genes, but gene expression level of <i>bax</i> decreased and gene expression level of <i>p21</i> increased in cerebellum of mice exposed pre-and postnatally to RFR.
Gökçek-Saraç et al. (2020)	Decreased RNA expressions of acetylcholinesterase (AChE), choline acetyltransferase (ChAT), and vesicular acetylcholine transporter (VAcHT)

	in the hippocampus of rats exposed to 2100 MHz RFR.
Gulati et al. (2018)	A significant association of genetic polymorphism of antioxidant genes (for MnSOD and CAT) with oxidative damage has been observed in human population exposed to radiations emitted from mobile towers.
Habauzit et al. (2014)	7 genes were differentially expressed in human keratinocytes, associated to the cellular response to hyperthermia.
Hao et al. (2010)	Significant induced phosphorylation of STAT3, increased transcription levels of the inflammation-associated genes, iNOS and TNF-alpha murine N9 microglial cells. (Signal transducer and activator of transcription 3 (STAT3) is a transcription activator that mediates the expression of a variety of genes in response to cell stimuli, and thus plays a key role in many cellular processes such as cell growth and apoptosis.)
He et al. (2016)	Mouse bone marrow stromal cells showed increased PARP-1 mRNA expression.(PARP-1 involves in differentiation, tumor transformation and DNA repair.)
He et al. (2017)	Mouse bone marrow stromal cells showed increased PARP-1 mRNA expression. Gamma radiation decreased RFR-induced PARP-1 expression.
Karaca et al. (2012)	Decreased STAT3 expression in mouse brain. (STAT3 acts as transcription activator).
Kumari et al. (2017)	Increased expression of the pro-inflammatory cytokine tumor necrosis factor alpha mRNA in the hippocampal region.
Kumar, R. (2020)	Altered expression of DNA (epigenetic) methylating enzymes, DNA methyltransferase1 (DNMT1) and histone methylating enzymes euchromatic histone methyltransferase1 (EHMT1) in hippocampus.
Jeong et al. (2020)	Increased expression of Epha8 and Wnt6 genes in the hippocampi of mice. (Both genes are involved in development, particularly, Epha8 coded protein mediates developmental events in the nervous system in axonal guidance).
Lameth et al. (2020)	Altered gene expressions in rat cerebral cortex in an acute neuroinflammation. Gene responses to RFR can differ according to pathologies affecting the CNS.
Le Quément et al.(2012)	Human skin cells showed differential expression of genes involved in functions such as cardiovascular development, facilitate pathogen recognition by macrophages, inhibition of angiogenesis, nonspecific ion channels, etc.
Lee et al. (2005)	Many genes were affected in human HL60 cells. Apoptosis-related genes were among the upregulated ones and the cell cycle genes among the downregulated ones.
Li et al. (2020)	Offspring of pregnant female rats exposed to RFR showed differential expression of methyl-D-aspartate receptors

	(NMDARs) genes in the hippocampus.
Lin et al. (2016)	Upregulated the expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway. Transcription levels of 29 genes were upregulated and 24 genes were downregulated.
López-Martín et al. (2009)	c-Fos expressions in brain of picrotoxin-treated and untreated rats.
Manta et al. (2017)	168 genes differentially expressed in the house fly <i>Drosophila melanogaster</i> , associated with multiple and critical biological processes, such as basic metabolism and cellular subroutines related to stress response and apoptotic death.
Martin et al. (2020)	Four different types of human keratinocytes showed different patterns of expression of ADAMTS6, IL7R, and NOG genes
Megha et al. (2015)	Downregulation in mRNA expression of enzymes involved in monoamine transmitter synthesis in rat hippocampus.
Mildažienė et al. (2019)	Leaves from exposed common sunflower (<i>Helianthus annuus</i> L.) seeds showed gene expression mostly of proteins involved in photosynthetic processes and their regulation.
Millenbaugh et al. (2008)	Genes associated with regulation of transcription, protein folding, oxidative stress, immune response, and tissue matrix turnover were affected in rat skin.
Nittby et al. (2008)	Altered gene expression in both cortex and hippocampus of the rat: extracellular region, signal transducer activity, intrinsic to membrane, and integral to membrane.
Nylund and Leszczynski (2006)	Gene and protein expressions altered differentially in two human endothelial cell lines.
Ohtani et al. (2016)	Heat-shock proteins (Hsp) and heat-shock transcription factors (Hsf) gene expression levels were significantly upregulated in the cerebral cortex and cerebellum of the rat.
Ohtani et al. (2019)	No change in transcription gene expression in brain and liver of mice exposed to a 85-kHZ field.
Pacini et al. (2002)	Human skin fibroblasts showed increased expression of mitogenic signal transduction genes (e.g., MAP kinase kinase 3, G2/mitotic-specific cyclin G1), cell growth inhibitors (e.g., transforming growth factor-beta), and genes controlling apoptosis (e.g., bax).
Qin et al. (2018)	Altered the expression of genes involved in testosterone synthesis (<i>Star</i> , <i>P450scc</i> , <i>P450c17</i> and <i>3β-Hsd</i>) in mouse testicular tissue.
Qin et al. (2019)	Exposed Leydig cells showed downregulated of testosterone synthase genes (<i>Star</i> , <i>Cyp11a1</i> , and <i>Hsd-3β</i>) and clock genes

	(<i>Clock</i> , <i>Bmall</i> , and <i>Rora</i>),
Rammal et al. (2014)	Increased expression of two wound-plant gene in tomato.
Remondini et al. (2006)	Different human cell types responded differently in gene expression. Affected gene families did not point towards a stress response, but suggested upregulating of cellular metabolism.
Romano-Spica et al. (2000)	Overexpression of the proto-oncogene ets1 mRNA in Jurkat T-lymphoblastoid and Leydig TM3 cell lines
Roux et al. (2006)	Leaves of tomato plants showed increased stress-related transcripts (calmodulin, protease inhibitor and chloroplast mRNA-binding protein).
Roux et al. (2008)	Tomato plant showed increase in stress-related mRNA (calmodulin, calcium-dependent protein kinase and proteinase inhibitor), similar to wound responses.
Said-Salman et al. (2019)	101 genes were differentially expressed in Escherichia coli. Up-regulated genes are involved in metabolic pathways, transposition, response to stimuli, motility, chemotaxis, and cell adhesion, while the down-regulated genes are associated with metabolic pathways and localization of ions and organic molecules.
Silva et al. (2016)	No effect on expressions of Ki-67 (involved in cell proliferation) p53 (tumor suppression) HSP-70 (stress biomarker), and reactive oxygen species in human thyroid cells.
Soubere Mahamoud et al. (2016)	Exposed human keratinocytes treated with the glycolysis inhibitor, 2-deoxyglucose showed changes in genes encode transcription factors or inhibitors of cytokine pathways,
Souza et al. (2014)	Cells from oral mucosa of individual used cellular phones more than 5 h/week high number of broken egg which may be associated with gene amplification.
Sun Y. et al. (2017)	Decreased gene expression in mitochondria of HL-60 human leukemia cells. Free radicals involved.
Tohidi et al. (2020)	Apoptotic genes Bax and Bc12 expression in the hippocampus were upregulated in mice exposed to RFR from a cell phone jammer for 1, 2, twice a day for 30 days and down-regulated with longer exposure schedule.
Trivino Pardo et al. (2012)	Gene expression affected in acute T-lymphoblastoid leukemia cells. Genes which act as sensors of DNA damage (<i>ATM</i> , <i>RAD17</i> , <i>RAD50</i> , and <i>PRKDC</i>) are activated. This over-expression could produce a signal cascade that causes the activation of the main DNA repair signaling. Some of the genes that were defined as essentials in double-strands repair

	(<i>BRCA1, LIG4, XRCC2</i>) and single-chain DNA repair process (<i>XPC, MSH5</i>) were found to over-express. More cells in S-phase.
Vafaei et al. (2020)	Increased superoxide dismutase, CDKN1A, GADD45a, Bax mRNA, and decreased Bcl-2 mRNA. (CDKN1A and GADD45a are involved in DNA repair and cellular responses to stressors.)
Valbonesi et al. (2014)	HSP70 transcription was significantly increased in rat neuronal-like PC12 cells.
Varghese et al. (2018)	Increased caspase-3 gene expression in brain tissues of rats exposed to 2450 MHz RFR
Vian et al. (2006)	Rapid induction of mRNA encoding the stress-related bZIP transcription factor in plants.
Yan et al. (2008)	Brain of exposed rat showed mRNA up-regulation of several injury-associated proteins. RFR exposure may result in cumulative injuries that could eventually lead to clinically significant neurological damage.
Yao et al. (2004)	Rabbit lens epithelial cells showed increased expression of P27kip1 protein, also G/G1 cell cycle arrest. (p27kip1 is a cyclin-dependent kinase inhibitor which binds to cyclinE/cdk2, blocking the G1/S transition.)
Zhang et al. (2008)	Primary culture neurons showed gene up- and down-regulation. Genes are associated with multiple cellular functions (cytoskeleton, signal transduction pathway, metabolism, etc.)
Zhao et al. (2007)	Up-regulation of caspase-2, caspase-6 genes occurred in both GSM 1900-MHz "on" and "stand-by" modes in neurons, but only in "on" mode in astrocytes. Additionally, astrocytes showed up-regulation of the Bax gene.
<i>Static/extremely-low frequency EMF</i>	
Agliassa et al. (2018)	Near-null MF condition (i.e., <100 nT) delayed transition to flowering in <i>Arabidopsis thaliana</i> and changes in expression of several genes in leaf and floral meristem.
Ashta et al. (2020)	Temozolomide (TMZ) with static MF or ELF MF (10 Hz) together increased p53 protein expression in the human glioblastoma cell line (A172) and increased cytotoxicity.
Baraúna et al. (2015)	The bacteria <i>Chromobacterium violaceum</i> , exposed to ELF MF, showed differential expression of 5 proteins. Expression of the protein, DNA-binding stress protein, may help to prevent DNA damage.
Bertea et al. (2015)	Exposing <i>Arabidopsis thaliana</i> to artificially reversed

	geomagnetic field conditions induced gene expressions.
Chen et al. (2008)	Human breast cancer MCF-7 cells exposed to a 50-Hz MF induced expression of three responsive genes.
Chen et al. (2020)	Human choriocarcinoma cells exposed to DC electric field showed increased gene expressions of ErbB and HIF-1 signaling pathways
Collard et al. (2013)	Epidermis cultures harvested from human abdominoplasty exposed to ELF electric fields induced expression of various genes. Some genes are involved in cell proliferation or differentiation, mitosis, cell cycle, or in the DNA replication transcription and translation.
Consales et al. (2018)	Exposure to a 50-Hz magnetic field in vitro. We demonstrate that ELF-MFs drive an early reduction of the expression level of miR-34b and miR-34c in SH-SY5Y human neuroblastoma cells, as well as in mouse primary cortical neurons, by affecting the transcription of the common pri-miR-34. Data also indicate epigenetic control of gene expression in vitro and shed light on the possible mechanism(s) producing detrimental effects and predisposing neurons to degeneration.
Cuccurazzu et al. (2010)	Exposure to a 50-Hz MF in vivo induced increases in the transcription of pro-neuronal genes (Mash1, NeuroD2, Hes1) and genes encoding Ca(v)1.2 channel α (1C) subunits in the hippocampus of the mouse. Hippocampal neurogenesis also observed.
Del Re et al. (2006)	ELF-MF influenced the synthesis of heat shock proteins in <i>E. coli</i> in a way that critically depends on the signal characteristics (static or pulsed MF).
Di Campli et al. (2010)	<i>Helicobacter pylori</i> biofilm exposed to a 50-Hz EMF showed <i>amiA</i> gene expression and decreased cell adhesion. (AmiA protein is responsible for transition of <i>H. pylori</i> from bacillary to coccoid forms. These coccoid forms can escape detection by the immune system and therefore could participate in the persistence of <i>H. pylori</i> infection during the lifetime of its human host.)
Dong et al. (2019)	16 T static magnetic field markedly blocked the expression of osteoclast-associated transcription factors and osteoclast marker genes and inhibited iron absorption and iron storage-related protein expression.
Fan et al. (2015)	Rat bone marrow derived-mesenchymal stem cells and mesenchymal stem cells exposed to a 50-Hz EMF induced expressions of various genes. Expressions of hematopoietic growth factors increase proliferation and migration of macrophagocytes.
Fan et al. (2018)	Static magnetic field up-regulated the expression of stress gene (<i>dnaK</i>) and virulence genes (<i>efaA</i> and <i>ace</i>).
Fedrowitz and Loscher	50-Hz MF-exposed F344 rat breast tissue showed alterations in

(2012)	gene expression, which were absent in Lewis rats.
Frisch et al. (2013)	Rat primary fibroblasts exposed to a 10-Hz electric fields induced HSP70 expression.
Heredia-Rojas et al. (2010)	“Electromagnetic field” plasmid transfected into INER-37 and RMA E7 cell lines exposed to a 60-Hz MF. An increased luciferase gene expression was observed in INER-37 cells but had no effect on the RMA E7 cell line.
Jin et al. (2019)	Arabidopsis seedling exposed to static magnetic field showed increased auxin (a plant growth hormone) from expression of PIN3 and AUX1 genes in root tips; cryptochromes (cry1 and cry 2 genes) are also involved.
Ki et al. (2020)	Human hair follicle dermal papilla cells exposed to a 70-Hz EMF enhance cell activation and proliferation via the GSK-3 β /ERK/Akt signaling pathway. Various genes were activated.
Kim et al. (2010)	Human normal and cancer cells exposed repeatedly to a 60-Hz MF showed p38 gene expression and induction of checkpoint kinase 2 critical to the DNA damage checkpoint pathway.(P38 mitogen-activated protein kinases are a class of mitogen-activated protein kinases (MAPKs) that are responsive to stress stimuli, and are involved in cell differentiation, apoptosis and autophagy.)
Kimsa-Dudek et al. (2018)	Static magnetic field attenuated expression of antioxidant defense genes (SOD1, PLK3, CLN8, XPA, HAO1) induced by sodium fluoride.
Kimsa-Dudek et al. (2020)	Exposure of human fibroblast cultures that had been co-treated with fluoride ions to a static MF caused specific genes expression that were involved in apoptosis.
Kimura et a. (2008)	Caenorhabditis elegans exposed to high intensity (2, 3, 5 T) static magnetic fields showed induction of genes involved in motor activity, actin binding, cell adhesion, and cuticles; also upregulation of hsp (heat shock protein) 12 and 16 family genes.
Lacy-Hulbert et al. (1995)	No effect on MYC and beta-actin gene expression in human leukemic cells.
Laramee et al. (2014)	Transfected rat primary cells in monolayer were exposed to a static MF caused HSP expression.
Lee et al. (2010)	C. elegans exposed to a 200 mT static magnetic field showed up-regulation of genes involved in development and aging (clk-1,unc-3, age-1,daf-2, lim-7).
Lee et al. (2016)	MCF7 cells showed up-regulation of PMAIP1 gene (gene involved in apoptosis) after 60-Hz magnetic field exposure.
Leone et al. (2014)	ELF-EMF enhanced proliferation and neuronal differentiation of hippocampal neural stem cells by regulation of epigenetic mechanisms leading to pro-neuronal gene expression.
Li et al. (2013)	Male Drosophila melanogaster exposed to ELF-EMF showed changes in gene expression. Differentially expressed genes following short-term exposures were involved in metabolic processes, cytoskeletal organization, mitotic spindle organization,

	cell death, protein modification and proteolysis. Long-term exposure led to changes in expression of genes involved in metabolic processes, response to stress, mitotic spindle organization, aging, cell death, and cellular respiration.
Li et al. (2014)	Zebra fish embryos exposed to a 50-Hz MF showed transcription of apoptosis-related genes (caspase-3, caspase-9) was significantly upregulation.
Li et al. (2015)	Rat oligodendrocyte precursor cells exposed to DC electric field showed upregulated mitogen-activated protein kinase pathway that signals cell migration and downregulation of differentially expressed genes in chemotaxis.
Li et al. (2019)	Pulsed EMF (20 Hz) increased expression of insulin growth factor 2 (IGF-2) in the hippocampus of streptozotocin-induced dementia rats.
Lin et al. (2016)	Budding yeast exposed to a 50-Hz EMF caused upregulation expression of genes involved in glucose transportation and the tricarboxylic acid (TCA) cycle, but not the glycolysis pathway. (A response to environmental stress.)
Lupke et al. (2006)	Human umbilical cord blood-derived monocytes exposed to ELF-MF caused expression of 5 genes.
Ma et al. (2014)	Mouse embryonic neural stem cells exposed to a 50-Hz EMF induced expression of genes regulating neuronal differentiation although cell proliferation and the percentages of neurons and astrocytes differentiated from eNSCs were not affected which might be compensation by post-transcriptional mechanisms to support cellular homeostasis.
Mahaki et al. (2019)	A 50-Hz EMF reduced the expression levels of c-Maf, STAT6, and ROR α genes in the spleen of rats.
Mahmoudinasab and Saadat (2016)	Human MCF-7 breast cancer cells exposed to a 50-Hz EMF showed decreased NQO1 and increased NQO2 gene expression. (NQO1 and NQO2 are detoxification enzymes).
Mahmoudinasab and Saadat (2018a)	Patterns of up-regulation of antioxidant genes are different between MCF-7 and SH-SY5Y cells exposed to an intermittent 50-Hz EMF.
Mahmoudinasab and Saadat (2018b)	SH-SY5Y cells exposed to a 50-Hz EMF. NQO1 mRNA level decreased in the "15 min field-on/15 min field-off" condition, the expression level of NQO2 was increased.
Mahmoudinasab et al. (2016)	Human MCF-7 breast cancer cells exposed to a 50-Hz EMF showed up and down regulations of 7 antioxidant genes.
Manzella et al. (2015)	50-Hz magnetic field affected in human dermal fibroblasts expression of clock genes: BMAL1, PER2, PER3, CRY1, and CRY2.
Mastrodonato et al. (2018)	50-Hz EMF exposure increased Wnt3 (neurogenesis gene) mRNA expression in subventricular zone of the lateral ventricle of mice.
Moraveji et al. (2016)	50-Hz EMF activated MAP2 gene in dermal papilla mesenchymal

	cells.
Mouhoub et al. (2017)	Static magnetic field enhanced expression of gene involved in the production of acdiolipin and phosphatidylethanolamine in <i>Salmonella hadar</i> .
Nasrabadi et al. (2018)	In neonatal human retinal pigment epithelial cells exposed to pulsed 50-Hz EMF, gene expressions of NES, RPE65, and PAX6 were decreased. (NES gene encodes nestin involved in radial growth of neurons. The RPE65 gene provides instructions for making a protein that is essential for normal vision. PAX6 acts as a "master control" gene for the development of eyes and other sensory organs.)
Nikolova et al. (2005)	Mouse embryonic stem cells exposed to a 50-Hz EMF changed transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes.
Pesqueira et al. (2017)	Short term exposure (8 h) upregulated the expression of tendon-associated genes SCX, COL1A1, TNC and DCN. Long-term exposure (8 h every 24 h up to 14 days) significantly upregulated COL1A1, COL3A1 and TNC.
Potenza et al. (2004a)	<i>Escherichia coli</i> exposed to static magnetic field showed three cDNAs to be expressed only in the exposed cells, whereas one cDNA was more expressed in the controls.
Reyes-Guerrero et al. (2010)	ELF EMF exerted a biphasic effect on female olfactory bulb estrogen receptor-beta mRNA gene expression, which increased during diestrous and decreased during estrous. No effect on estrogen receptor-alpha gene expression and in male rats.
Sadri et al. (2017)	Static magnetic field decreased expression of Sox-2, Nanog, and Oct-4 genes in human mesenchymal stem cells derived from newborn umbilical cords.
Sanie-Jahromi et al. (2016)	Human MCF-7 breast cancer cells and neuroblastoma SH-SY5Y cells exposed to a 50-Hz EMF had mostly down regulation of 7 DNA repair genes in MCF-7 cells. Co-treatment with cisplatin and EMF can enhance down-regulation of the genes involved in non-homologous end-joining pathway in both cell types.
Sanie-Jahromi et al. (2017)	ELF-EMF enhanced the effects of cisplatin + bleomycin on viability of MCF-7 cells, while SH-SY5Y cells were not affected. MCF-7 and SH-SY5Y cells showed non-random disagreement in DNA repair gene expression in these conditions.
Sanie-Jahromi and Saadat (2018)	MCF-7 and SH-SY5Y cells were treated with morphine and then exposed to a 50-Hz EMF. Non-homologous end joining (NHEJ) related genes were significantly decreased in co-treatment of cisplatin and "morphine + EMF".
Seong et al. (2014)	Human bone marrow-mesenchymal stem cells were exposed to a 50-Hz EMF. Analysis of neurons derived from these cells showed that early growth response protein 1 (Egr1) is one of the key transcription factors in ELF-EMF-induced neuronal

	differentiation.
Shokrollahi et al. (2018)	Soybean plants exposed to static magnetic field had decreased gene expression of Fe transporter at 20 mT. Opposite response observed at 30 mT. The results suggest that SMF triggered a signaling pathway that is mediated by iron.
Wang et al. (2009)	Human embryonic cells exposed to static magnetic field showed a short-term (<24 h) activation of IL-6 involved the coordinate up-regulation of toll-like receptor-4 (TLR4) with complementary changes to NEU3 and ST3GAL5 that reduced ganglioside GM3 and augmented the activation of TLR4 and IL-6. Loss of GM3 also provided a plausible mechanism for the attenuation of cellular responses to SMF that occurred over longer exposure periods.
Wang et al. (2020)	Caenorhabditis elegans exposed to 50-Hz, 3 mT EMF for 15 generations showed enhanced up-regulations of genes encoding ATPase and superoxide dismutase.
Yao et al. (2015)	Rat Schwann cells exposed to DC electric field showed expression of genes participate in multiple cellular signaling pathways involved in the regulation of cell migration, including pathways of regulation of actin cytoskeleton, focal adhesion, and PI3K-Akt.
Zhang H et al. (2016)	Mice exposed to a 50-Hz EMF showed a significant suppression in Bcl-2 expression and increase in Bax, Caspase-3 and Caspase-9 expression in splenic cells. G0/G1 cycle arrest observed.
Zhao et al. (2020)	Escherichia coli exposed to 3.1 THz RFR for 8 h showed increased plasmid copy number and protein expression.
Zheng et al. (2018)	Static magnetic field increased expression of several growth factors, migration genes, and upregulated the two YAP/TAZ-regulated genes in human dental pulp mesenchymal stem cells.

Supplement 4

Genetic effects at low intensity exposure to RFR and static/ELF EMF (literature up to January 2021)

	Power density/SAR (<0.1 W/Kg) or magnetic flux density	Effects observed
<i>RFR studies</i>		
Aitken et al. (2005)	Mice to 900-MHz RFR for 7 days at 12 h/day; SAR 0.09 W/kg	Mitochondrial genome damage in epididymal spermatozoa.
Akdag et al. (2016)	Male Wistar-Albino rats to 2400 MHz RFR from a Wi-Fi signal generator for a year; SAR 0.000141 (min)-0.007127 (max) W/kg	DNA damage in testes.
Alkis et al. (2019a)	Rats exposed to 900 MHz (brain SAR 0.0845 W/kg), 1800 MHz (0.04563 W/kg), and 2100 MHz (0.03957 W/kg) RFR 2 h/day for 6 months	Increased DNA strand breaks and oxidative DNA damage in brain.
Alkis et al. (2019b)	Rats exposed to 900 MHz, 1800 MHz, and 2100 MHz RFR 2 h/day for 6 months; maximum SAR over the rat 0.017 W/kg	
Atasoy et al. (2013)	Male Wister rats exposed to 2437 MHz (Wi-Fi) RFR; 24 h/day for 20 weeks; maximum SAR 0.091 W/kg	Oxidative DNA damage in blood and testes.
Beaubois et al. (2007)	Leaves of tomato plant exposed to 900-MHz RFR for 10 min at 0.0066 mW/cm ²	Increased expression of leucine-zipper transcription factor (bZIP) gene.
Belyaev et al. (2005)	Lymphocytes from human subjects exposed to GSM 915 MHz RFR for 2 h ; SAR 0.037 W/kg;	Increased condensation of chromatin.

Belyaev et al. (2009)	Human lymphocytes exposed to UMTS cell phone signal (1947.4 MHz, 5 MHz band width) for 1 h; SAR 0.04 W/kg	Chromatin affected and inhibition of DNA double-strand break.
Bourdineaud et al. (2017)	Eisenia fetida earthworms exposed to 900 MHz for 2 h; SAR 0.00013-0.00933 W/kg	DNA genotoxic effect and HSP70 gene expressions up regulated.
Campisi et al. (2010)	Rat neocortical astroglial to CW 900 MHz RFR for 5, 10, or 20 min; incident power density 0.0265 mW/cm ²	Significant increases in DNA fragmentation.
Chaturvedi et al. (2011)	Male mice exposed to 2450 MHz RFR, 2 h/day for 30 days; SAR 0.03561 W/kg	Increased DNA strand breaks in brain cells.
Deshmukh et al. (2013)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 30 days.	Increased DNA strand breaks in brain tissues.
Deshmukh et al. (2015)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 180 days.	Increased DNA strand breaks in brain tissues.
Deshmukh et al. (2016)	Male Fischer rats exposed to 900 MHz (0.0005953 W/kg), 1800 MHz (0.0005835 W/kg), and 2450 MHz (0.0006672 W/kg) RFR for 2 h/day, 5 days/week for 90 days.	Increased DNA strand breaks in brain tissues.

Eker et al. (2018)	Female Wistar albino rats exposed to 1800-MHz RFR for 2 h/day for 8 weeks; SAR 0.06 W/kg	Caspase-3 and p38MAPK gene expressions increased in eye tissues.
Furtado-Filho et al. (2014)	Rats of different ages (0-30 days) exposed to 950 MHz RFR for 0.5 h/day for 51 days (21 days of gestation and 6-30 days old): SAR pregnant rat 0.01-0.03 W/kg; neonate 0.88 W/kg, 6-day old 0.51 W/kg, 15-day old 0.18 W/kg, 30-day old 0.06 W/kg.	Decreased DNA strand breaks in liver of 15-day old and increased breaks in 30-day old rats.
Gulati et al. (2016)	Blood and buccal cells of people lived close (<400 meters) to a cell tower; 1800 MHz, Maximum power density (at 150 meters) 0.00122 mW/cm ² , some subjects lived in the area for more than 9 yrs	Increased DNA strand breaks in lymphocytes and micronucleus in buccal cells.
Gürler (2014)	Wistar rats exposed to 2450 MHz RFR 1 h/day for 30 consecutive days; power density 0.0036 mW/cm ²	Increased oxidative DNA damage in brain and blood.
Hanci et al. (2013)	Pregnant rats exposed 1 h/day on days 13-21 of pregnancy to 900-MHz RFR at power density 0.0265 mW/cm ² .	Testicular tissue of 21-day old offspring showed increased DNA oxidative damage.
He et al. (2016)	Mouse bone marrow stromal cells exposed to 900 MHz RFR 3 h/day for 5 days; SAR 4.1 x 10 ⁻⁴ W/kg (peak), 2.5 x 10 ⁻⁴ W/kg (average)	Increased expression of PARP-1 mRNA

Hekmat et al. (2013)	Calf thymus exposed to 940 MHz RFR for 45 min; SAR 0.04 W/kg	Altered DNA structure at 0 and 2 h after exposure.
Kesari and Behari (2009)	Male Wistar rats exposed to 50 GHz RFR for 2 h/day for 45 days; SAR 0.0008 W/kg	Increased in brain tissue DNA strand.
Kumar R. et al. (2021)	Male Wistar rats exposed to 900 MHz, 1800 MHz and 2450 MHz RFR at a specific absorption rate (SAR) of 5.84×10^{-4} W/kg, 5.94×10^{-4} W/kg and 6.4×10^{-4} W/kg, respectively for 2 h per day for 1-month, 3-month and 6-month periods.	Epigenetic modifications in the hippocampus, bigger effects with increasing frequency and duration of exposure.
Kumar S. et al. (2010)	Male Wistar rats exposed to 10-GHz RFR for 2 h a day for 45 days, SAR 0.014 W/kg	Increased micronucleus in blood cells.
Kumar S. et al. (2013)	Male Wistar rats exposed to 10 GHz RFR for 2 h a day for 45 days; SAR 0.014 W/kg	Increased micronucleus in blood cells and DNA strand breaks in spermatozoa.
Marinelli et al. (2004)	Acute T-lymphoblastoid leukemia cells exposed to 900 MHz RFR for 2-48 h, SAR 0.0035 W/kg	Increased DNA damage and activation of genes involved in pro-survival signaling.
Markova et al. (2005)	Human lymphocytes exposed to 905 and 915 MHz GSM signals for 1 h; SAR 0.037 W/kg	Affected chromatin conformation and 53BP1/gamma-H2AX foci
Markova et al. (2010)	Human diploid VH-10 fibroblasts and human adipose-tissue derived mesenchymal stem	Inhibited tumor suppressor TP53 binding protein 1 (53BP1) foci that are typically formed at the sites of DNA double strand break

	cells exposed to GSM (905 MHz or 915 MHz) or UMTS (1947.4 MHz, middle channel) RFR for 1, 2, or 3 hr; SAR 0.037-0.039 W/kg	location.
Megha et al. (2015a)	Fischer rats exposed to 900 and 1800 MHz RFR for 30 days (2 h/day, 5 days/week), SAR 0.00059 and 0.00058 W/kg	Reduced levels of neurotransmitters dopamine, norepinephrine, epinephrine, and serotonin, and downregulation of mRNA of tyrosine hydroxylase and tryptophan hydroxylase (synthesizing enzymes for the transmitters) in the hippocampus.
Megha et al. (2015b)	Fischer rats exposed to 900, 1800, and 2450 MHz RFR for 60 days (2 h/day, 5 days/week); SAR 0.00059, 0.00058, and 0.00066 W/kg	Increased DNA damage in the hippocampus
Nittby et al. (2008)	Fischer 344 rats exposed to 1800 MHz GSM RFR for 6 h; SAR whole body average 0.013 W/kg, head 0.03 W/kg	Expression in cortex and hippocampus of genes connected with membrane functions.
Odaci et al. (2016)	Pregnant Sprague - Dawley rats exposed to 900 MHz RFR 1 h each day during days 13 - 21 of pregnancy; whole body average SAR 0.024 W/kg	Testis and epididymis of offspring showed higher DNA oxidation.
Pandey et al. (2017)	Swiss albino mice exposed to 900-MHz RFR for 4 or 8 h per day for 35 days; SAR 0.0054-0.0516 W/kg	DNA strand breaks in germ cells.
Pesnya and Romanovsky (2013)	Onion (<i>Allium cepa</i>) exposed to GSM 900-MHz RFR from a cell phone for 1 h/day or 9 h/day for 3 days;	Increased the mitotic index, the frequency of mitotic and chromosome abnormalities, and the micronucleus frequency in an exposure-duration manner.

	incident power density 0.0005 mW/cm ²	
Phillips et al. (1998)	Human Molt-4 T-lymphoblastoid cells exposed to pulsed signals at cellular telephone frequencies of 813.5625 MHz (iDEN signal) and 836.55 MHz (TDMA signal) for 2 or 21 h. SAR 0.0024 and 0.024 W/kg for iDEN and 0.0026 and 0.026 W/kg for TDMA)	Changes in DNA strand breaks
Qin et al. (2018)	Male mice exposed to 1800-MHz RFR 2 h/day for 32 days, SAR 0.0553 W/kg	Might be mediated through CaMKI/ROR α signaling pathway.
Rammal et al. (2014)	Tomato exposed to a 1250-MHz RFR for 10 days at 0.0095 mW/cm ²	Increased expression of two wound-plant genes.
Roux et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 2-10 min at 0.0066 mW/cm ²	Induction of stress gene expression.
Roux et al. (2008)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm ²	Induction of stress gene expression.
Sarimov et al. (2004)	Human lymphocytes exposed to GSM 895-915 MHz signals for 30 min; SAR 0.0054 W/kg	Condensation of chromatin was observed.
Shahin et al. (2013)	Female mice (<i>Mus musculus</i>) exposed to continuous-wave 2.45 GHz RFR 2 h/day for 45 days; SAR 0.023 W/kg	Increased DNA strand breaks in the brain.
Sokolovic et al. (2015)	Wistar rats exposed to RFR (4 h/day, for 20, 40, and 60 days) from a Nokia 3110 cell	DNA fragmentation and oxidative changes in testicular tissues.

	phone; SAR 0.043-0.135 W/kg.	
Sun Y. et al. (2017)	Human HL-60 cells exposed to 900 Hz RFR 5 h/day for 5 days; peak and average SAR 4.1×10^{-4} and 2.5×10^{-4} W/kg	Increased oxidative DNA damage and decreased mitochondrial gene expression.
Tkalec et al. (2013)	Earthworm (<i>Eisenia fetida</i>) exposed to continuous-wave and AM-modulated 900-MHz RFR for 2 - 4 h; SAR 0.00013, 0.00035, 0.0011, and 0.00933 W/kg	Increased DNA strand breaks.
Tsybulin et al. (2013)	Japanese Quail embryos exposed in ovo to GSM 900 MHz signal from a cell phone intermittently (48 sec ON/12 sec OFF) during initial 38 h of brooding or for 158 h (120 h before brooding plus initial 38 h of brooding): SAR 0.000003 W/kg	The lower duration of exposure decreased DNA strand breaks, whereas higher duration resulted in a significant increase in DNA damage.
Vian et al. (2006)	Tomato plants exposed to a 900-MHz RFR for 10 min at 0.0066 mW/cm^2	Induction of mRNA encoding the stress-related bZIP transcription factor.
Yakymenko et al. (2018)	Quail embryos exposed to GSM 1800 GHz signal from a smart phone (48 s ON/12 s OFF) for 5 days before and 14 days during incubation, power density 0.00032 mW/cm^2	Increased DNA strand breaks and oxidative DNA damage.
Zong et al. (2015)	Mice exposed to 900 MHz RFR 4 h/day for 7 days; SAR 0.05 W/kg	Attenuated bleomycin-induced DNA breaks and repair.

<u>Static and ELF EMF Studies</u>		
Agliassa et al. (2018)	Arabidopsis thaliana (thale cress) exposed to 0.00004 mT static magnetic field for 38 days after sowing	Changes in gene expression in leaf and floral meristem.
Back et al. (2019)	Mouse embryonic stem cells exposed to hypomagnetic field (<0.005 mT) up to 12 days	Induced abnormal DNA methylation.
Bagheri Hosseinabadi et al. (2020)	Blood samples from thermal power plant workers; mean levels of exposure to ELF magnetic and electric fields were 0.0165 mT (± 6.46) and 22.5 V/m (± 5.38), respectively.	DNA strand breaks .in lymphocytes.
Baraúna et al. (2015)	Chromobacterium violaceum bacteria cultures exposed to ELF-EMF for 7 h at 0.00066 mT	Five differentially expressed proteins detected including the DNA-binding stress protein.
Belyaev et al. (2005)	Human lymphocytes exposed to 50 Hz magnetic field at 0.015 mT (peak) for 2 h (measurements made at 24 and 48 h after exposure).	Induced chromatin conformation changes.
Dominici et al. (2011)	Lymphocytes from welders (average magnetic field exposure from personal dosimeters 0.00781 mT (general environmental level 0.00003 mT)	Higher micronucleus frequency correlated with EMF exposure levels; decreased in sister chromatid exchange frequency.
Heredia-Rojas et al. (2010)	Human non-small cell lung cancer cells (INER-37) and mouse lymphoma cells (RMA E7) (transfected with a	An increased in luciferase gene expression was observed in INER-37 cells.

	plasmid with hsp70 expression when exposed to magnetic field and contains the reporter for the luciferases gene) exposed to a 60-Hz magnetic field at 0.008 and 0.00008 mT for 20 min.	
Sarimov et al. (2011)	Human lymphocytes exposed to 50-Hz magnetic field at 0.005-0.02 mT for 15-180 min	Magnetic field condensed relaxed chromatin and relaxed condensed chromatin.
Villarini et al. (2015)	Blood leukocytes from electric arc welders presumably exposed to 50-Hz EMF (mean 0.0078 mT; range: 0.00003-0.171 mT)	Decreased DNA strand breaks.
Wahab et al. (2007)	Human peripheral blood lymphocytes exposed to 50 Hz sinusoidal (continuous or pulsed) or square (continuous or pulsed) magnetic fields at 0.001 or 1 mT for 72 h.	Increase in the number of sister chromatid exchange/cell
Zendehdel et al. (2019)	Peripheral blood cells of male power line workers in a power plant. The median value of the magnetic field at the working sites was 0.00085 mT.	Increased in DNA strand breaks.

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Effects of EMF wave-form and cell types (*in italic*) studied (Literature up to January 2021)

RFR	
Belyaev et al. (2009)	UMTS different from GSM signal on DNA repair foci in human lymphocytes.
Campisi et al. (2010)	Increased DNA fragmentation in rat neocortical astroglial by 50-Hz modulated 900-MHz RFR, but no effect from continuous wave field.
D'Ambrosia et al. (1995)	Micronucleus frequency in human lymphocytes affected by pulsed but not CW 9 GHz RFR.
D'Ambrosia et al. (2002)	Micronucleus frequency in human lymphocytes affected by pulsed but not CW 1748-MHz RFR.
<i>Del Re et al. (2019)</i>	<i>Changes in repetitive-DNA in human cell exposed to GSM 900-MHz RFR depended on cell type studied (HeLa, BE(2)C, and SH SY5Y).</i>
Franzellitti et al. (2008)	HSP70C gene expression enhanced after 24 h exposure to GSM-217Hz signals and reduced after 4 and 16 h exposure to GSM-Talk signals In human trophoblasts.
Franzellitti et al. (2010)	DNA damage in human trophoblasts induced by GSM 1800 MHz RFR, but not by continuous-wave field.
Gapeyev et al. (2014)	Protective effect to x-ray induced DNA strand break in mouse lymphocytes with pulse-modulated and not continuous-wave RFR.
<i>Heredia-Rojas et al. (2010)</i>	<i>"Electromagnetic field" plasmid transfected into INER-37 and RMA E7 cell lines exposed to a 60-Hz MF. An increased luciferase gene expression was observed in INER-37 cells but had no effect on the RMA E7 cell line.</i>
Kumar et al. (2020)	1800-MHz more effective than 900-MHz RFR on inducing DNA damage in onion.
Lopaz-Martin et al. (2009)	Unmodulated RFR caused higher neuronal c-fos expression than pulsed modulated 900-MHz GSM field.
Luukkonen et al. (2009)	872-MHz continuous-wave RFR increased DNA strand breaks in SH-SY5Y human neuroblastoma cells, but no effect from GSM – modulated field.
Markova et al. (2005)	GSM-915 MHz RFR induced more consistent

	effect on human lymphocyte chromatin conformation than GSM-905 MHz RFR.
<i>Martin et al. (2020)</i>	<i>Four different types of human keratinocytes showed different patterns (Up- and down-regulation or no change) of expression of ADAMTS6, IL7R, and NOG genes exposed to a 60-GHz RFR.</i>
Ozgur et al. (2014)	1800-MHz RFR more potent than 900-MHz RFR on inducing DNA fragmentation (apoptosis) in hepatocarcinoma cells.
<i>Nylund and Leszczynski (2006)</i>	<i>Gene and protein expressions in response to GSM 900-MHz RFR depended on the type of human endothelial cell line (EA.hy926 and EA.hy926v1).</i>
<i>Remondini et al. (2006)</i>	<i>Gene expressions after exposure to 900 and 1800 –MHz RFR- NB69 neuroblastoma cells, T lymphocytes, and CHME5 microglial cells did not show significant changes, whereas EA.hy926 endothelial cells, U937 lymphoblastoma cells, and HL-60 leukemia cells showed up- or down-regulated genes.</i>
Romano-Spica et al. (2000)	Oncogene expression only occurred when exposed to 16-Hz modulated 50MHz RFR
Sarimov et al. (2004)	Different potencies between 915 MHz and 905-MHz RFR on chromatin conformation in human lymphocytes.
<i>Schwartz et al. (2008)</i>	<i>UMTS 1950-MHz RFR increased DNA breaks and micronucleus frequency in human fibroblasts, but not in lymphocytes.</i>
Semin et al. (1994)	4000-8000 MHz RFR, 1-6 Hz modulated RFR showed narrow “window” peak intensity and modulation frequency effects on DNA secondary structure.
Shckorbatov et al. (2009)	35-GHz RFR caused condensation of chromatin in human buccal epithelium cells- left circularly polarized radiation induced less effect than linearly polarized radiation.
Shckorbatov et al. (2010)	36.65-GHz RFR caused chromosome condensation in human fibroblasts –right-handed elliptically polarized radiation was more biological activity than the left-handed polarized one.
Tkalec et al. (2013)	AM-modulated 900- MHz RFR more potent than continuous-wave field in inducing DNA damage in earthworms coelomocytes.
Valbonesi et al. (2014)	GSM 1800-MHz signal, but not continuous-

	wave field, induced HSP-70 gene expression in rat PC-12 cells.
Vilic et al. (2017)	DNA damage in honey bee larvae- AM-modulated 900-MHz RFR more potent than continuous-wave field.
<i>Xu et al. (2013)</i>	<i>Gamma-H2AX foci after exposure to GSM 1800-MHz RFR induced in Chinese hamster lung cells and Human skin fibroblasts (HSFs), but not in rat astrocytes, human amniotic epithelial cells, human lens epithelial cells, and human umbilical vein endothelial cells.</i>
Zhang et al. (2008)	Intermittent 1800-MHz RFR more potent than continuous exposure on gene expression in rat neurons.
<i>Zhao et al. (2007)</i>	<i>Capase-2 and Capase-6 expressions up-regulated in neuron, but not in astrocytes.</i>
Static/ELF EMF	
Del Re et al. (2006)	50-Hz sinusoidal MF increased whereas pulse square wave decreased heat-shock protein induction in E. coli.
Focke et al (2010)	Increased DNA fragmentation by intermittent 50-Hz MF, but no effect by continuous exposure.
Giorgi et al. (2011)	E. coli gene expression decreased by sinusoidal MF and increased by pulsed square-wave MF- not frequency dependent (25, 50, 75 Hz)
<i>Heredia-Rojas et al. (2010)</i>	<i>60-Hz MF induced luciferase gene expression in INER-37 cells, but not in RMA E7 cells.</i>
Ivancsits et al. (2002)	Intermittent more potent than continuous exposure of a 50-Hz MF on DNA damage in human fibroblasts.
<i>Lee et al. (2016)</i>	<i>60-Hz MF induced delay of cell cycle progression in MCF7 and MCF10A cells, but not in Jurkat and NIH3T3 cells.</i>
<i>Mahmoudinasab and Saadat (2018a)</i>	<i>Patterns of up-regulation of antioxidant genes are different between MCF-7 and SH-SY5Y cells exposed to an intermittent 50-Hz EMF.</i>
Mahmondinasab et al. (2016)	Different schedules of intermittent exposure to a 50-Hz MF had different effect on gene expression in human MCF-7 breast cancer cells
Mercado-Saenz et al. (2019)	Decreased spontaneous mitochondrial mutation in yeast by pulsed MF (25-Hz), no effect by sinusoidal field.

<i>Robison et al. (2002)</i>	<i>60-Hz MF exposure decreased DNA repair rate in HL-60 and HL-60R cells, but not in Raji cells.</i>
<i>Sanie-Jahromi and Saadat (2018)</i>	<i>co-treatment of “cisplatin +morphine + EMF” made bleomycin more cytotoxic in SH-SY5Y cells, but not in MCF-7cells.</i>
<i>Sanie-Jahromi et al. (2016)</i>	<i>Significant differences in DNA-repair gene expression in MCF-7 cell exposed under 3 different patterns of 50-Hz EMF (5 min field-on/5 min field-off (30 min), 15 min field-on/15 min field-off (30 min), 30 min field-on continuously.)</i>
<i>Sanie-Jahromi et al. (2017)</i>	<i>50-Hz MF exposure synergistic with cisplatin and bleomycin on DNA-repair gene expression and cell viability in MCF-7 cells, but not in SH-SY5Y cells.</i>
<i>Udroiu et al. (2015)</i>	<i>50-Hz MF exposure affected genotoxic effect of x-ray in mouse male germ cells, but not in peripheral blood erythrocytes.</i>
<i>Wahab et al. (2007)</i>	<i>Sister chromatid exchange in human lymphocytes exposed to a 50-Hz MF (continuous or pulsed sinusoidal or continuous or pulsed square-wave). Square continuous-wave MF was the most potent.</i>



SECTION 6

Genetic Effects of Non-Ionizing Electromagnetic Fields

2014 Supplement

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I. INTRODUCTION

The following is an update of information and abstracts on research papers published since 2006/2007 on the genetic effects of nonionizing electromagnetic fields (EMF) in the radiofrequency (RF) and extremely-low frequency (ELF) ranges. Two static magnetic field papers (Jouni et al. 2012; Wang et al., 2009) are also included. Where additional information is relevant, some earlier papers, or papers not specifically related to genetic effects, are also included with citations contained within the discussion below. A list of abstracts, with summary sentences underlined for reader convenience, can be found at the end of this paper.

Analysis of these recent publications shows that there are more papers reporting effects than no effect.

In summary, the new radiofrequency studies report that 65% of genetic studies show effects and 35% do not show effects. **[Effects = 74 (65%) No Effects = 40 (35%)]**

In summary, the new ELF-EMF studies report that 82% of genetic studies show effects and 18% do not show effects **[Effects= 49 (83%) No Effects= 10 (17%)]**

Appendix A has references and abstracts for the RFR literature. Appendix B has references and abstracts for the ELF-EMF literature.

II. GENOTOXIC EFFECTS OF RADIOFREQUENCY RADIATION (RFR) AND OF EXTREMELY LOW FREQUENCY ELECTROMAGNETIC FIELDS (ELF-EMF) (2007-2014)

The following is an update of information and abstracts on research papers published since 2006/2007 on the genetic effects of nonionizing electromagnetic fields (EMF) in the radiofrequency (RF) and extremely-low frequency (ELF) ranges. Two static magnetic field papers (Jouni et al. 2012; Wang et al., 2009) are also included. Where additional information is relevant, some earlier papers, or papers not specifically related to genetic effects, are also included with citations contained within the discussion below. A list of abstracts, with summary sentences underlined for reader convenience, can be found at the end of this paper.

Analysis of these recent publications shows that there are more papers reporting effects than no effect. With E representing a biological effect, and NE representing no biological effects, the recent literature finds RFR-genetic effects at: E=74 publications (65%); NE=40 publications (35%); and ELF-genetic effects at: E=49 (83%); NE=10 (17%).

Discussion

1. The effects of both RF and ELF fields are very similar. This is surprising because the energies carried by these EMFs are billions of folds different. An explanation for similar genetic effects has been provided by a recent paper by Blank and Goodman ([Blank M, Goodman R](#). DNA is a fractal antenna in electromagnetic fields. [Int. J. Radiat. Biol.](#) 87(4):409-415, 2011) in which they stated that ‘...the wide frequency range of interaction with EMF is the functional characteristic of a fractal antenna, and DNA appears to possess the two structural characteristics of fractal antennas, electronic conduction and self symmetry.’ However, similarities in effects between ELF and RF fields have also been reported in studies of other physiological processes, e.g., neurochemical and behavioral effects (Cf. Lai, H., Carino, M.A., Horita, A. and Guy, A.W. Opioid receptor subtypes that mediate a microwave-induced decrease in central cholinergic activity in the rat. *Bioelectromagnetics* 13:237-246, 1992; Lai, H. and Carino, M.A. Intracerebroventricular injections of mu and delta-opiate receptor antagonists block 60-Hz magnetic field-induced decreases in cholinergic activity in the frontal cortex and hippocampus of the rat. *Bioelectromagnetics* 19:433-437, 1998; Lai, H., Carino, M.A. and Ushijima, I. Acute exposure to a 60 Hz magnetic field affects rats' performance in the water maze. *Bioelectromagnetics* 19:117-122, 1998; Wang, B.M. and Lai, H. Acute exposure to pulsed 2450-MHz microwaves affects water maze learning in the rat. *Bioelectromagnetics* 21:52-56, 2000.) Thus, there is a basic interaction mechanism of biological tissues with electromagnetic fields that is independent of frequency. Many studies have implicated the involvement of free radical processes in the genetic effects of EMF: ELF-EMF (Butdak et al., 2012; Jouni et al., 2012; Luukkonen et al., 2014; Tiwari et al., 2014); RFR (Agarwal et al., 2009; Atasoy et al., 2012; Burlaka et al., 2013; Campisi et al., 2010; De Iuliis et al., 2009; Esmekaya et al., 2011; Ferreira et al., 2006; Gajski and Garaj-Vrhovac, 2009; Garaj-Vrhovac et al., 2011; Guler et al., 2010, 2012; Kesari and Behari, 2009; Kesari et al., 2010; Khalil et al., 2012; Kumar et al., 2010; Liu et al., 2013a,b; Luukkonan et al., 2009; Tomruk et al., 2010; Tkalec et al., 2013; Wu et al., 2008; Xu et al., 2010; Yao et al., 2003). Increase in free radical activity and changes in enzymes involved in cellular oxidative processes are the most consistent effects observed in cells and animals after EMF exposure. However, they are reports indicating that EMF could induce genetic effects without the involvement of free radicals (ELF- Alcaraz et al., 2013; RFR- Ferreira et al., 2006; Furtado-Filho et al., 2013) and increase in free radical after EMF exposure did not lead to genetic effects (Frahm et al., 2006). There are at least a couple of hundred published papers on the effects of EMF exposure on cellular oxidative processes. Many biological effects of EMF can be explained by intracellular changes in oxidative status, including the genetic effects reported in this review.
2. An important observation of the studies is that EMF can interact with other entities and synergistically cause genetic effects. These entities include: ELF-EMF- cisplatin (Buldak et al., 2012; El-Bialy et al., 2013), bleomycin (Cho et al., 2007), gadolinium (Cho et al., 2014); hydrogen peroxide and methyl methane sulfonate (Koyama et al., 2008), menadione (Luukkonan et al., 2011, 2014; Markkanen et al., 2008), ionizing radiation (Mairs et al., 2007; Journi et al., 2012 Yoon et al., 2014); RFR- chemical

mutagens (Baohong et al., 2005), clastogens (Kim et al., 2008), x-rays (Manti et al., 2008), ultraviolet ray (Baohong et al., 2007), aphidicolin (Tiwari et al., 2008), picrotoxin (López-Martín et al., 2009), doxorubicin (Zhijian et al., 2010), and incoherent electromagnetic noise (Wu et al., 2008; Yao et al., 2008). Most of the compounds that interact with EMF are mutagens. This is important because in real life situations, a person is usually exposed to many different environmental factors simultaneously. Synergism of these factors with EMF should be considered more seriously.

3. Several long term/repeated exposure papers are included in this update: ELF-EMF (Borhani et al., 2011; Cuccurazzu et al., 2010; Erdal et al., 2007; Fedrowitz and Loscher, 2012; Mariucci et al., 2010; Panagopoulous et al., 2013; Udrouiu et al., 2006), and RFR (Asasoy et al., 2012; Atli Serkeroglu et al., 2013; Burlaka et al., 2013; Chavdoula et al., 2010; Deshmukh et al., 2013; Ferreira et al., 2006; Garaj-Vrhovac et al., 2011; Guler et al., 2010, 2012; Kesari and Behari, 2009; Kesari et al., 2010; Lakshmi et al., 2010; Paulraj and Behari, 2006; Tomruk et al., 2010; Yan et al., 2008). These data are important in the understanding of the biological effects of EMF exposure in real life situation, since human environmental EMF exposure is both chronic and intermittent. Within these long-term exposure studies, there are several that investigated the effect of EMF exposure on developing animals (ELF-EMF: Borhani et al., 2011; Cuccurazzu et al., 2010; Panagopoulous et al., 2013; Udrouiu et al., 2006, RFR: Burlaka et al., 2013; Ferreira et al., 2006; Guler et al., 2010, 2012; Serkeroglu et al., 2013; Tomruk et al., 2010; Zalata et al., In press). Data of effects of EMF exposure on growth and development of young animals are urgently needed. There are several studies indicating that RFR may affect reproduction, particularly with effects on sperm physiology and DNA (Agarwal et al., 2009; Atasoy et al., 2012; Avendano et al., 2012; Chavdoula et al., 2010; de Iuliis et al., 2009; Liu et al., 2013b; Panagopoulous et al., 2007). Similar effects of ELF-EMF on sperm have also been reported, e.g., Hong R, Zhang Y, Liu Y, Weng EQ. Effects of extremely low frequency electromagnetic fields on DNA of testicular cells and sperm chromatin structure in mice. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi.* 23(6):414-417, 2005; Iorio R, Scrimaglio R, Rantucci E, Delle Monache S, Di Gaetano A, Finetti N, Francavilla F, Santucci R, Tettamanti E, Colonna R. A preliminary study of oscillating electromagnetic field effects on human spermatozoon motility. *Bioelectromagnetics.* 28(1):72-75, 2007; Iorio R, Delle Monache S, Bennato F, Di Bartolomeo C, Scrimaglio R, Cinque B, Colonna RC. Involvement of mitochondrial activity in mediating ELF-EMF stimulatory effect on human sperm motility. *Bioelectromagnetics.* 32(1):15-27, 2011.
4. Another area that needs more research is the biological effects of low-intensity exposure. This is particularly true for ELF-EMF, since intensities of ELF-EMF in the environment are in microtesla (μ T) levels. There are many studies on biological effects of low-intensity RFR (see Table 1 in Levitt, B.B. and Lai, H. Biological effects from exposure to electromagnetic radiation emitted by cell tower base stations and other antenna arrays. *Environ. Rev.* 18:369-395, 2010.) However, most cell and animal studies in ELF-EMF used fields in the millitesla (mT) level. Exceptions are the study of Sarimov et al. (2011) listed below in the reference section and the study of de Bruyn and de Jager (2010) ([de Bruyn L](#) and [de Jager L](#). Effect of long-term exposure to a randomly varied 50

Hz power frequency magnetic field on the fertility of the mouse. [Electromag. Biol. Med.](#) 29(1-2):52-61, 2010).

5. Two other important findings of these recent studies are that the effects of EMF are shown to be waveform specific and cell-type specific. Regarding waveform specificity, Campisi et al. (2010) reported increases in free radical activity and DNA fragmentation in brain cells after acute exposure to a 50-Hz amplitude-modulated 900-MHz RFR, whereas a continuous-wave 9000-MHz field produced no effect. Franzellitti et al. (2010) showed increased DNA strand breaks in trophoblasts after exposure to a 217-Hz modulated 1.8 GHz-RFR, but a continuous-wave field of the same carrier frequency was without effect. Tkalec et al (2013) reported that AM-modulated (1 KHz sinusoidal) 900-MHz RFR is more potent than non-modulated field in causing DNA damage in coelomocytes of exposed earthworms. Luukkonen et al. (2009) reported a continuous-wave 872-MHz RFR increased chemically-induced DNA strand breaks and free radicals in human neuroblastoma cells, whereas a GSM-modulated 872-MHz field had no significant effect. Zhang et al. (2008) found that gene expression in rat neurons is more sensitive to intermittent than continuous exposure to a 1.8 GHz-RFR. López-Martín et al. (2009) found that GSM and unmodulated RFR caused different effects on c-Fos gene expression in the rat brain. Regarding cell-type specificity, Nylund and Leszczynski (2006) and Remondini et al. (2006) reported different patterns of gene expression in different types of cells after exposure to RFR. Zhao et al. (2007) found that neurons are more sensitive to a 1.9 GHz cell phone radiation than astrocytes. Schwarz et al. (2008) reported DNA strand breaks and micronucleus formation in human fibroblasts, but not in lymphocytes, after exposure to a 1950-MHz UMTS field. Furthermore, Xu et al (2013) found DNA damages in some cell types and not in others after exposure to 1800-MHz RFR. Valbonesi et al. (2014) reported that HSP70 expression and MAPK signaling pathways in PC12 cells were affected by GSM-217 Hz signal and not by CW or GSM-talk signals. In ELF-EM research, Giorgi et al. (2011) found that DNA transposition in *E. coli* was *decreased* after exposure to a sinusoidal magnetic field and *increased* after exposure to a pulsed magnetic field. Kim et al. (2012) described DNA strand breaks in human fibroblasts after exposure to ELF magnetic field. They found that the pattern of changes depended on the eddy current and Lorentz force in the field. Nahab et al. (2007) reported that a square-continuous ELF magnetic field was more effective than sinusoidal-continuous or pulsed field in inducing sister chromatid exchange in human lymphocytes. These findings underscore the complicity of interaction of EMF with biological tissues and may partially explain why effects were observed in some studies and not others. It is essential to understand why and how certain wave-characteristics of an EMF are more effective than other characteristics in causing biological effects, and why certain types of cells are more susceptible to the effect of EMF? That there are different biological effects elicited by different EMF wave characteristics is critical proof for the existence of nonthermal effects.
6. Many biological/health effects have been reported in cells and animals after exposure to EMFs in both the ELF and RF ranges. (Sixty-five percent of the RFR papers and 82% of the ELF-EMF papers in the publication list below reported effects.) It is highly dishonest for a scientist to summarily deny the existence of biological effects of EMF. A

biological effect of EMF can be detrimental to health, but can also be turned into a beneficial means for the treatment of human diseases. Denying any effects hampers the development of electromagnetic treatments for diseases. Examples of possible clinical uses of EMF are: Alzheimer's disease ([Arendash GW](#), [Sanchez-Ramos J](#), [Mori T](#), [Mamcarz M](#), [Lin X](#), [Runfeldt M](#), [Wang L](#), [Zhang G](#), [Sava V](#), [Tan J](#), [Cao C](#)).

Electromagnetic field treatment protects against and reverses cognitive impairment in Alzheimer's disease mice. [J Alzheimers Dis](#). 19(1):191-210, 2010); Parkinson's disease (Wang Z, Che PL, Du J, Ha B, Yarema KJ. Static magnetic field exposure reproduces cellular effects of the Parkinson's disease drug candidate ZM241385. [PLoS One](#). 5(11):e13883, 2010); bone regeneration ([Lee HM](#), [Kwon UH](#), [Kim H](#), [Kim HJ](#), [Kim B](#), [Park JO](#), [Moon ES](#), [Moon SH](#)). Pulsed electromagnetic field stimulates cellular proliferation in human intervertebral disc cells. [Yonsei Med. J](#). 51(6):954-959, 2010); cancer treatment (Costa FP, de Oliveira AC, Meirelles R, Machado MC, Zanesco T, Surjan R, Chammas MC, de Souza Rocha M, Morgan D, Cantor A, Zimmerman J, Brezovich I, Kuster N, Barbault A, Pasche B. Treatment of advanced hepatocellular carcinoma with very low levels of amplitude-modulated electromagnetic fields. [Br. J. Cancer](#). 105(5):640-648, 2011), and tissue regeneration ([Gaetani R](#), [Ledda M](#), [Barile L](#), [Chimenti I](#), [De Carlo F](#), [Forte E](#), [Ionta V](#), [Giuliani L](#), [D'Emilia E](#), [Frati G](#), [Miraldi F](#), [Pozzi D](#), [Messina E](#), [Grimaldi S](#), [Giacomello A](#), [Lisi A](#)). Differentiation of human adult cardiac stem cells exposed to extremely low-frequency electromagnetic fields. [Cardiovasc. Res](#). 82(3):411-420, 2009).

7. It must be pointed out that, consistent with previous research, not very much of the cellular and animal genetic research data directly indicate that EMF (both RF and ELF EMF) is a carcinogen. However, the data show that EMF can possibly alter genetic functions and thus it is advisable that one should limit one's exposure to EMF.

APPENDIX A - ABSTRACTS ON GENETIC EFFECTS OF RADIOFREQUENCY AND CELL PHONE RADIATION (2007-2014)

Below is a key to abbreviations used throughout the following list of abstracts for recent papers published since 2006 and serve as my comments to help the reader quickly identify the significance of each work. The summary sentences by each author are underlined. The list is divided into RF effects papers, and ELF effects papers.

(E- effect observed; NE- no effect observed) (LE- long term exposure; GT- genotoxic effect, e.g., DNA damage, micronucleus formation, chromosome alterations; GE- gene expression; HU- human study; OX- oxidative effects, i.e., involvement of free radicals and oxidative enzymes; IA- interaction with other factors to cause genetic effects; DE- effects on developing animals; RP- reproduction, e.g., sperm damage; EH- compared with electro-hypersensitive subjects; WS- waveform specific effect, e.g., modulation and frequency; CS- cell type specific effect).

(E) Agarwal A, Desai NR, Makker K, Varghese A, Mouradi R, Sabanegh E, Sharma R. Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study. Fertil Steril 92 1318-1325, 2009. (GT, RP, OX)

OBJECTIVE: To evaluate effects of cellular phone radiofrequency electromagnetic waves (RF-EMW) during talk mode on unprocessed (neat) ejaculated human semen. DESIGN: Prospective pilot study. SETTING: Center for reproductive medicine laboratory in tertiary hospital setting. SAMPLES: Neat semen samples from normal healthy donors (n = 23) and infertile patients (n = 9). INTERVENTION(S): After liquefaction, neat semen samples were divided into two aliquots. One aliquot (experimental) from each patient was exposed to cellular phone radiation (in talk mode) for 1 h, and the second aliquot (unexposed) served as the control sample under identical conditions. MAIN OUTCOME MEASURE(S): Evaluation of sperm parameters (motility, viability), reactive oxygen species (ROS), total antioxidant capacity (TAC) of semen, ROS-TAC score, and sperm DNA damage. 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 and DNA damage showed no significant differences from the unexposed group. CONCLUSION(S): Radiofrequency electromagnetic waves emitted from cell phones may lead to oxidative stress in human semen. We speculate that keeping the cell phone in a trouser pocket in talk mode may negatively affect spermatozoa and impair male fertility.

(E) Atasoy HI, Gunal MY, Atasoy P, Elgun S, Bugdayci G. Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices. J Pediatr Urol. 2012 Mar 30. [Epub ahead of print] (GT, OX, LE, RP)

OBJECTIVE: To investigate effects on rat testes of radiofrequency radiation emitted from indoor Wi-Fi Internet access devices using 802.11.g wireless standards. **METHODS:** Ten Wistar albino male rats were divided into experimental and control groups, with five rats per group. Standard wireless gateways communicating at 2.437 GHz were used as radiofrequency wave sources. The experimental group was exposed to radiofrequency energy for 24 h a day for 20 weeks. The rats were sacrificed at the end of the study. Intracardiac blood was sampled for serum 8-hydroxy-2'-deoxyguanosine levels. Testes were removed and examined histologically and immunohistochemically. Testis tissues were analyzed for malondialdehyde levels and prooxidant-antioxidant enzyme activities. **RESULTS:** We observed significant increases in serum 8-hydroxy-2'-deoxyguanosine levels and 8-hydroxyguanosine staining in the testes of the experimental group indicating DNA damage due to exposure ($p < 0.05$). We also found decreased levels of catalase and glutathione peroxidase activity in the experimental group, which may have been due to radiofrequency effects on enzyme activity ($p < 0.05$). **CONCLUSIONS:** These findings raise questions about the safety of radiofrequency exposure from Wi-Fi Internet access devices for growing organisms of reproductive age, with a potential effect on both fertility and the integrity of germ cells.

(E) Atlı Şekeroğlu Z, Akar A, Sekeroğlu V. Evaluation of the cytogenotoxic damage in immature and mature rats exposed to 900 MHz radio frequency electromagnetic fields. Int J Radiat Biol. 89(11):985-992, 2013. [Epub ahead of print] (GT, DE, LE)

Abstract Purpose: One of the most important issues regarding radio frequency electromagnetic fields (RF-EMF) is their effect on genetic material. Therefore, we investigated the cytogenotoxic effects of 900 MHz radio frequency electromagnetic fields (RF-EMF) and the effect of a recovery period after exposure to RF-EMF on bone marrow cells of immature and mature rats. **Materials and methods:** The immature and mature rats in treatment groups were exposed to RF-EMF for 2 h/day for 45 days. Average electrical field values for immature and mature rats were 28.1 ± 4.8 V/m and 20.0 ± 3.2 V/m, respectively. Whole-body specific absorption rate (SAR) values for immature and mature rats were in the range of 0.38-0.78 W/kg, and 0.31-0.52 W/kg during the 45 days, respectively. Two recovery groups were kept for 15 days after RF-EMF exposure. **Results:** Significant differences were observed in chromosome aberrations (CA), micronucleus (MN) frequency, mitotic index (MI) and ratio of polychromatic erythrocytes (PCE) in all treatment and recovery groups. The cytogenotoxic damage in immature rats was statistically higher than the mature rats. The recovery period did not reduce the damage to the same extent as the corresponding control groups. **Conclusions:** The exposure of RF-EMF leads to cytotoxic and genotoxic damage in immature and mature rats. More sensitive studies are required to elucidate the possible carcinogenic risk of EMF exposure in humans, especially children.

(E) Avendaño C, Mata A, Sanchez Sarmiento CA, Doncel GF. Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. FertilSteril 97:39-45, 2012. (GT, RP)

OBJECTIVE: To evaluate the effects of laptop computers connected to local area networks wirelessly (Wi-Fi) on human spermatozoa. **DESIGN:** Prospective in vitro study. **SETTING:** Center for reproductive medicine. **PATIENT(S):** Semen samples from 29 healthy donors. **INTERVENTION(S):** Motile sperm were selected by swim up. Each sperm suspension was divided into two aliquots. One sperm aliquot (experimental) from each patient was exposed to an

internet-connected laptop by Wi-Fi for 4 hours, whereas the second aliquot (unexposed) was used as control, incubated under identical conditions without being exposed to the laptop. MAIN OUTCOME MEASURE(S): Evaluation of sperm motility, viability, and DNA fragmentation. RESULT(S): Donor sperm samples, mostly normozoospermic, exposed ex vivo during 4 hours to a wireless internet-connected laptop showed a significant decrease in progressive sperm motility and an increase in sperm DNA fragmentation. Levels of dead sperm showed no significant differences between the two groups. CONCLUSION(S): To our knowledge, this is the first study to evaluate the direct impact of laptop use on human spermatozoa. Ex vivo exposure of human spermatozoa to a wireless internet-connected laptop decreased motility and induced DNA fragmentation by a nonthermal effect. We speculate that keeping a laptop connected wirelessly to the internet on the lap near the testes may result in decreased male fertility. Further in vitro and in vivo studies are needed to prove this contention.

(E) Baohong Wang, Jiliang H, Lifen J, Deqiang L, Wei Z, Jianlin L, Hongping D. 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. Mutat Res 578:149-57, 2005. (GT, IA)

The aim of this investigation was to study the synergistic DNA damage effects in human lymphocytes induced by 1.8GHz radiofrequency field radiation (RFR, SAR of 3W/kg) with four chemical mutagens, i.e. mitomycin C (MMC, DNA crosslinker), bleomycin (BLM, radiomimetic agent), methyl methanesulfonate (MMS, alkylating agent), and 4-nitroquinoline-1-oxide (4NQO, UV-mimetic agent). The DNA damage of lymphocytes exposed to RFR and/or with chemical mutagens was detected at two incubation time (0 or 21h) after treatment with comet assay in vitro. Three combinative exposure ways were used. Cells were exposed to RFR and chemical mutagens for 2 and 3h, respectively. Tail length (TL) and tail moment (TM) were utilized as DNA damage indexes. The results showed no difference of DNA damage indexes between RFR group and control group at 0 and 21h incubation after exposure (P>0.05). There were significant difference of DNA damage indexes between MMC group and RFR+MMC co-exposure group at 0 and 21h incubation after treatment (P<0.01). Also the significant difference of DNA damage indexes between 4NQO group and RFR+4NQO co-exposure group at 0 and 21h incubation after treatment was observed (P<0.05 or P<0.01). The DNA damage in RFR+BLM co-exposure groups and RFR+MMS co-exposure groups was not significantly increased, as compared with corresponding BLM and MMS groups (P>0.05). The experimental results indicated 1.8GHz RFR (SAR, 3W/kg) for 2h did not induce the human lymphocyte DNA damage effects in vitro, but could enhance the human lymphocyte DNA damage effects induced by MMC and 4NQO. The synergistic DNA damage effects of 1.8GHz RFR with BLM or MMS were not obvious.

(E) Baohong W, Lifen J, Lanjuan L, Jianlin L, Deqiang L, Wei Z, Jiliang H. Evaluating the combinative effects on human lymphocyte DNA damage induced by ultraviolet ray C plus 1.8GHz microwaves using comet assay in vitro. Toxicology. 232(3):311-316, 2007. (GT, IA)

The objective of this study was to observe whether 1.8GHz microwaves (MW) (SAR, 3 W/kg) exposure can influence human lymphocyte DNA damage induced by ultraviolet ray C (UVC). The lymphocytes, which were from three young healthy donors, were exposed to 254 nm UVC at the doses of 0.25, 0.5, 0.75, 1.0, 1.5 and 2.0 J m⁻², respectively. The lymphocytes were irradiated by 1.8GHz MW (SAR, 3 W/kg) for 0, 1.5 and 4 h. The combinative exposure of UVC

plus MW was conducted. The treated cells were incubated for 0, 1.5 and 4 h. Finally, comet assay was used to measure DNA damage of above treated lymphocytes. The results indicated that the difference of DNA damage induced between MW group and control group was not significant ($P>0.05$). The MTLs induced by UVC were 1.71 ± 0.09 , 2.02 ± 0.08 , 2.27 ± 0.17 , 2.27 ± 0.06 , 2.25 ± 0.12 , 2.24 ± 0.11 microm, respectively, which were significantly higher than that (0.96 ± 0.05 microm) of control ($P<0.01$). MTLs of some sub-groups in combinative exposure groups at 1.5-h incubation were significantly lower than those of corresponding UVC sub-groups ($P<0.01$ or $P<0.05$). However, MTLs of some sub-groups in combinative exposure groups at 4-h incubation were significantly higher than those of corresponding UVC sub-groups ($P<0.01$ or $P<0.05$). In this experiment it was found that 1.8GHz (SAR, 3 W/kg) MW exposure for 1.5 and 4 h did not enhance significantly human lymphocyte DNA damage, but could reduce and increase DNA damage of human lymphocytes induced by UVC at 1.5-h and 4-h incubation, respectively.

(E) Belyaev IY, Hillert L, Protopopova M, Tamm C, Malmgren LO, Persson BR, Selivanova G, Harms-Ringdahl M. 915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons. Bioelectromagnetics 26:173-184, 2005. (GT, EH)

We used exposure to microwaves from a global system for mobile communication (GSM) mobile phone (915 MHz, specific absorption rate (SAR) 37 mW/kg) and power frequency magnetic field (50 Hz, 15 μ T peak value) to investigate the response of lymphocytes from healthy subjects and from persons reporting hypersensitivity to electromagnetic field (EMF). The hypersensitive and healthy donors were matched by gender and age and the data were analyzed blind to treatment condition. The changes in chromatin conformation were measured with the method of anomalous viscosity time dependencies (AVTD). 53BP1 protein, which has been shown to colocalize in foci with DNA double strand breaks (DSBs), was analyzed by immunostaining in situ. Exposure at room temperature to either 915 MHz or 50 Hz resulted in significant condensation of chromatin, shown as AVTD changes, which was similar to the effect of heat shock at 41 degrees C. No significant differences in responses between normal and hypersensitive subjects were detected. Neither 915 MHz nor 50 Hz exposure induced 53BP1 foci. On the contrary, a distinct decrease in background level of 53BP1 signaling was observed upon these exposures as well as after heat shock treatments. This decrease correlated with the AVTD data and may indicate decrease in accessibility of 53BP1 to antibodies because of stress-induced chromatin condensation. Apoptosis was determined by morphological changes and by apoptotic fragmentation of DNA as analyzed by pulsed-field gel electrophoresis (PFGE). No apoptosis was induced by exposure to 50 Hz and 915 MHz microwaves. In conclusion, 50 Hz magnetic field and 915 MHz microwaves under specified conditions of exposure induced comparable responses in lymphocytes from healthy and hypersensitive donors that were similar but not identical to stress response induced by heat shock.

(E) Belyaev IY, Koch CB, Terenius O, Roxstrom-Lindquist K, Malmgren LO, H Sommer W, Salford LG, Persson BR. 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. Bioelectromagnetics 27:295-306, 2006. (GE)

We investigated whether exposure of rat brain to microwaves (MWs) of global system for mobile communication (GSM) induces DNA breaks, changes in chromatin conformation and in gene expression. An exposure installation was used based on a test mobile phone employing a GSM signal at 915 MHz, all standard modulations included, output power level in pulses 2 W, specific absorption rate (SAR) 0.4 mW/g. Rats were exposed or sham exposed to MWs during 2 h. After exposure, cell suspensions were prepared from brain samples, as well as from spleen and thymus. For analysis of gene expression patterns, total RNA was extracted from cerebellum. Changes in chromatin conformation, which are indicative of stress response and genotoxic effects, were measured by the method of anomalous viscosity time dependencies (AVTD). DNA double strand breaks (DSBs) were analyzed by pulsed-field gel electrophoresis (PFGE). Effects of MW exposure were observed on neither conformation of chromatin nor DNA DSBs. Gene expression profiles were obtained by Affymetrix U34 GeneChips representing 8800 rat genes and analyzed with the Affymetrix Microarray Suite (MAS) 5.0 software. In cerebellum from all exposed animals, 11 genes were upregulated in a range of 1.34-2.74 fold and one gene was downregulated 0.48-fold ($P < .0025$). The induced genes encode proteins with diverse functions including neurotransmitter regulation, blood-brain barrier (BBB), and melatonin production. The data shows that GSM MWs at 915 MHz did not induce PFGE-detectable DNA double stranded breaks or changes in chromatin conformation, but affected expression of genes in rat brain cells

(E) Belyaev IY, Markovà E, Hillert L, Malmgren LO, Persson BR. Microwaves from UMTS/GSM mobile phones induce long-lasting inhibition of 53BP1/gamma-H2AX DNA repair foci in human lymphocytes. Bioelectromagnetics 30:129-41, 2009. (GT, EH)

We have recently described frequency-dependent effects of mobile phone microwaves (MWs) of global system for mobile communication (GSM) on human lymphocytes from persons reporting hypersensitivity to electromagnetic fields and healthy persons. Contrary to GSM, universal global telecommunications system (UMTS) mobile phones emit wide-band MW signals. Hypothetically, UMTS MWs may result in higher biological effects compared to GSM signal because of eventual "effective" frequencies within the wideband. Here, we report for the first time that UMTS MWs affect chromatin and inhibit formation of DNA double-strand breaks co-localizing 53BP1/gamma-H2AX DNA repair foci in human lymphocytes from hypersensitive and healthy persons and confirm that effects of GSM MWs depend on carrier frequency. Remarkably, the effects of MWs on 53BP1/gamma-H2AX foci persisted up to 72 h following exposure of cells, even longer than the stress response following heat shock. The data are in line with the hypothesis that the type of signal, UMTS MWs, may have higher biological efficiency and possibly larger health risk effects compared to GSM radiation emissions. No significant differences in effects between groups of healthy and hypersensitive subjects were observed, except for the effects of UMTS MWs and GSM-915 MHz MWs on the formation of the DNA repair foci, which were different for hypersensitive ($P < 0.02[53BP1]/0.01[\text{gamma-H2AX}]$) but not for control subjects ($P > 0.05$). The non-parametric statistics used here did not indicate specificity of the differences revealed between the effects of GSM and UMTS MWs on cells from hypersensitive subjects and more data are needed to study the nature of these differences.

(NE) Bourthoumieu S, Joubert V, Marin B, Collin A, Leveque P, Terro F, Yardin C. Cytogenetic studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using R-banded karyotyping. Radiat Res 174:712-718, 2010. (GT)

It is important to determine the possible effects of exposure to radiofrequency (RF) radiation on the genetic material of cells since damage to the DNA of somatic cells may be linked to cancer development or cell death and damage to germ cells may lead to genetic damage in next and subsequent generations. The objective of this study was to investigate whether exposure to radiofrequency radiation similar to that emitted by mobile phones of second-generation standard Global System for Mobile Communication (GSM) induces genotoxic effects in cultured human cells. The cytogenetic effects of GSM-900 MHz (GSM-900) RF radiation were investigated using R-banded karyotyping after in vitro exposure of human cells (amniotic cells) for 24 h. The average specific absorption rate (SAR) was 0.25 W/kg. The exposures were carried out in wire-patch cells (WPCs) under strictly controlled conditions of temperature. The genotoxic effect was assessed immediately or 24 h after exposure using four different samples. One hundred metaphase cells were analyzed per assay. Positive controls were provided by using bleomycin. We found no direct cytogenetic effects of GSM-900 either 0 h or 24 h after exposure. To the best of our knowledge, our work is the first to study genotoxicity using complete R-banded karyotyping, which allows visualizing all the chromosomal rearrangements, either numerical or structural.

(NE) Bourthoumieu S, Terro F, Leveque P, Collin A, Joubert V, Yardin C. Aneuploidy studies in human cells exposed in vitro to GSM-900 MHz radiofrequency radiation using FISH. Int J Radiat Biol 87:400-408, 2011. (GT)

PURPOSE: Since previous research found an increase in the rate of aneuploidies in human lymphocytes exposed to radiofrequencies, it seems important to perform further studies. The objective of this study was then to investigate whether the exposure to RF (radiofrequency) radiation similar to that emitted by mobile phones of a second generation standard, i.e., Global System for Mobile communication (GSM) may induce aneuploidy in cultured human cells. **MATERIALS AND METHODS:** The potential induction of genomic instability by GSM-900 MHz radiofrequency (GSM-900) was investigated after in vitro exposure of human amniotic cells for 24 h to average-specific absorption rates (SAR) of 0.25, 1, 2 and 4 W/kg in the temperature range of 36.3-39.7°C. The exposures were carried out in a wire-patch cell (WPC). The rate of aneuploidy of chromosomes 11 and 17 was determined by interphase FISH (Fluorescence In Situ Hybridisation) immediately after independent exposure of three different donors for 24 h. At least 100 interphase cells were analysed per assay. **RESULTS:** No significant change in the rate of aneuploidy of chromosomes 11 and 17 was found following exposure to GSM-900 for 24 h at average SAR up to 4 W/kg. **CONCLUSION:** Our study did not show any in vitro aneuploidogenic effect of GSM using FISH and is not in agreement with the results of previous research.

(NE) Bourthoumieu S, Magnaudeix A, Terro F, Leveque P, Collin A, Yardin C. Study of p53 expression and post-transcriptional modifications after GSM-900 radiofrequency exposure of human amniotic cells. Bioelectromagnetics. 2012 Jul 5. doi: 10.1002/bem.21744. [Epub ahead of print] (GE)

The potential effects of radiofrequency (RF) exposure on the genetic material of cells are very important to determine since genome instability of somatic cells may be linked to cancer development. In response to genetic damage, the p53 protein is activated and can induce cell cycle arrest allowing more time for DNA repair or elimination of damaged cells through

apoptosis. The objective of this study was to investigate whether the exposure to RF electromagnetic fields, similar to those emitted by mobile phones of the second generation standard, Global System for Mobile Communications (GSM), may induce expression of the p53 protein and its activation by post-translational modifications in cultured human cells. The potential induction of p53 expression and activation by GSM-900 was investigated after in vitro exposure of human amniotic cells for 24 h to average specific absorption rates (SARs) of 0.25, 1, 2, and 4 W/kg in the temperature range of 36.3-39.7 °C. The exposures were carried out using a wire-patch cell (WPC) under strictly controlled conditions of temperature. Expression and activation of p53 by phosphorylation at serine 15 and 37 were studied using Western blot assay immediately after three independent exposures of cell cultures provided from three different donors. Bleomycin-exposed cells were used as a positive control. According to our results, no significant changes in the expression and activation of the p53 protein by phosphorylation at serine 15 and 37 were found following exposure to GSM-900 for 24 h at average SARs up to 4 W/kg in human embryonic cells.

(E) Burlaka A, Tsybulin O, Sidorik E, Lukin S, Polishuk V, Tshmistrenko S, Yakymenko I. Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation. Exp Oncol. 35(3):219-225, 2013. (GT, LE, DE, OX)

Aim: Long-term exposure of humans to low intensity radiofrequency electromagnetic radiation (RF-EMR) leads to a statistically significant increase in tumor incidence. Mechanisms of such the effects are unclear, but features of oxidative stress in living cells under RF-EMR exposure were previously reported. Our study aims to assess a production of initial free radical species, which lead to oxidative stress in the cell. Materials and Methods: Embryos of Japanese quails were exposed in ovo to extremely low intensity RF-EMR of GSM 900 MHz (0.25 μ W/cm²) during 158-360 h discontinuously (48 c - ON, 12 c - OFF) before and in the initial stages of development. The levels of superoxide (O₂^{·-}), nitrogen oxide (NO[·]), thiobarbituric acid reactive substances (TBARS), 8-oxo-2'-deoxyguanosine (8-oxo-dG) and antioxidant enzymes' activities were assessed in cells/tissues of 38-h, 5- and 10-day RF-EMR exposed and unexposed embryos. Results: The exposure resulted in a significant persistent overproduction of superoxide and nitrogen oxide in embryo cells during all period of analyses. As a result, significantly increased levels of TBARS and 8-oxo-dG followed by significantly decreased levels of superoxide dismutase and catalase activities were developed in the exposed embryo cells. 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.

(E) Buttiglione M, Roca L, Montemurno E, Vitiello F, Capozzi V, Cibelli G. Radiofrequency radiation (900 MHz) induces Egr-1 gene expression and affects cell-cycle control in human neuroblastoma cells. J Cell Physiol. 213(3):759-767, 2007. (GE)

Many environmental signals, including ionizing radiation and UV rays, induce activation of Egr-1 gene, thus affecting cell growth and apoptosis. The paucity and the controversial knowledge about the effect of electromagnetic fields (EMF) exposure of nerve cells prompted us to investigate the bioeffects of radiofrequency (RF) radiation on SH-SY5Y neuroblastoma cells. The effect of a modulated RF field of 900 MHz, generated by a wire patch cell (WPC) antenna

exposure system on Egr-1 gene expression, was studied as a function of time. Short-term exposures induced a transient increase in Egr-1 mRNA level paralleled with activation of the MAPK subtypes ERK1/2 and SAPK/JNK. The effects of RF radiations on cell growth rate and apoptosis were also studied. Exposure to RF radiation had an anti-proliferative activity in SH-SY5Y cells with a significant effect observed at 24 h. RF radiation impaired cell cycle progression, reaching a significant G2-M arrest. In addition, the appearance of the sub-G1 peak, a hallmark of apoptosis, was highlighted after a 24-h exposure, together with a significant decrease in mRNA levels of Bcl-2 and survivin genes, both interfering with signaling between G2-M arrest and apoptosis. Our results provide evidence that exposure to a 900 MHz-modulated RF radiation affect both Egr-1 gene expression and cell regulatory functions, involving apoptosis inhibitors like Bcl-2 and survivin, thus providing important insights into a potentially broad mechanism for controlling in vitro cell viability.

(E) Cam ST, Seyhan N. Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation. Int J Radiat Biol 88(5):420-424, 2012 (GT, HU)

Purpose: To analyze the short term effects of radiofrequency radiation (RFR) exposure on genomic deoxyribonucleic acid (DNA) of human hair root cells. Subjects and methods: Hair samples were collected from 8 healthy human subjects immediately before and after using a 900-MHz GSM (Global System for Mobile Communications) mobile phone for 15 and 30 minutes. Single-strand DNA breaks of hair root cells from the samples were determined using the 'comet assay'. Results: The data showed that talking on a mobile phone for 15 or 30 minutes significantly increased ($p < .05$) single-strand DNA breaks in cells of hair roots close to the phone. Comparing the 15-min and 30-min data using the paired t-test also showed that significantly more damages resulted after 30 minutes than after 15 minutes of phone use. Conclusions: A short-term exposure (15 and 30 minutes) 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.

(E) Campisi A, Gulino M, Acquaviva R, Bellia P, Raciti G, Grasso R, Musumeci F, Vanella A, Triglia A. Reactive oxygen species levels and DNA fragmentation on astrocytes in primary culture after acute exposure to low intensity microwave electromagnetic field. Neurosci Lett 473:52-55. 2010. (GT, OX, WS)

The exposure of primary rat neocortical astroglial cell cultures to acute electromagnetic fields (EMF) in the microwave range was studied. Differentiated astroglial cell cultures at 14 days in vitro were exposed for 5, 10, or 20 min to either 900 MHz continuous waves or 900 MHz waves modulated in amplitude at 50 Hz using a sinusoidal waveform and 100% modulation index. The strength of the electric field (rms value) at the sample position was 10V/m. No change in cellular viability evaluated by MTT test and lactate dehydrogenase release was observed. A significant increase in ROS levels and DNA fragmentation was found only after exposure of the astrocytes to modulated EMF for 20 min. No evident effects were detected when shorter time intervals or continuous waves were used. The irradiation conditions allowed the exclusion of any possible thermal effect. 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.

(E) 1 Cervellati F, Valacchi G, Lunghi L, Fabbri E, Valbonesi P, Marci R, Biondi C, Vesce F. **17- β -estradiol counteracts the effects of high frequency electromagnetic fields on trophoblastic connexins and integrins.** *Oxid Med Cell Longev.* 2013;2013:280850. doi: 10.1155/2013/280850. **(GE)**

We investigated the effect of high-frequency electromagnetic fields (HF-EMFs) and 17- β -estradiol on connexins (Cxs), integrins (Ints), and estrogen receptor (ER) expression, as well as on ultrastructure of trophoblast-derived HTR-8/SVneo cells. HF-EMF, 17- β -estradiol, and their combination induced an increase of Cx40 and Cx43 mRNA expression. HF-EMF decreased Int alpha1 and β 1 mRNA levels but enhanced Int alpha5 mRNA expression. All the Ints mRNA expressions were increased by 17- β -estradiol and exposure to both stimuli. ER- β mRNA was reduced by HF-EMF but augmented by 17- β -estradiol alone or with HF-EMF. ER- β immunofluorescence showed a cytoplasmic localization in sham and HF-EMF exposed cells which became nuclear after treatment with hormone or both stimuli. Electron microscopy evidenced a loss of cellular contact in exposed cells which appeared counteracted by 17- β -estradiol. We demonstrate that 17- β -estradiol modulates Cxs and Ints as well as ER- β expression induced by HF-EMF, suggesting an influence of both stimuli on trophoblast differentiation and migration.

(NE) Chang SK, Choi JS, Gil HW, Yang JO, Lee EY, Jeon YS, Lee ZW, Lee M, Hong MY, Ho Son T, Hong SY. **Genotoxicity evaluation of electromagnetic fields generated by 835-MHz mobile phone frequency band.** *Eur J Cancer Prev* 14:175-179, 2005. **(GT, IA)**
(Some interaction effects with chemicals are reported in this paper.)

It is still unclear whether the exposure to electromagnetic fields (EMFs) generated by mobile phone radiation is directly linked to cancer. We examined the biological effects of an EMF at 835 MHz, the most widely used communication frequency band in Korean CDMA mobile phone networks, on bacterial reverse mutation (Ames assay) and DNA stability (in vitro DNA degradation). In the Ames assay, tester strains alone or combined with positive mutagen were applied in an artificial mobile phone frequency EMF generator with continuous waveform at a specific absorption rate (SAR) of 4 W/kg for 48 h. In the presence of the 835-MHz EMF radiation, incubation with positive mutagen 4-nitroquinoline-1-oxide and cumene hydroxide further increased the mutation rate in Escherichia coli WP2 and TA102, respectively, while the contrary results in Salmonella typhimurium TA98 and TA1535 treated with 4-nitroquinoline-1-oxide and sodium azide, respectively, were shown as antimutagenic. However, these mutagenic or co-mutagenic effects of 835-MHz radiation were not significantly repeated in other relevant strains with same mutation type. In the DNA degradation test, the exposure to 835-MHz EMF did not change the rate of degradation observed using plasmid pBluescriptSK(+) as an indicator. Thus, we suggest that 835-MHz EMF under the conditions of our study neither affected the reverse mutation frequency nor accelerated DNA degradation in vitro.

(NE) Chauhan V, Mariampillai A, Bellier PV, Qutob SS, Gajda GB, Lemay E, Thansandote A, McNamee JP. **Gene expression analysis of a human lymphoblastoma cell**

line exposed in vitro to an intermittent 1.9 GHz pulse-modulated radiofrequency field. [Radiat Res.](#) 165(4):424-429, 2006. (GE)

This study was designed to determine whether radiofrequency (RF) fields of the type used for wireless communications could elicit a cellular stress response. As general indicators of a cellular stress response, we monitored changes in proto-oncogene and heat-shock protein expression. Exponentially growing human lymphoblastoma cells (TK6) were exposed to 1.9 GHz pulse-modulated RF fields at average specific absorption rates (SARs) of 1 and 10 W/kg. Perturbations in the expression levels of the proto-oncogenes FOS, JUN and MYC after exposure to sham and RF fields were assessed by real-time RT-PCR. In addition, the transcript levels of the cellular stress proteins HSP27 and inducible HSP70 were also monitored. We demonstrated that transcript levels of these genes in RF-field-exposed cells showed no significant difference in relation to the sham treatment group. However, concurrent positive (heat-shock) control samples displayed a significant elevation in the expression of HSP27, HSP70, FOS and JUN. Conversely, the levels of MYC mRNA were found to decline in the positive (heat-shock) control. In conclusion, our study found no evidence that the 1.9 GHz RF-field exposure caused a general stress response in TK6 cells under our experimental conditions.

(NE) [Chauhan V](#), [Mariampillai A](#), [Gajda GB](#), [Thansandote A](#), [McNamee JP](#). 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. [Int J Radiat Biol.](#) 82(5):347-354, 2006. (GE)

Purpose: Several studies have reported that radiofrequency (RF) fields, as emitted by mobile phones, may cause changes in gene expression in cultured human cell-lines. The current study was undertaken to evaluate this possibility in two human-derived immune cell-lines. Materials and methods: HL-60 and Mono-Mac-6 (MM6) cells were individually exposed to intermittent (5 min on, 10 min off) 1.9 GHz pulse-modulated RF fields at a average specific absorption rate (SAR) of 1 and 10 W/kg at 37 +/- 0.5 degrees C for 6 h. Concurrent negative and positive (heat-shock for 1 h at 43 degrees C) controls were conducted with each experiment. Immediately following RF field exposure (T = 6 h) and 18 h post-exposure (T = 24 h), cell pellets were collected from each of the culture dishes and analyzed for transcript levels of proto-oncogenes (c-jun, c-myc and c-fos) and the stress-related genes (heat shock proteins (HSP) HSP27 and HSP70B) by quantitative reverse transcriptase polymerase chain reaction (RT-PCR). Results: No significant effects were observed in mRNA expression of HSP27, HSP70, c-jun, c-myc or c-fos between the sham and RF-exposed groups, in either of the two cell-lines. However, the positive (heat-shock) control group displayed a significant elevation in the expression of HSP27, HSP70, c-fos and c-jun in both cell-lines at T = 6 and 24 h, relative to the sham and negative control groups. Conclusion: This study found no evidence that exposure of cells to non-thermalizing levels of 1.9 GHz pulse-modulated RF fields can cause any detectable change in stress-related gene expression.

(NE) [Chauhan V](#), [Qutob SS](#), [Lui S](#), [Mariampillai A](#), [Bellier PV](#), [Yauk CL](#), [Douglas GR](#), [Williams A](#), [McNamee JP](#). Analysis of gene expression in two human-derived cell lines exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field. [Proteomics.](#) 7(21):3896-3905, 2007. (GE)

There is considerable controversy surrounding the biological effects of radiofrequency (RF) fields, as emitted by mobile phones. Previous work from our laboratory has shown no effect related to the exposure of 1.9 GHz pulse-modulated RF fields on the expression of 22,000 genes in a human glioblastoma-derived cell-line (U87MG) at 6 h following a 4 h RF field exposure period. As a follow-up to this study, we have now examined the effect of RF field exposure on the possible expression of late onset genes in U87MG cells after a 24 h RF exposure period. In addition, a human monocyte-derived cell-line (Mono-Mac-6, MM6) was exposed to intermittent (5 min ON, 10 min OFF) RF fields for 6 h and then gene expression was assessed immediately after exposure and at 18 h postexposure. Both cell lines were exposed to 1.9 GHz pulse-modulated RF fields for 6 or 24 h at specific absorption rates (SARs) of 0.1-10.0 W/kg. In support of our previous results, we found no evidence that nonthermal RF field exposure could alter gene expression in either cultured U87MG or MM6 cells, relative to nonirradiated control groups. However, exposure of both cell-lines to heat-shock conditions (43 degrees C for 1 h) caused an alteration in the expression of a number of well-characterized heat-shock proteins.

(E) Chavdoula ED, Panagopoulos DJ, Margaritis LH. Comparison of biological effects between continuous and intermittent exposure to GSM-900-MHz mobile phone radiation: detection of apoptotic cell-death features. *Mutat Res* 700:51-61, 2010. (RP, LE, GT)

In the present study we used a 6-min daily exposure of dipteran flies, *Drosophila melanogaster*, to GSM-900 MHz (Global System for Mobile Telecommunications) mobile phone electromagnetic radiation (EMR), to compare the effects between the continuous and four different intermittent exposures of 6min total duration, and also to test whether intermittent exposure provides any cumulative effects on the insect's reproductive capacity as well as on the induction of apoptotic cell death. According to our previous experiments, a 6-min continuous exposure per day for five days to GSM-900 MHz and DCS-1800 MHz (Digital Cellular System) mobile phone radiation, brought about a large decrease in the insect's reproductive capacity, as defined by the number of F pupae. This decrease was found to be non thermal and correlated with an increased percentage of induced fragmented DNA in the egg chambers' cells at early- and mid-oogenesis. In the present experiments we show that intermittent exposure also decreases the reproductive capacity and alters the actin cytoskeleton network of the egg chambers, another known aspect of cell death that was not investigated in previous experiments, and that the effect is also due to DNA fragmentation. Intermittent exposures with 10-min intervals between exposure sessions proved to be almost equally effective as continuous exposure of the same total duration, whereas longer intervals between the exposures seemed to allow the organism the time required to recover and partly overcome the above-mentioned effects of the GSM exposure.

(E) [Chen G](#), [Lu D](#), [Chiang H](#), [Leszczynski D](#), [Xu Z](#). Using model organism *Saccharomyces cerevisiae* to evaluate the effects of ELF-MF and RF-EMF exposure on global gene expression. *Bioelectromagnetics*. 33(7):550-560, 2012 . (GE)

The potential health hazard of exposure to electromagnetic fields (EMF) continues to cause public concern. However, the possibility of biological and health effects of exposure to EMF remains controversial and their biophysical mechanisms are unknown. In the present study, we used *Saccharomyces cerevisiae* to identify genes responding to extremely low frequency magnetic fields (ELF-MF) and to radiofrequency EMF (RF-EMF) exposures. The yeast cells were exposed for 6 h to either 0.4 mT 50 Hz ELF-MF or 1800 MHz RF-EMF at a specific

absorption rate of 4.7 W/kg. Gene expression was analyzed by microarray screening and confirmed using real-time reverse transcription-polymerase chain reaction (RT-PCR). We were unable to confirm microarray-detected changes in three of the ELF-MF responsive candidate genes using RT-PCR ($P > 0.05$). On the other hand, out of the 40 potential RF-EMF responsive genes, only the expressions of structural maintenance of chromosomes 3 (SMC3) and aquaporin 2 (AQY2 (m)) were confirmed, while three other genes, that is, halotolerance protein 9 (HAL9), yet another kinase 1 (YAK1) and one function-unknown gene (open reading frame: YJL171C), showed opposite changes in expression compared to the microarray data ($P < 0.05$). In conclusion, the results of this study suggest that the yeast cells did not alter gene expression in response to 50 Hz ELF-MF and that the response to RF-EMF is limited to only a very small number of genes. The possible biological consequences of the gene expression changes induced by RF-EMF await further investigation.

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(E) De Iuliis GN, Newey RJ, King BV, Aitken RJ. Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro. PLoS One 4:e6446, 2009. (GT, OX, RP)

BACKGROUND: In recent times there has been some controversy over the impact of electromagnetic radiation on human health. The significance of mobile phone radiation on male reproduction is a key element of this debate since several studies have suggested a relationship between mobile phone use and semen quality. The potential mechanisms involved have not been established, however, human spermatozoa are known to be particularly vulnerable to oxidative stress by virtue of the abundant availability of substrates for free radical attack and the lack of cytoplasmic space to accommodate antioxidant enzymes. Moreover, the induction of oxidative stress in these cells not only perturbs their capacity for fertilization but also contributes to sperm DNA damage. The latter has, in turn, been linked with poor fertility, an increased incidence of miscarriage and morbidity in the offspring, including childhood cancer. In light of these associations, we have analyzed the influence of RF-EMR on the cell biology of 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 DNA base 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.

(E) Del Vecchio G, Giuliani A, Fernandez M, Mesirca P, Bersani F, Pinto R, Ardoino L, Lovisolo GA, Giardino L, Calzà L. Continuous exposure to 900MHz GSM-modulated EMF alters morphological maturation of neural cells. Neurosci Lett. 455(3):173-177, 2009. (GE, DE)

The effects of radiofrequency electromagnetic field (RF-EMF) exposure on neuronal phenotype maturation have been studied in two different in vitro models: murine SN56 cholinergic cell line and rat primary cortical neurons. The samples were exposed at a dose of 1W/kg at 900 MHz GSM modulated. The phenotype analysis was carried out at 48 and 72 h (24 and 48 h of SN56 cell line differentiation) or at 24, 72, 120 h (2, 4 and 6 days in vitro for cortical neurons) of exposure, on live and immunolabeled neurons, and included the morphological study of neurite emission, outgrowth and branching. Moreover, cortical neurons were studied to detect alterations in the expression pattern of cytoskeleton regulating factors, e.g. beta-thymosin, and of early genes, e.g. c-Fos and c-Jun through real-time PCR on mRNA extracted after 24h exposure to EMF. We found that RF-EMF exposure reduced the number of neurites generated by both cell systems, and this alteration correlates to increased expression of beta-thymosin mRNA.

(E) Deshmukh PS, Megha K, Banerjee BD, Ahmed RS, Chandna S, Abegaonkar MP, Tripathi AK. Detection of Low Level Microwave Radiation Induced Deoxyribonucleic Acid Damage Vis-à-vis Genotoxicity in Brain of Fischer Rats. Toxicol Int. 20(1):19-24, 2013. (GT, LE)

BACKGROUND: Non-ionizing radiofrequency radiation has been increasingly used in industry, commerce, medicine and especially in mobile phone technology and has become a matter of serious concern in present time. OBJECTIVE: The present study was designed to investigate the possible deoxyribonucleic acid (DNA) damaging effects of low-level microwave radiation in brain of Fischer rats. MATERIALS AND METHODS: Experiments were performed on male Fischer rats exposed to microwave radiation for 30 days at three different frequencies: 900, 1800 and 2450 MHz. Animals were divided into 4 groups: Group I (Sham exposed): Animals not exposed to microwave radiation but kept under same conditions as that of other groups, Group II: Animals exposed to microwave radiation at frequency 900 MHz at specific absorption rate (SAR) $5.953 \times 10(-4)$ W/kg, Group III: Animals exposed to 1800 MHz at SAR $5.835 \times 10(-4)$ W/kg and Group IV: Animals exposed to 2450 MHz at SAR $6.672 \times 10(-4)$ W/kg. At the end of the exposure period animals were sacrificed immediately and DNA damage in brain tissue was assessed using alkaline comet assay. RESULTS: In the present study, we demonstrated DNA damaging effects of low level microwave radiation in brain. CONCLUSION: We concluded that low SAR microwave radiation exposure at these frequencies may induce DNA strand breaks in brain tissue.

(E) Engelmann JC, Deeken R, Müller T, Nimtz G, Roelfsema MR, Hedrich R. Is gene activity in plant cells affected by UMTS-irradiation? A whole genome approach. Adv Appl Bioinform Chem. 1:71-83, 2008. (GE)

Mobile phone technology makes use of radio frequency (RF) electromagnetic fields transmitted through a dense network of base stations in Europe. Possible harmful effects of RF fields on humans and animals are discussed, but their effect on plants has received little attention. In search for physiological processes of plant cells sensitive to RF fields, cell suspension cultures of *Arabidopsis thaliana* were exposed for 24 h to a RF field protocol representing typical microwave exposition in an urban environment. mRNA of exposed cultures and controls was used to hybridize Affymetrix-ATH1 whole genome microarrays. Differential expression analysis revealed significant changes in transcription of 10 genes, but they did not exceed a fold change

of 2.5. Besides that 3 of them are dark-inducible, their functions do not point to any known responses of plants to environmental stimuli. The changes in transcription of these genes were compared with published microarray datasets and revealed a weak similarity of the microwave to light treatment experiments. Considering the large changes described in published experiments, it is questionable if the small alterations caused by a 24 h continuous microwave exposure would have any impact on the growth and reproduction of whole plants.

(E) Esmekaya MA, Aytekin E, Ozgur E, Güler G, Ergun MA, Omeroğlu S, Seyhan N. 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). Sci Total Environ. 410-411:59-64, 2011. (GT, OX)

The mutagenic and morphologic effects of 1.8GHz Global System for Mobile Communications (GSM) modulated RF (radiofrequency) radiation alone and in combination with Ginkgo biloba (EGb 761) pre-treatment in human peripheral blood lymphocytes (hPBLs) were investigated in this study using Sister Chromatid Exchange (SCE) and electron microscopy. Cell viability was assessed with 3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide (MTT) reduction assay. The lymphocyte cultures were exposed to GSM modulated RF radiation at 1.8GHz for 6, 8, 24 and 48h with and without EGb 761. We observed morphological changes in pulse-modulated RF radiated lymphocytes. Longer exposure periods led to destruction of organelle and nucleus structures. Chromatin change and the loss of mitochondrial crista occurred in cells exposed to RF for 8h and 24h and were more pronounced in cells exposed for 48h. Cytoplasmic lysis and destruction of membrane integrity of cells and nuclei were also seen in 48h RF exposed cells. There was a significant increase ($p < 0.05$) in SCE frequency in RF exposed lymphocytes compared to sham controls. EGb 761 pre-treatment significantly decreased SCE from RF radiation. RF radiation also inhibited cell viability in a time dependent manner. The inhibitory effects of RF radiation on the growth of lymphocytes were marked in longer exposure periods. EGb 761 pre-treatment significantly increased cell viability in RF+EGb 761 treated groups at 8 and 24h when compared to RF exposed groups alone. The results of our study showed that RF radiation affects cell morphology, increases SCE and inhibits cell proliferation. However, EGb 761 has a protective role against RF induced mutagenity. We concluded that RF radiation induces chromosomal damage in hPBLs but this damage may be reduced by EGb 761 pre-treatment.

(NE) Falzone N, Huyser C, Franken DR, Leszczynski D. Mobile phone radiation does not induce pro-apoptosis effects in human spermatozoa. Radiat Res 174:169-176, 2010. (GT, OX)

Abstract Recent reports suggest that mobile phone radiation may diminish male fertility. However, the effects of this radiation on human spermatozoa are largely unknown. The present study examined effects of the radiation on induction of apoptosis-related properties in human spermatozoa. Ejaculated, density-purified, highly motile human spermatozoa were exposed to mobile phone radiation at specific absorption rates (SARs) of 2.0 and 5.7 W/kg. At various times after exposure, flow cytometry was used to examine caspase 3 activity, externalization of phosphatidylserine (PS), induction of DNA strand breaks, and generation of reactive oxygen species. Mobile phone radiation had no statistically significant effect on any of the parameters

studied. This suggests that the impairment of fertility reported in some studies was not caused by the induction of apoptosis in spermatozoa.

(E) Ferreira AR, Knakievicz T, de Bittencourt Pasquali MA, Gelain DP, Dal-Pizzol F, Fernandez CE, de Almeida de Salles AA, Ferreira HB, Moreira JC. Ultra high frequency-electromagnetic field irradiation during pregnancy leads to an increase in erythrocytes micronuclei incidence in rat offspring. Life Sci 80: 43-50, 2006. (GT, OX, LE, DE)

Mobile telephones and their base stations are an important ultra high frequency-electromagnetic field (UHF-EMF) source and their utilization is increasing all over the world. Epidemiological studies suggested that low energy UHF-EMF emitted from a cellular telephone may cause biological effects, such as DNA damage and changes on oxidative metabolism. An in vivo mammalian cytogenetic test, the micronucleus (MN) assay, was used to investigate the occurrence of chromosomal damage in erythrocytes from rat offspring exposed to a non-thermal UHF-EMF from a cellular phone during their embryogenesis; the irradiated group showed a significant increase in MN occurrence. In order to investigate if UHF-EMF could also alter oxidative parameters in the peripheral blood and in the liver - an important hematopoietic tissue in rat embryos and newborns - we also measured the activity of antioxidant enzymes, quantified total sulfhydryl content, protein carbonyl groups, thiobarbituric acid-reactive species and total non-enzymatic antioxidant defense. No significant differences were found in any oxidative parameter of offspring blood and liver. The average number of pups in each litter has also not been significantly altered. Our results suggest that, under our experimental conditions, UHF-EMF is able to induce a genotoxic response in hematopoietic tissue during the embryogenesis through an unknown mechanism.

(NE) Finnie JW, Cai Z, Blumbergs PC, Manavis J, Kuchel TR. 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. Pathology. 38(4):333-335, 2006. (GE, DE)

AIMS: To study immediate early gene, c-fos, expression as a marker of neural stress after whole of gestation exposure of the fetal mouse brain to mobile telephone-type radiofrequency fields. METHODS: Using a purpose-designed exposure system at 900 MHz, pregnant mice were given a single, far-field, whole body exposure at a specific absorption rate of 4 W/kg for 60 min/day from day 1 to day 19 of gestation. Pregnant control mice were sham-exposed or freely mobile in a cage without further restraint. Immediately prior to parturition on gestational day 19, fetal heads were collected, fixed in 4% paraformaldehyde and paraffin embedded. Any stress response in the brain was detected by c-fos immunohistochemistry in the cerebral cortex, basal ganglia, thalamus, hippocampus, midbrain, cerebellum and medulla. RESULTS: c-fos expression was of limited, but consistent, neuroanatomical distribution and there was no difference in immunoreactivity between exposed and control brains. CONCLUSION: In this animal model, no stress response was detected in the fetal brain using c-fos immunohistochemistry after whole of gestation exposure to mobile telephony.

(E) Franzellitti S, Valbonesi P, Ciancaglini N, Biondi C, Contin A, Bersani F, Fabbri E. Transient DNA damage induced by high-frequency electromagnetic fields (GSM 1.8 GHz)

in the human trophoblast HTR-8/SVneo cell line evaluated with the alkaline comet assay. Mutat Res 683(1-2):35-42, 2010. (GT, WS)

One of the most controversial issue regarding high-frequency electromagnetic fields (HF-EMF) is their putative capacity to affect DNA integrity. This is of particular concern due to the increasing use of HF-EMF in communication technologies, including mobile phones. Although epidemiological studies report no detrimental effects on human health, the possible disturbance generated by HF-EMF on cell physiology remains controversial. In addition, the question remains as to whether cells are able to compensate their potential effects. We have previously reported that a 1-h exposure to amplitude-modulated 1.8 GHz sinusoidal waves (GSM-217 Hz, SAR=2 W/kg) largely used in mobile telephony did not cause increased levels of primary DNA damage in human trophoblast HTR-8/SVneo cells. Nevertheless, further investigations on trophoblast cell responses after exposure to GSM signals of different types and durations were considered of interest. In the present work, HTR-8/SVneo cells were exposed for 4, 16 or 24h to 1.8 GHz continuous wave (CW) and different GSM signals, namely GSM-217 Hz and GSM-Talk (intermittent exposure: 5 min field on, 10 min field off). The alkaline comet assay was used to evaluate primary DNA damages and/or strand breaks due to uncompleted repair processes in HF-EMF exposed samples. The amplitude-modulated signals GSM-217 Hz and GSM-Talk induced a significant increase in comet parameters in trophoblast cells after 16 and 24h of exposure, while the un-modulated CW was ineffective. However, alterations were rapidly recovered and the DNA integrity of HF-EMF exposed cells was similar to that of sham-exposed cells within 2h of recovery in the absence irradiation. Our data suggest that HF-EMF with a carrier frequency and modulation scheme typical of the GSM signal may affect the DNA integrity.

(E) Furtado-Filho OV, Borba JB, Dallegrave A, Pizzolato TM, Henriques JA, Moreira JC, Saffi J. 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. Int J Radiat Biol. 2013 Jul 25. [Epub ahead of print] (LE, GT, OX)

Purpose: To assess the effect of 950 MHz ultra-high-frequency electromagnetic radiation (UHF EMR) on biomarkers of oxidative damage, as well as to verify the concentration of unsaturated fatty acids (UFA) and the expression of the catalase in the livers of rats of different ages. Materials and methods: Twelve rats were equally divided into two groups as controls (CR) and exposed (ER), for each age (0, 6, 15 and 30 days). Radiation exposure lasted half an hour per day for up to 51 days (21 days of gestation and 6, 15 or 30 days of life outside the womb). The specific absorption rate (SAR) ranged from 1.3-1.0 W/kg. The damage to lipids, proteins and DNA was verified by thiobarbituric acid reactive substances (TBARS), protein carbonyls and comets, respectively. UFA were determined by gas chromatography with a flame ionization detector. The expression of catalase was by Western blotting. Results: The neonates had low levels of TBARS and concentrations of UFA after exposure. There was no age difference in the accumulation of protein carbonyls for any age. The DNA damage of ER 15 or 30 days was different. The exposed neonates exhibited lower expression of catalase. Conclusions: 950 MHz UHF EMR does not cause oxidative stress (OS), and it is not genotoxic to the livers of neonates or those of 6 and 15 day old rats, but it changes the concentrations of polyunsaturated fatty acid (PUFA) in neonates. For rats of 30 days, no OS, but it is genotoxic to the livers of ER to total body irradiation.

(E) Gajski G, Garaj-Vrhovac V. Radioprotective effects of honeybee venom (*Apis mellifera*) against 915-MHz microwave radiation-induced DNA damage in wistar rat lymphocytes: in vitro study. Int J Toxicol 28:88-98, 2009. (GT, OX)

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. Whole blood lymphocytes of Wistar rats are treated with 1 microg/mL bee venom 4 hours prior to and immediately before irradiation. Standard and formamidopyrimidine-DNA glycosylase (Fpg)-modified comet assays are used to assess basal and oxidative DNA damage produced by reactive oxygen species. Bee venom shows a decrease in DNA damage compared with irradiated samples. Parameters of Fpg-modified comet assay are statistically different from controls, making this assay more sensitive and suggesting that oxidative stress is a possible mechanism of DNA damage induction. 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.

(E) Gandhi G, Anita, Genetic damage in mobile phone users: some preliminary findings. Ind J Hum Genet 11:99-104, 2005. (GT, HU)

BACKGROUND: The impact of microwave (MW)/radio frequency radiation (RFR) on important biological parameters is probably more than a simply thermal one. Exposure to radio frequency (RF) signals generated by the use of cellular telephones have increased dramatically and reported to affect physiological, neurological, cognitive and behavioural changes and to induce, initiate and promote carcinogenesis. Genotoxicity of RFR has also been reported in various test systems after in vitro and/or in vivo exposure but none in mobile phone users. **AIMS:** 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. **METHODS:** DNA damage was assessed using the single cell gel electrophoresis assay and aneugenic and clastogenic damage by the in vivo capillary blood micronucleus test (MNT) in a total of 24 mobile phone users. **RESULTS:** Mean comet tail length (26.76 ± 0.054 mm; 39.75% of cells damaged) in mobile phone users was highly significant from that in the control group. The in vivo capillary blood MNT also revealed highly significant (0.25) frequency of micronucleated (MNd) cells. **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.

(E) Gandhi G, Singh P. Cytogenetic damage in mobile phone users: preliminary data. Int J Hum Genet 5:259-265, 2005. (GT, HU)

Mobile telephones, sometimes called cellular (cell) phones or handies, are now an integral part of modern life. The mobile phone handsets are low-powered radiofrequency transmitters, emitting maximum powers in the range of 0.2 to 0.6 watts. Scientific concerns have increased sufficiently over the possible hazard to health from using cell phones. The reported adverse health effects include physiological, behavioural and cognitive changes as well as tumour formation and genetic damage. However findings are controversial and no consensus exists. Genotoxicity has been observed either in lower organisms or in vitro studies. The aim of the present study hence was to detect any cytogenetic damage in mobile phone users by analysing short term peripheral lymphocyte 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.

(E) Garaj-Vrhovac V, Gajski G, Pažanin S, Sarolić A, Domijan AM, Flajs D, Peraica M. Assessment of cytogenetic damage and oxidative stress in personnel occupationally exposed to the pulsed microwave radiation of marine radar equipment. Int J Hyg Environ Health. 4(1):59-65, 2011. (GT, HU, OX)

Due to increased usage of microwave radiation, there are concerns of its adverse effect in today's society. Keeping this in view, study was aimed at workers occupationally exposed to pulsed microwave radiation, originating from marine radars. Electromagnetic field strength was measured at assigned marine radar frequencies (3 GHz, 5.5 GHz and 9.4 GHz) and corresponding specific absorption rate values were determined. Parameters of the comet assay and micronucleus test were studied both in the exposed workers and in corresponding unexposed subjects. Differences between mean tail intensity (0.67 vs. 1.22) and moment (0.08 vs. 0.16) as comet assay parameters and micronucleus test parameters (micronuclei, nucleoplasmic bridges and nuclear buds) were statistically significant between the two examined groups, suggesting that cytogenetic alterations occurred after microwave exposure. Concentrations of glutathione and malondialdehyde were measured spectrophotometrically and using high performance liquid chromatography. The glutathione concentration in exposed group was significantly lower than in controls (1.24 vs. 0.53) whereas the concentration of malondialdehyde was significantly higher (1.74 vs. 3.17), indicating oxidative stress. Results suggests that pulsed microwaves from working environment can be the cause of genetic and cell alterations and that oxidative stress can be one of the possible mechanisms of DNA and cell damage.

(E) Guler G, Tomruk A, Ozgur E, Seyhan N. The effect of radiofrequency radiation on DNA and lipid damage in non-pregnant and pregnant rabbits and their newborns. Gen Physiol Biophys 29:59-66, 2010. (GT, OX, LE, DE)

The concerns of people on possible adverse health effects of radiofrequency radiation (RFR) generated from mobile phones as well as their supporting transmitters (base stations) have increased markedly. RFR effect on oversensitive people, such as pregnant women and their developing fetuses, and older people is another source of concern that should be considered. In this study, oxidative DNA damage and lipid peroxidation levels in the brain tissue of pregnant and non-pregnant New Zealand White rabbits and their newborns exposed to RFR were investigated. Thirteen-month-old rabbits were studied in four groups as non-pregnant-control, non-pregnant-RFR exposed, pregnant-control and pregnant-RFR exposed. They were exposed to RFR (1800 MHz GSM; 14 V/m as reference level) for 15 min/day during 7 days. Malondialdehyde (MDA) and 8-hydroxy-2'-deoxyguanosine (8-OHdG) levels were analyzed. MDA and 8-OHdG levels of non-pregnant and pregnant-RFR exposed animals significantly increased with respect to controls ($p < 0.001$, Mann-Whitney test). No difference was found in the newborns ($p > 0.05$, Mann-Whitney). There exist very few experimental studies on the effects of RFR during pregnancy. It would be beneficial to increase the number of these studies in order to establish international standards for the protection of pregnant women from RFR.

(E) Güler G, Tomruk A, Ozgur E, Sahin D, Sepici A, Altan N, Seyhan N. The effect of radiofrequency radiation on DNA and lipid damage in female and male infant rabbits. Int J Radiat Biol. 88(4):367-373, 2012. (LE, GT, OX, DE)

PURPOSE: We aimed to design a prolonged radiofrequency (RF) radiation exposure and investigate in an animal model, possible bio-effects of RF radiation on the ongoing developmental stages of children from conception to childhood. **MATERIALS AND METHODS:** A total of 72 New Zealand female and male white rabbits aged one month were used. Females were exposed to RF radiation for 15 min/day during 7 days, whereas males were exposed to the same level of radiation for 15 min/day during 14 days. Thirty-six female and 36 male infant rabbits were randomly divided into four groups: Group I [Intrauterine (IU) exposure (-); Extrauterine (EU) exposure (-)]: Sham exposure which means rabbits were exposed to 1800 MHz Global System for Mobile Telecommunication (GSM)-like RF signals neither in the IU nor in the EU periods. Group II [IU exposure (-); EU exposure (+)]: Infant rabbits were exposed to 1800 MHz GSM-like RF signals when they reached one month of age. Group III [IU exposure (+); EU exposure (-)]: Infant rabbits were exposed to 1800 MHz GSM-like RF signals in the IU period (between 15th and 22nd days of the gestational period). Group IV [IU exposure (+); EU exposure (+)]: Infant rabbits were exposed to 1800 MHz GSM-like RF signals both in the IU period (between 15th and 22nd days of the gestational period) and in the EU period when they reached one month of age. Biochemical analysis for lipid peroxidation and DNA damage were carried out in the livers of all rabbits. **RESULTS:** Lipid peroxidation levels in the liver tissues of female and male infant rabbits increased under RF radiation exposure. Liver 8-hydroxy-2'-deoxyguanosine (8-OHdG) levels of female rabbits exposed to RF radiation were also found to increase when compared with the levels of non-exposed infants. However, there were no changes in liver 8-OHdG levels of male rabbits under RF exposure. **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.

(NE) Gurbuz N, Sirav B, Yuvaci HU, Turhan N, Coskun ZK, Seyhan N. Is there any possible genotoxic effect in exfoliated bladder cells of rat under the exposure of 1800 MHz GSM-like modulated radio frequency radiation (RFR)? Electromagn Biol Med. 29(3):98-104, 2010. (LE, GT)

People are exposed to many carcinogenic and mutagenic chemicals in their everyday lives. These include antineoplastic drugs, Polycyclic aromatic hydrocarbons (PAH)s, aromatic amines, nitrosamines, metals, and electromagnetic radiation. Based on the state of knowledge acquired during the last 50 years of research on possible biological effects of electromagnetic fields (EMF), the majority of the scientific community is convinced that exposure to EMF below the existing security limits does not cause a risk to the health of the general public. However, this position is questioned by others, who are of the opinion that the available research data are contradictory or inconsistent and, therefore, unreliable. In this study, we aimed to investigate if there is any effect of 1800 MHz GSM modulated radio frequency radiation (RFR) on the number of micronucleus in exfoliated bladder cells of rat which will be informative about the genotoxic damage. Exposure period was 20 min/day, 5 days/week during a month. Six female Wistar rats were used for two groups: Group I (n=6): controls; Group II (n=6): 1.8 GHz exposed animals.

1800 MHz RFR did not showed a significant MN frequencies in rat bladder cells when compared with the control group ($p>0.05$). 1800 MHz RFR-exposed animals did not produce any genotoxic effect when compared with the control group ($p>0.05$). Kinetic studies are important for any biomarker, especially those in which tissue differentiation and maturation processes will heavily influence the time between induction of damage and collection of damaged cells for micronucleus analysis.

(NE) Gurbuz N, Sirav B, Colbay M, Yetkin I, Seyhan N. No genotoxic effect in exfoliated bladder cells of rat under the exposure of 1800 and 2100-MHz radio frequency radiation. Electromagn Biol Med. 2013 Nov 27. [Epub ahead of print] (GT, LE)

Abstract In this study, we aimed to investigate the effects of 1800 and 2100 MHz Radio Frequency (RF) radiation on the number of micronucleus (MN) in exfoliated bladder cells of rat which shows the genotoxic damage. Exposure period was 30 min/day, 6 days/week for a month and two months exposure periods. Thirty male wistar albino rats were used for five groups: Group I (n = 6): 1800 MHz RF exposed animals for one month, Group II (n = 6): 2100 MHz RF exposed animals for one month, Group III (n = 6): 2100 MHz RF exposed for two months, Group IV (n = 6): control group for one month, Group V (n = 6): control group for two months. Rats of the control groups were housed in their home cages during the entire experimental period without subjecting to any experimental manipulation. 1800 and 2100 MHz RF exposures did not result in any significant MN frequencies in rat bladder cells with respect to the control groups ($p>0.05$). There was no statistically significant difference between 2100 MHz RF exposed groups, either. Further studies are needed to demonstrate if there is any genotoxic effect, micronucleus formation in other tissues of rats.

(NE) Hansteen IL, Lågeide L, Clausen KO, Haugan V, Svendsen M, Eriksen JG, Skiaker R, Hauger E, Vistnes AI, Kure EH. Cytogenetic effects of 18.0 and 16.5 GHz microwave radiation on human lymphocytes in vitro. Anticancer Res 29:2885-2892, 2009. (GT, IA, WS)

BACKGROUND: There are few cell studies on the direct genotoxic effects of microwave radiation. In this study, cytogenetic effects of microwave radiation alone or in combination with mitomycin C (MMC) were investigated. MATERIALS AND METHODS: Lymphocytes from two smoking and four non-smoking donors were exposed for 53 hours in vitro to 1.0 W/m continuous-wave radiation at 18.0 GHz or 10 W/m pulsed-wave at 16.5 GHz, alone or in combination with MMC. DNA synthesis and repair were inhibited in vitro in some cultures. RESULTS: No synergistic effect was observed in cells exposed to combinations of microwave radiation and in vitro exposure to MMC, or to cells pre-exposed in vivo to tobacco smoke. For the 16.5 GHz pulsed exposure, a non-significant trend consisting of an increase in aberration frequencies with microwave radiation was shown for the DNA synthesis and repair inhibited cultures both with and without MMC. CONCLUSION: Neither 18.0 GHz continuous-wave nor 16.5 GHz pulsed-wave exposure to human lymphocytes in vitro induced statistically significant increases in chromosomal aberration frequencies. 16.5 GHz pulsed-wave exposure requires further documentation before a true negative conclusion can be drawn.

(NE) Hansteen IL, Clausen KO, Haugan V, Svendsen M, Svendsen MV, Eriksen JG, Skiaker R, Hauger E, Lågeide L, Vistnes AI, Kure EH. Cytogenetic effects of exposure to

2.3 GHz radiofrequency radiation on human lymphocytes in vitro. Anticancer Res 29:4323-4330, 2009. (GT, IA)

BACKGROUND: No previous in vitro studies have tested radio frequency radiation for at least one full cell cycle in culture. The aim was to test if exposure used in mobile phones and wireless network technologies would induce DNA damage in cultured human lymphocytes with and without a known clastogen. MATERIALS AND METHODS: Lymphocytes from six donors were exposed to 2.3 GHz, 10 W/m continuous waves, or 2.3 GHz, 10 W/m pulsed waves (200 Hz pulse frequency, 50% duty cycle). Mitomycin C was added to half of the cultures. DNA synthesis and repair were inhibited in one experiment. RESULTS: No statistically significant differences were observed between control and exposed cultures. A weak trend for more chromosomal damage with the interaction of pulsed fields with mitomycin C compared to a constant field was observed. CONCLUSION: Exposure during the whole cell cycle in inhibited cultures did not result in significant differences in chromosomal aberrations as compared to controls.

(E) Hekmat A, Saboury AA, Moosavi-Movahedi AA. The toxic effects of mobile phone radiofrequency (940MHz) on the structure of calf thymus DNA. Ecotoxicol Environ Saf. 2012 Nov 16. pii: S0147-6513(12)00368-5. doi: 10.1016/j.ecoenv.2012.10.016. [Epub ahead of print] (GT)

Currently, the biological effects of nonionizing electromagnetic fields (EMFs) including radiofrequency (RF) radiation have been the subject of numerous experimental and theoretical studies. The aim of this study is to evaluate the possible biological effects of mobile phone RF (940MHz, 15V/m and SAR=40mW/kg) on the structure of calf thymus DNA (ct DNA) immediately after exposure and 2h after 45min exposure via diverse range of spectroscopic instruments. The UV-vis and circular dichroism (CD) experiments depict that mobile phone EMFs can remarkably cause disturbance on ct DNA structure. In addition, the DNA samples, immediately after exposure and 2h after 45min exposure, are relatively thermally unstable compared to the DNA solution, which was placed in a small shielded box (unexposed ct DNA). Furthermore, the exposed DNA samples (the DNA samples that were exposed to 940MHz EMF) have more fluorescence emission when compared with the unexposed DNA, which may have occurred attributable to expansion of the exposed DNA structure. The results of dynamic light scattering (DLS) and zeta potential experiments demonstrate that RF-EMFs lead to increment in the surface charge and size of DNA. The structure of DNA immediately after exposure is not significantly different from the DNA sample 2h after 45min exposure. In other words, the EMF-induced conformational changes are irreversible. Collectively, our results reveal that 940MHz can alter the structure of DNA. The displacement of electrons in DNA by EMFs may lead to conformational changes of DNA and DNA disaggregation. Results from this study could have an important implication on the health effects of RF-EMFs exposure. In addition, this finding could proffer a novel strategy for the development of next generation of mobile phone.

(NE) [Hintzsche H](#), [Stopper H](#). Micronucleus frequency in buccal mucosa cells of mobile phone users. [Toxicol Lett](#). 193(1):124-130, 2010. (GT, HU)

Mobile phones are being used extensively throughout the world, with more than four billion accounts existing in 2009. This technology applies electromagnetic radiation in the microwave

range. Health effects of this radiation have been subject of debate for a long time, both within the scientific community and within the general public. This study investigated the effect of mobile phone use on genomic instability of the human oral cavity's mucosa cells. 131 Individuals donated buccal mucosa cells extracted by slightly scraping the oral cavity with a cotton swab. Every participant filled out a questionnaire about mobile phone use including duration of weekly use, overall period of exposure and headset usage. 13 Individuals did not use mobile phones at all, 85 reported using the mobile phone for three hours per week or less, and 33 reported use of more than three hours per week. Additionally, information on age, gender, body weight, smoking status, medication and nutrition was retrieved. For staining of the cells a procedure using alpha-tubulin-antibody and chromomycin A(3) was applied. Micronuclei and other markers were evaluated in 1000 cells per individual at the microscope. A second scorer counted another 1000 cells, resulting in 2000 analyzed cells per individual. Mobile phone use did not lead to a significantly increased frequency of micronuclei.

(NE) Hintzsche H, Jastrow C, Kleine-Ostmann T, Schrader T, Stopper H. 900 MHz radiation does not induce micronucleus formation in different cell types. Mutagenesis. 27(4):477-483, 2012 . (GT)

The exposure of the population to non-ionising electromagnetic radiation is still increasing, mainly due to mobile communication. Whether low-intensity electromagnetic fields can cause other effects apart from heating has been a subject of debate. One of the effects, which were proposed to be caused by mobile phone radiation, is the occurrence of mitotic disturbances. The aim of this study was to investigate possible consequences of these mitotic disturbances as manifest genomic damage, i.e. micronucleus induction. Cells were irradiated at a frequency of 900 MHz, which is located in one of the main frequency bands applied for mobile communication. Two cell types were used, HaCaT cells as human cells and A(L) cells (human-hamster hybrid cells), in which mitotic disturbances had been reported to occur. After different post-exposure incubation periods, cells were fixed and micronucleus frequencies were evaluated. Both cell types did not show any genomic damage after exposure. To adapt the protocol for the micronucleus test into the direction of the protocol for mitotic disturbances, the post-exposure incubation period was reduced and exposure time was extended to one cell cycle length. This did not result in any increase of the genomic damage. In conclusion, micronucleus induction was not observed as a consequence of exposure to non-ionising radiation, even though this agent was reported to cause mitotic disturbances under similar experimental conditions.

(NE) Hirose H, Sakuma N, Kaji N, Suhara T, Sekijima M, Nojima T, Miyakoshi J. Phosphorylation and gene expression of p53 are not affected in human cells exposed to 2.1425 GHz band CW or W-CDMA modulated radiation allocated to mobile radio base stations. Bioelectromagnetics 27:494-504, 2006. (GT)

A large-scale in vitro study focusing on low-level radiofrequency (RF) fields from mobile radio base stations employing the International Mobile Telecommunication 2000 (IMT-2000) cellular system was conducted to test the hypothesis that modulated RF fields induce apoptosis or other cellular stress response that activate p53 or the p53-signaling pathway. First, we evaluated the response of human cells to microwave exposure at a specific absorption rate (SAR) of 80 mW/kg, which corresponds to the limit of the average whole-body SAR for general public exposure defined as a basic restriction by the International Commission on Non-Ionizing

Radiation Protection (ICNIRP) guidelines. Second, we investigated whether continuous wave (CW) and wideband code division multiple access (W-CDMA) modulated signal RF fields at 2.1425 GHz induced apoptosis or any signs of stress. Human glioblastoma A172 cells were exposed to W-CDMA radiation at SARs of 80, 250, and 800 mW/kg, and CW radiation at 80 mW/kg for 24 or 48 h. Human IMR-90 fibroblasts from fetal lungs were exposed to both W-CDMA and CW radiation at a SAR of 80 mW/kg for 28 h. Under the RF field exposure conditions described above, no significant differences in the percentage of apoptotic cells were observed between the test groups exposed to RF signals and the sham-exposed negative controls, as evaluated by the Annexin V affinity assay. No significant differences in expression levels of phosphorylated p53 at serine 15 or total p53 were observed between the test groups and the negative controls by the bead-based multiplex assay. Moreover, microarray hybridization and real-time RT-PCR analysis showed no noticeable differences in gene expression of the subsequent downstream targets of p53 signaling involved in apoptosis between the test groups and the negative controls. Our results confirm that exposure to low-level RF signals up to 800 mW/kg does not induce p53-dependent apoptosis, DNA damage, or other stress response in human cells.

(NE) Hirose H, Sakuma N, Kaji N, Nakayama K, Inoue K, Sekijima M, Nojima T, Miyakoshi J. Mobile phone base station-emitted radiation does not induce phosphorylation of Hsp27. Bioelectromagnetics 28:99-108, 2007. (GE)

An in vitro study focusing on the effects of low-level radiofrequency (RF) fields from mobile radio base stations employing the International Mobile Telecommunication 2000 (IMT-2000) cellular system was conducted to test the hypothesis that modulated RF fields act to induce phosphorylation and overexpression of heat shock protein hsp27. First, we evaluated the responses of human cells to microwave exposure at a specific absorption rate (SAR) of 80 mW/kg, which corresponds to the limit of the average whole-body SAR for general public exposure defined as a basic restriction in the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guidelines. Second, we investigated whether continuous wave (CW) and Wideband Code Division Multiple Access (W-CDMA) modulated signal RF fields at 2.1425 GHz induced activation or gene expression of hsp27 and other heat shock proteins (hsps). Human glioblastoma A172 cells were exposed to W-CDMA radiation at SARs of 80 and 800 mW/kg for 2-48 h, and CW radiation at 80 mW/kg for 24 h. Human IMR-90 fibroblasts from fetal lungs were exposed to W-CDMA at 80 and 800 mW/kg for 2 or 28 h, and CW at 80 mW/kg for 28 h. Under the RF field exposure conditions described above, no significant differences in the expression levels of phosphorylated hsp27 at serine 82 (hsp27[pS82]) were observed between the test groups exposed to W-CDMA or CW signal and the sham-exposed negative controls, as evaluated immediately after the exposure periods by bead-based multiplex assays. Moreover, no noticeable differences in the gene expression of hsps were observed between the test groups and the negative controls by DNA Chip analysis. Our results confirm that exposure to low-level RF field up to 800 mW/kg does not induce phosphorylation of hsp27 or expression of hsp gene family.

(NE) Huang TQ, Lee MS, Oh E, Zhang BT, Seo JS, Park WY. Molecular responses of Jurkat T-cells to 1763 MHz radiofrequency radiation. Int J Radiat Biol 84:734-741, 2008. (GT, GE)

PURPOSE: The biological effects of exposure to mobile phone emitted radiofrequency (RF) radiation are the subject of intense study, yet the hypothesis that RF exposure is a potential health hazard remains controversial. In this paper, we monitored cellular and molecular changes in Jurkat human T lymphoma cells after irradiating with 1763 MHz RF radiation to understand the effect on RF radiation in immune cells. **MATERIALS AND METHODS:** Jurkat T-cells were exposed to RF radiation to assess the effects on cell proliferation, cell cycle progression, DNA damage and gene expression. Jurkat cells were exposed to 1763 MHz RF radiation at 10 W/kg specific absorption rate (SAR) and compared to sham exposed cells. **RESULTS:** RF exposure did not produce significant changes in cell numbers, cell cycle distributions, or levels of DNA damage. In genome-wide analysis of gene expressions, there were no genes changed more than two-fold upon RF-radiation while ten genes change to 1.3 approximately 1.8-fold. Among ten genes, two cytokine receptor genes such as chemokine (C-X-C motif) receptor 3 (CXCR3) and interleukin 1 receptor, type II (IL1R2) were down-regulated upon RF radiation, but they were not directly related to cell proliferation or DNA damage responses. **CONCLUSION:** These results indicate that the alterations in cell proliferation, cell cycle progression, DNA integrity or global gene expression was not detected upon 1763 MHz RF radiation under 10 W/kg SAR for 24 h to Jurkat T cells.

(NE) Huang TQ, Lee MS, Oh EH, Kalinec F, Zhang BT, Seo JS, Park WY. Characterization of biological effect of 1763 MHz radiofrequency exposure on auditory hair cells. Int J Radiat Biol 84:909-915, 2008. (GT, GE)

Purpose: Radiofrequency (RF) exposure at the frequency of mobile phones has been reported not to induce cellular damage in in vitro and in vivo models. We chose HEI-OC1 immortalized mouse auditory hair cells to characterize the cellular response to 1763 MHz RF exposure, because auditory cells could be exposed to mobile phone frequencies. **Materials and methods:** Cells were exposed to 1763 MHz RF at a 20 W/kg specific absorption rate (SAR) in a code division multiple access (CDMA) exposure chamber for 24 and 48 h to check for changes in cell cycle, DNA damage, stress response, and gene expression. **Results:** Neither of cell cycle changes nor DNA damage was detected in RF-exposed cells. The expression of heat shock proteins (HSP) and the phosphorylation of mitogen-activated protein kinases (MAPK) did not change, either. We tried to identify any alteration in gene expression using microarrays. Using the Applied Biosystems 1700 full genome expression mouse microarray, we found that only 29 genes (0.09% of total genes examined) were changed by more than 1.5-fold on RF exposure. **Conclusion:** From these results, we could not find any evidence of the induction of cellular responses, including cell cycle distribution, DNA damage, stress response and gene expression, after 1763 MHz RF exposure at an SAR of 20 W/kg in HEI-OC1 auditory hair cells.

(E) Jiang B, Nie J, Zhou Z, Zhang J, Tong J, Cao Y. Adaptive response in mice exposed to 900 MHz radiofrequency fields: primary DNA damage. PLoS One. 7(2):e32040, 2012. (LE, GT, IA)

The phenomenon of adaptive response (AR) in animal and human cells exposed to ionizing radiation is well documented in scientific literature. We have examined whether such AR could be induced in mice exposed to non-ionizing radiofrequency fields (RF) used for wireless communications. Mice were pre-exposed to 900 MHz RF at 120 $\mu\text{W}/\text{cm}^2$ power density for 4 hours/day for 1, 3, 5, 7 and 14 days and then subjected to an acute dose of 3 Gy γ -radiation. The

primary DNA damage in the form of alkali labile base damage and single strand breaks in the DNA of peripheral blood leukocytes was determined using the alkaline comet assay. The results indicated that the extent of damage in mice which were pre-exposed to RF for 1 day and then subjected to γ -radiation was similar and not significantly different from those exposed to γ -radiation alone. However, mice which were pre-exposed to RF for 3, 5, 7 and 14 days showed progressively decreased damage and was significantly different from those exposed to γ -radiation alone. Thus, the data indicated that RF pre-exposure is capable of inducing AR and suggested that the pre-exposure for more than 4 hours for 1 day is necessary to elicit such AR.

(NE) Juutilainen J, Heikkinen P, Soikkeli H, Mäki-Paakkanen J. Micronucleus frequency in erythrocytes of mice after long-term exposure to radiofrequency radiation. Int J Radiat Biol. 83(4):213-220, 2007. (LE, GT)

PURPOSE: The aim of the study was to investigate genotoxicity of long-term exposure to radiofrequency (RF) electromagnetic fields by measuring micronuclei in erythrocytes. The blood samples were collected in two animal studies evaluating possible cocarcinogenic effects of RF fields. **METHODS:** In study A, female CBA/S mice were exposed for 78 weeks (1.5 h/d, 5 d/week) to either a continuous 902.5 MHz signal similar to that emitted by analog NMT (Nordic Mobile Telephone) phones at a whole-body specific absorption rate (SAR) of 1.5 W/kg, or to a pulsed 902.4 MHz signal similar to that of digital GSM (Global System for Mobile Communications) phones at 0.35 W/kg. A third group was sham-exposed, and a fourth group served as cage controls. All but the cage control animals were exposed to 4 Gy of x-rays during three first weeks of the experiment. In study B, female transgenic mice (line K2) and their nontransgenic littermates were exposed for 52 weeks (1.5 h/d, 5 d/week). Two digital mobile phone signals, GSM and DAMPS (Digital Advanced Mobile Phone System), were used at 0.5 W/kg. All but the cage-control animals were exposed 3 times per week to an ultraviolet radiation dose of 1.2 MED (minimum erythema dose). **RESULTS AND CONCLUSIONS:** The results did not show any effects of RF fields on micronucleus frequency in polychromatic or normochromatic erythrocytes. The results were consistent in two mouse strains (and in a transgenic variant of the second strain), after 52 or 78 weeks of exposure, at three SAR levels relevant to human exposure from mobile phones, and for three different mobile signals.

(E) Karaca E, Durmaz B, Altug H, Yildiz T, Guducu C, Irgi M, Koksal MG, Ozkinay F, Gunduz C, Cogulu O. The genotoxic effect of radiofrequency waves on mouse brain. J Neurooncol 106:53-58, 2012. (GT, GE)

Erratum: J Neurooncol 2012 May;107:665.

Concerns about the health effects of radiofrequency (RF) waves have been raised because of the gradual increase in usage of cell phones, and there are scientific questions and debates about the safety of those instruments in daily life. The aim of this study is to evaluate the genotoxic effects of RF waves in an experimental brain cell culture model. Brain cell cultures of the mice were exposed to 10.715 GHz with specific absorption 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.

(E) Kesari KK, Behari J. Fifty-gigahertz Microwave exposure effect of radiations on rat brain. Appl Biochem Biotechnol 158:126-139, 2009. (GT, OX, LE)

The object of this study is to investigate the effects of 50-GHz microwave radiation on the brain of Wistar rats. Male rats of the Wistar strain were used in the study. Animals of 60-day age were divided into two groups-group 1, sham-exposed, and group 2, experimental (microwave-exposed). The rats were housed in a temperature-controlled room (25 degrees C) with constant humidity (40-50%) and received food and water ad libitum. During exposure, rats were placed in Plexiglas cages with drilled ventilation holes and kept in an anechoic chamber. The animals were exposed for 2 h a day for 45 days continuously at a power level of 0.86 $\mu\text{W}/\text{cm}^2$ with nominal specific absorption rate 8.0×10^{-4} w/kg. After the exposure period, the rats were killed and homogenized, and protein kinase C (PKC), DNA double-strand break, and antioxidant enzyme activity [superoxides dismutase (SOD), catalase, and glutathione peroxidase (GPx)] were estimated in the whole brain. Result shows that the chronic exposure to these radiations causes DNA double-strand break (head and tail length, intensity and tail migration) and a significant decrease in GPx and SOD activity ($p < 0.05$) in brain cells, whereas catalase activity shows significant increase in the exposed group of brain samples as compared with control ($p = <0.001$). In addition to these, PKC decreased significantly in whole brain and hippocampus ($p < 0.05$). All data are expressed as mean \pm standard deviation. We conclude that these radiations can have a significant effect on the whole brain.

(E) Kesari KK, Behari J, Kumar S. Mutagenic response of 2.45 GHz radiation exposure on rat brain. Int J Radiat Biol 86:334-343, 2010. (GT, OX, LE)

Purpose: To investigate the effect of 2.45 GHz microwave radiation on rat brain of male wistar strain. Material and methods: Male rats of wistar strain (35 days old with 130 ± 10 g body weight) were selected for this study. Animals were divided into two groups: Sham exposed and experimental. Animals were exposed for 2 h a day for 35 days to 2.45 GHz frequency at 0.34 mW/cm^2 power density. The whole body specific absorption rate (SAR) was estimated to be 0.11 W/Kg . Exposure took place in a ventilated Plexiglas cage and kept in anechoic chamber in a far field configuration from the horn antenna. After the completion of exposure period, rats were sacrificed and the whole brain tissue was dissected and used for study of double strand DNA (Deoxyribonucleic acid) breaks by micro gel electrophoresis and the statistical analysis was carried out using comet assay (IV-2 version software). Thereafter, antioxidant enzymes and histone kinase estimation was also performed. Results: A significant increase was observed in comet head ($P < 0.002$), tail length ($P < 0.0002$) and in tail movement ($P < 0.0001$) in exposed brain cells. An analysis of antioxidant enzymes glutathione peroxidase ($P < 0.005$), and superoxide dismutase ($P < 0.006$) showed a decrease while an increase in catalase ($P < 0.006$) was observed. A significant decrease ($P < 0.023$) in histone kinase was also recorded in the exposed group as compared to the control (sham-exposed) ones. One-way analysis of variance (ANOVA) method was adopted for statistical analysis. Conclusion: The study concludes that the chronic exposure to these radiations may cause significant damage to brain, which may be an indication of possible tumour promotion (Behari and Paulraj 2007).

(E) Khalil AM, Gagaa M, Alshamali A. 8-Oxo-7, 8-dihydro-2'-deoxyguanosine as a biomarker of DNA damage by mobile phone radiation. Hum Exp Toxicol 31(7):734-740, 2012. (GT, OX)

We examined the effect of exposure to mobile phone 1800 MHz radio frequency radiation (RFR) upon the urinary excretion of 8-oxo-7, 8-dihydro-2'-deoxyguanosine (8-oxodG), one major form of oxidative DNA damage, in adult male Sprague-Dawley rats. Twenty-four rats were used in three independent experiments (RFR exposed and control, 12 rats, each). The animals were exposed to RFR for 2 h from Global System for Mobile Communications (GSM) signal generator with whole-body-specific absorption rate of 1.0 W/kg. Urine samples were collected from the rat while housed in a metabolic cage during the exposure period over a 4-h period at 0.5, 1.0, 2.0 and 4.0 h from the beginning of exposure. In the control group, the signal generator was left in the turn-off position. The creatinine-standardized concentrations of 8-oxodG were measured. With the exception of the urine collected in the last half an hour of exposure, significant elevations were noticed in the levels of 8-oxodG in urine samples from rats exposed to RFR when compared to control animals. Significant differences were seen overall across time points of urine collection with a maximum at 1 h after exposure, suggesting repair of the DNA lesions leading to 8-oxodG formation.

(E) Kim JY, Hong SY, Lee YM, Yu SA, Koh WS, Hong JR, Son T, Chang SK, Lee M. In vitro assessment of clastogenicity of mobile-phone radiation (835 MHz) using the alkaline comet assay and chromosomal aberration test. Environ Toxicol 23:319-327, 2008. (GT, IA)

Recently we demonstrated that 835-MHz radiofrequency radiation electromagnetic fields (RF-EMF) neither affected the reverse mutation frequency nor accelerated DNA degradation in vitro. Here, two kinds of cytogenetic endpoints were further investigated on mammalian cells exposed to 835-MHz RF-EMF (the most widely used communication frequency band in Korean CDMA mobile phone networks) alone and in combination with model clastogens: in vitro alkaline comet assay and in vitro chromosome aberration (CA) test. No direct cytogenetic effect of 835-MHz RF-EMF was found in the in vitro CA test. The combined exposure of the cells to RF-EMF in the presence of ethylmethanesulfonate (EMS) revealed a weak and insignificant cytogenetic effect when compared to cells exposed to EMS alone in CA test. Also, the comet assay results to evaluate the ability of RF-EMF alone to damage DNA were nearly negative, although showing a small increase in tail moment. However, the applied RF-EMF had potentiation effect in comet assay when administered in combination with model clastogens (cyclophosphamide or 4-nitroquinoline 1-oxide). Thus, our results imply that we cannot confidently exclude any possibility of an increased risk of genetic damage, with important implications for the possible health effects of exposure to 835-MHz electromagnetic fields.

(E) Kumar S, Kesari KK, Behari J. Evaluation of genotoxic effects in male Wistar rats following microwave exposure. Indian J Exp Biol 48:586-592, 2010. (GT, OX)

Wistar rats (70 days old) were exposed for 2 h a day for 45 days continuously at 10 GHz [power density 0.214 mW/cm², specific absorption rate (SAR) 0.014 W/kg] and 50 GHz (power density 0.86 microW/cm², SAR 8.0 x10⁽⁻⁴⁾ W/kg). Micronuclei (MN), reactive oxygen species (ROS), and antioxidant enzymes activity were estimated in the blood cells and serum. These radiations induce micronuclei formation and significant increase in ROS production. Significant changes in the level of serum glutathione peroxidase, superoxide dismutase and catalase were observed in exposed group as compared with control group. It is concluded that microwave exposure can be affective at genetic level. This may be an indication of tumor promotion, which comes through the overproduction of reactive oxygen species.

(E) Lakshmi NK, Tiwari R, Bhargava SC, Ahuja YR. Investigations on DNA damage and frequency of micronuclei in occupational exposure to electromagnetic fields (EMFs) emitted from video display terminals (VDTs). Gen MolBiol 33, 154-158, 2010. (GT, HU, LE)

The potential effect of electromagnetic fields (EMFs) emitted from video display terminals (VDTs) to elicit biological response is a major concern for the public. The software professionals are subjected to cumulative EMFs in their occupational environments. This study was undertaken to evaluate DNA damage and incidences of micronuclei in such professionals. To the best of our knowledge, the present study is the first attempt to carry out cytogenetic investigations on assessing bioeffects in personal computer users. The study subjects (n = 138) included software professionals using VDTs for more than 2 years with age, gender, socioeconomic status matched controls (n = 151). DNA damage and frequency of micronuclei were evaluated using alkaline comet assay and cytochalasin blocked micronucleus assay respectively. Overall DNA damage and incidence of micronuclei showed no significant differences between the exposed and control subjects. With exposure characteristics, such as total duration (years) and frequency of use (minutes/day) sub-groups were assessed for such parameters. Although cumulative frequency of use showed no significant changes in the DNA integrity of the classified sub-groups, the long-term users (> 10 years) showed higher induction of DNA damage and increased frequency of micronuclei and micro nucleated cells.

(E) Liu C, Duan W, Xu S, Chen C, He M, Zhang L, Yu Z, Zhou Z. Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line. Toxicol Lett 218(1): 2-9, 2013a. (GT, OX, RP)

Whether exposure to radiofrequency electromagnetic radiation (RF-EMR) emitted from mobile phones can induce DNA damage in male germ cells remains unclear. In this study, we conducted a 24 h intermittent exposure (5 min on and 10 min off) of a mouse spermatocyte-derived GC-2 cell line to 1800 MHz Global System for Mobile Communication (GSM) signals in GSM-Talk mode at specific absorption rates (SAR) of 1 W/kg, 2 W/kg or 4 W/kg. Subsequently, through the use of formamidopyrimidine DNA glycosylase (FPG) in a modified comet assay, we determined that the extent of DNA migration was significantly increased at a SAR of 4 W/kg. Flow cytometry analysis demonstrated that levels of the DNA adduct 8-oxoguanine (8-oxoG) were also increased at a SAR of 4 W/kg. These increases were concomitant with similar increases in the generation of reactive oxygen species (ROS); these phenomena were mitigated by co-treatment with the antioxidant α -tocopherol. However, no detectable DNA strand breakage was observed by the alkaline comet assay. Taking together, these findings may imply the novel possibility that RF-EMR with insufficient energy for the direct induction of DNA strand breaks may produce genotoxicity through oxidative DNA base damage in male germ cells.

(E) Liu C, Gao P, Xu SC, Wang Y, Chen CH, He MD, Yu ZP, Zhang L, Zhou Z. Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin. Int J Radiat Biol. 2013b Aug 19. [Epub ahead of print] (GT, OX, RP)

Purpose: To evaluate whether exposure to mobile phone radiation (MPR) can induce DNA damage in male germ cells. Materials and methods: A mouse spermatocyte-derived GC-2 cell line was exposed to a commercial mobile phone handset once every 20 minutes in standby,

listen, dialed or dialing modes for 24 h. DNA damage was determined using an alkaline comet assay. Results: The levels of DNA damage were significantly increased following exposure to 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. Conclusions: These results regarding mode-dependent DNA damage have important implications for the safety of inappropriate mobile phone use by males of reproductive age and also suggest a simple preventive measure, keeping our body from mobile phones as far away as possible, not only during conversations but during "dialed" and "dialing" operation modes as well. Since the "dialed" mode is actually part of the standby mode, mobile phones should be kept at a safe distance from our body even during standby operation. Furthermore, the protective role of melatonin suggests that it may be a promising pharmacological candidate for preventing mobile phone use-related reproductive impairments.

(E) Lixia S, Yao K, Kaijun W, Deqiang L, Huajun H, Xiangwei G, Baohong W, Wei Z, Jianling L, Wei W. Effects of 1.8GHz radiofrequency field on DNA damage and expression of heat shock protein 70 in human lens epithelial cells. Mutat Res 602(1-2):135-42, 2006. (GT, GE)

To investigate the DNA damage, expression of heat shock protein 70 (Hsp70) and cell proliferation of human lens epithelial cells (hLEC) after exposure to the 1.8GHz radiofrequency field (RF) of a global system for mobile communications (GSM). An Xc-1800 RF exposure system was used to employ a GSM signal at 1.8GHz (217Hz amplitude-modulated) with the output power in the specific absorption rate (SAR) of 1, 2 and 3W/kg. After 2h exposure to RF, the DNA damage of hLEC was accessed by comet assay at five different incubation times: 0, 30, 60, 120 and 240min, respectively. Western blot and RT-PCR were used to determine the expression of Hsp70 in hLECs after RF exposure. The proliferation rate of cells was evaluated by bromodeoxyuridine incorporation on days 0, 1 and 4 after exposure. The results show that the difference of DNA-breaks between the exposed and sham-exposed (control) groups induced by 1 and 2W/kg irradiation were not significant at any incubation time point ($P>0.05$). The DNA damage caused by 3W/kg irradiation was significantly increased at the times of 0 and 30min after exposure ($P<0.05$), a phenomenon that could not be seen at the time points of 60, 120 or 240min ($P>0.05$). Detectable mRNA as well as protein expression of Hsp70 was found in all groups. Exposure at SARs of 2 and 3W/kg for 2h exhibited significantly increased Hsp70 protein expression ($P<0.05$), while no change in Hsp70 mRNA expression could be found in any of the groups ($P>0.05$). No difference of the cell proliferation rate between the sham-exposed and exposed cells was found at any exposure dose tested ($P>0.05$). The results indicate that exposure to non-thermal dosages of RF for wireless communications can induce no or repairable DNA damage and the increased Hsp70 protein expression in hLECs occurred without change in the cell proliferation rate. The non-thermal stress response of Hsp70 protein increase to RF exposure might be involved in protecting hLEC from DNA damage and maintaining the cellular capacity for proliferation.

(E) López-Martín E, Bregains J, Relova-Quinteiro JL, Cadarso-Suárez C, Jorge-Barreiro FJ, Ares-Pena FJ. The action of pulse-modulated GSM radiation increases regional changes in brain activity and c-Fos expression in cortical and subcortical areas in a rat

model of picrotoxin-induced seizure proneness. J Neurosci Res. 87(6):1484-1499, 2009. (AS, GE, WS, IA)

The action of the pulse-modulated GSM radiofrequency of mobile phones has been suggested as a physical phenomenon that might have biological effects on the mammalian central nervous system. In the present study, GSM-exposed picrotoxin-pretreated rats showed differences in clinical and EEG signs, and in c-Fos expression in the brain, with respect to picrotoxin-treated rats exposed to an equivalent dose of unmodulated radiation. Neither radiation treatment caused tissue heating, so thermal effects can be ruled out. The most marked effects of GSM radiation on c-Fos expression in picrotoxin-treated rats were observed in limbic structures, olfactory cortex areas and subcortical areas, the dentate gyrus, and the central lateral nucleus of the thalamic intralaminar nucleus group. Nonpicrotoxin-treated animals exposed to unmodulated radiation showed the highest levels of neuronal c-Fos expression in cortical areas. These results suggest a specific effect of the pulse modulation of GSM radiation on brain activity of a picrotoxin-induced seizure-proneness rat model and indicate that this mobile-phone-type radiation might induce regional changes in previous preexcitability conditions of neuronal activation.

(E) Luukkonen J, Hakulinen P, Mäki-Paakkanen J, Juutilainen J, Naarala J. Enhancement of chemically induced reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells by 872MHz radiofrequency radiation. Mutat Res 662:54-58, 2009. (GT, OX, WS)

The objective of the study was to investigate effects of 872 MHz radiofrequency (RF) radiation on intracellular reactive oxygen species (ROS) production and DNA damage at a relatively high SAR value (5W/kg). The experiments also involved combined exposure to RF radiation and menadione, a chemical inducing intracellular ROS production and DNA damage. The production of ROS was measured using the fluorescent probe dichlorofluorescein and DNA damage was evaluated by the Comet assay. Human SH-SY5Y neuroblastoma cells were exposed to RF radiation for 1h with or without menadione. Control cultures were sham exposed. Both continuous waves (CW) and a pulsed signal similar to that used in global system for mobile communications (GSM) mobile phones were used. Exposure to the CW RF radiation increased DNA breakage (p<0.01) in comparison to the cells exposed only to menadione. Comparison of the same groups also showed that ROS level was higher in cells exposed to CW RF radiation at 30 and 60 min after the end of exposure (p<0.05 and p<0.01, respectively). No effects of the GSM signal were seen on either ROS production or DNA damage. The results of the present study suggest that 872MHz CW RF radiation at 5W/kg might enhance chemically induced ROS production and thus cause secondary DNA damage. However, there is no known mechanism that would explain such effects from CW RF radiation but not from GSM modulated RF radiation at identical SAR.

(NE) Luukkonen J, Juutilainen J, Naarala J. Combined effects of 872 MHz radiofrequency radiation and ferrous chloride on reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells. Bioelectromagnetics 31:417-424, 2010. (GT, OX)

The aim of the present study was to investigate possible cooperative effects of radiofrequency (RF) radiation and ferrous chloride (FeCl) on reactive oxygen species (ROS) production and

DNA damage. In order to test intracellular ROS production as a possible underlying mechanism of DNA damage, we applied the fluorescent probe DCFH-DA. Integrity of DNA was quantified by alkaline comet assay. The exposures to 872 MHz RF radiation were conducted at a specific absorption rate (SAR) of 5 W/kg using continuous waves (CW) or a modulated signal similar to that used in Global System for Mobile Communications (GSM) phones. Four groups were included: Sham exposure (control), RF radiation, Chemical treatment, Chemical treatment, and RF radiation. In the ROS production experiments, human neuroblastoma (SH-SY5Y) cells were exposed to RF radiation and 10 microg/ml FeCl for 1 h. In the comet assay experiments, the exposure time was 3 h and an additional chemical (0.015% diethyl maleate) was used to make DNA damage level observable. The chemical treatments resulted in statistically significant responses, but no effects from either CW or modulated RF radiation were observed on ROS production, DNA damage or cell viability.

(NE) Maes A, Van Gorp U, Verschaeve L. Cytogenetic investigation of subjects professionally exposed to radiofrequency radiation. *Mutagenesis* 21:139-42, 2006. (GT, IA)

Nowadays, virtually everybody is exposed to radiofrequency radiation (RFR) from mobile phone base station antennas or other sources. At least according to some scientists, this exposure can have detrimental health effects. We investigated cytogenetic effects in peripheral blood lymphocytes from subjects who were professionally exposed to mobile phone electromagnetic fields in an attempt to demonstrate possible RFR-induced genetic effects. These subjects can be considered well suited for this purpose as their RFR exposure is 'normal' though rather high, and definitely higher than that of the 'general population'. The alkaline comet assay, sister chromatid exchange (SCE) and chromosome aberration tests revealed no evidence of RFR-induced genetic effects. Blood cells were also exposed to the well known chemical mutagen mitomycin C in order to investigate possible combined effects of RFR and the chemical. No cooperative action was found between the electromagnetic field exposure and the mutagen using either the comet assay or SCE test.

(E) Manti L, Braselmann H, Calabrese ML, Massa R, Pugliese M, Scampoli P, Sicignano G, Grossi G. Effects of modulated microwave radiation at cellular telephone frequency (1.95 GHz) on X-ray-induced chromosome aberrations in human lymphocytes in vitro. *Radiat Res* 169:575-583, 2008. (GT, IA)

The case for a DNA-damaging action produced by radiofrequency (RF) signals remains controversial despite extensive research. With the advent of the Universal Mobile Telecommunication System (UMTS) the number of RF-radiation-exposed individuals is likely to escalate. Since the epigenetic effects of RF radiation are poorly understood and since the potential modifications of repair efficiency after exposure to known cytotoxic agents such as ionizing radiation have been investigated infrequently thus far, we studied the influence of UMTS exposure on the yield of chromosome aberrations induced by X rays. Human peripheral blood lymphocytes were exposed in vitro to a UMTS signal (frequency carrier of 1.95 GHz) for 24 h at 0.5 and 2.0 W/kg specific absorption rate (SAR) using a previously characterized waveguide system. The frequency of chromosome aberrations was measured on metaphase spreads from cells given 4 Gy of X rays immediately before RF radiation or sham exposures by fluorescence in situ hybridization. Unirradiated controls were RF-radiation- or sham-exposed. No significant variations due to the UMTS exposure were found in the fraction of aberrant cells. However, the frequency of exchanges per cell was affected by the SAR, showing a small but

statistically significant increase of 0.11 exchange per cell compared to 0 W/kg SAR. We conclude that, although the 1.95 GHz signal (UMTS modulated) does not exacerbate the yield of aberrant cells caused by ionizing radiation, the overall burden of X-ray-induced chromosomal damage per cell in first-mitosis lymphocytes may be enhanced at 2.0 W/kg SAR. Hence the SAR may either influence the repair of X-ray-induced DNA breaks or alter the cell death pathways of the damage response.

(E) Mazor R, Korenstein-Ilan A, Barbul A, Eshet Y, Shahadi A, Jerby E, Korenstein R. Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes to radiofrequency electromagnetic fields for 72 hours. Radiat Res. 169(1):28-37, 2008. (GT)

We investigated the effects of 72 h in vitro exposure of 10 human lymphocyte samples to radiofrequency electromagnetic fields (800 MHz, continuous wave) on genomic instability. The lymphocytes were exposed in a specially designed waveguide resonator at specific absorption rates (SARs) of 2.9 and 4.1 W/kg in a temperature range of 36-37 degrees C. The induced aneuploidy of chromosomes 1, 10, 11 and 17 was determined by interphase FISH using semi-automated image analysis. We observed increased levels of aneuploidy depending on the chromosome studied as well as on the level of exposure. In chromosomes 1 and 10, there was increased aneuploidy at the higher SAR, while for chromosomes 11 and 17, the increases were observed only for the lower SAR. Multisomy (chromosomal gains) appeared to be the primary contributor to the increased aneuploidy. The effect of temperature on the level of aneuploidy was examined over the range of 33.5-40 degrees C for 72 h with no statistically significant difference in the level of aneuploidy compared to 37 degrees C. These findings suggest the possible existence of an athermal effect of RF radiation that causes increased levels of aneuploidy. These results contribute to the assessment of potential health risks after continuous chronic exposure to RF radiation at SARs close to the current levels set by ICNIRP guidelines.

(E) Nikolova T, Czyz J, Rolletschek A, Blyszczuk P, Fuchs J, Jovtchev G, Schuderer J, Kuster N, Wobus AM. Electromagnetic fields affect transcript levels of apoptosis-related genes in embryonic stem cell-derived neural progenitor cells. ASEB J 19(12):1686-1688, 2005. (GT, GE)

Mouse embryonic stem (ES) cells were used as an experimental model to study the effects of electromagnetic fields (EMF). ES-derived nestin-positive neural progenitor cells were exposed to extremely low frequency EMF simulating power line magnetic fields at 50 Hz (ELF-EMF) and to radiofrequency EMF simulating the Global System for Mobile Communication (GSM) signals at 1.71 GHz (RF-EMF). Following EMF exposure, cells were analyzed for transcript levels of cell cycle regulatory, apoptosis-related, and neural-specific genes and proteins; changes in proliferation; apoptosis; and cytogenetic effects. Quantitative RT-PCR analysis revealed that ELF-EMF exposure to ES-derived neural cells significantly affected transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes, whereas mRNA levels of neural-specific genes were not affected. RF-EMF exposure of neural progenitor cells resulted in down-regulation of neural-specific Nurr1 and in up-regulation of bax and GADD45 mRNA levels. Short-term RF-EMF exposure for 6 h, but not for 48 h, resulted in a low and transient increase of DNA double-strand breaks. No effects of ELF- and RF-EMF on mitochondrial function, nuclear apoptosis, cell proliferation, and

chromosomal alterations were observed. We may conclude that EMF exposure of ES-derived neural progenitor cells transiently affects the transcript level of genes related to apoptosis and cell cycle control. However, these responses are not associated with detectable changes of cell physiology, suggesting compensatory mechanisms at the translational and posttranslational level.

(E) Nittby H, Widegren B, Krogh M, Grafström G, Berlin H, Rehn G, Eberhardt JL, Malmgren L, Persson BRR, Salford L. Exposure to radiation from global system for mobile communications at 1,800 MHz significantly changes gene expression in rat hippocampus and cortex. Environmentalist 28(4), 458-465, 2008. (GE)

We have earlier shown that radio frequency electromagnetic fields can cause significant leakage of albumin through the blood–brain barrier of exposed rats as compared to non-exposed rats, and also significant neuronal damage in rat brains several weeks after a 2 h exposure to a mobile phone, at 915 MHz with a global system for mobile communications (GSM) frequency modulation, at whole-body specific absorption rate values (SAR) of 200, 20, 2, and 0.2 mW/kg. We have now studied whether 6 h of exposure to the radiation from a GSM mobile test phone at 1,800 MHz (at a whole-body SAR-value of 13 mW/kg, corresponding to a brain SAR-value of 30 mW/kg) has an effect upon the gene expression pattern in rat brain cortex and hippocampus—areas where we have observed albumin leakage from capillaries into neurons and neuronal damage. Microarray analysis of 31,099 rat genes, including splicing variants, was performed in cortex and hippocampus of 8 Fischer 344 rats, 4 animals exposed to global system for mobile communications electromagnetic fields for 6 h in an anechoic chamber, one rat at a time, and 4 controls kept as long in the same anechoic chamber without exposure, also in this case one rat at a time. Gene ontology analysis (using the gene ontology categories biological processes, molecular functions, and cell components) of the differentially expressed genes of the exposed animals versus the control group revealed the following highly significant altered gene categories in both cortex and hippocampus: extracellular region, signal transducer activity, intrinsic to membrane, and integral to membrane. The fact that most of these categories are connected with membrane functions may have a relation to our earlier observation of albumin transport through brain capillaries.

(E) Nylund R, Leszczynski D. 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. Proteomics 6:4769-4780, 2006. (GE, CS)

We have examined in vitro cell response to mobile phone radiation (900 MHz GSM signal) using two variants of human endothelial cell line: EA.hy926 and EA.hy926v1. Gene expression changes were examined in three experiments using cDNA Expression Arrays and protein expression changes were examined in ten experiments using 2-DE and PDQuest software. Obtained results show that gene and protein expression were altered, in both examined cell lines, in response to one hour mobile phone radiation exposure at an average specific absorption rate of 2.8 W/kg. However, the same genes and proteins were differently affected by the exposure in each of the cell lines. This suggests that the cell response to mobile phone radiation might be genome- and proteome-dependent. Therefore, it is likely that different types of cells and from different species might respond differently to mobile phone radiation or might have different sensitivity to this weak stimulus. Our findings might also explain, at least in part, the origin of discrepancies in replication studies between different laboratories.

(E) Panagopoulos DJ, Chavdoula ED, Nezis IP, Margaritis LH. Cell death induced by GSM 900-MHz and DCS 1800-MHz mobile telephony radiation. Mutat Res 626:69-78, 2007. (GT, RP)

In the present study, the TUNEL (Terminal deoxynucleotidyltransferase/UTP Nick End Labeling) assay - a well known technique widely used for detecting fragmented DNA in various types of cells - was used to detect cell death (DNA fragmentation) in a biological model, the early and mid stages of oogenesis of the insect *Drosophila melanogaster*. The flies were exposed *in vivo* to either GSM 900-MHz (Global System for Mobile telecommunications) or DCS 1800-MHz (Digital Cellular System) radiation from a common digital mobile phone, for few minutes per day during the first 6 days of their adult life. The exposure conditions were similar to those to which a mobile phone user is exposed, and were determined according to previous studies of ours [D.J Panagopoulos, A. Karabarounis, L.H. Margaritis, Effect of GSM 900-MHz mobile phone radiation on the reproductive capacity of *D. melanogaster*, *Electromagn. Biol Med* 23 (2004) 29-43; D.J Panagopoulos, N. Messini, A. Karabarounis, A.L. Philippetis, L.H. Margaritis, Radio frequency electromagnetic radiation within "safety levels" alters the physiological function of insects, in: P. Kostarakis, P. Stavroulakis (Eds.), *Proceedings of the Millennium International Workshop on Biological Effects of Electromagnetic Fields*, Heraklion, Crete, Greece, October 17-20, 2000, pp. 169-175, ISBN: 960-86733-0-5; D.J Panagopoulos, L.H. Margaritis, Effects of electromagnetic fields on the reproductive capacity of *D. melanogaster*, in: P. Stavroulakis (Ed.), *Biological Effects of Electromagnetic Fields*, Springer, 2003, pp. 545-578], which had shown a large decrease in the oviposition of the same insect caused by GSM radiation. Our present results suggest that the decrease in oviposition previously reported, is due to degeneration of large numbers of egg chambers after DNA fragmentation of their constituent cells, induced by both types of mobile telephony radiation. Induced cell death is recorded for the first time, in all types of cells constituting an egg chamber (follicle cells, nurse cells and the oocyte) and in all stages of the early and mid-oogenesis, from germarium to stage 10, during which programmed cell death does not physiologically occur. Germarium and stages 7-8 were found to be the most sensitive developmental stages also in response to electromagnetic stress induced by the GSM and DCS fields and, moreover, germarium was found to be even more sensitive than stages 7-8.

(NE) Papparini A, Rossi P, Gianfranceschi G, Brugaletta V, Falsaperla R, De Luca P, Romano Spica V. No evidence of major transcriptional changes in the brain of mice exposed to 1800 MHz GSM signal. Bioelectromagnetics. 29(4):312-323, 2008. (GE)

To analyze possible effects of microwaves on gene expression, mice were exposed to global system for mobile communication (GSM) 1800 MHz signal for 1 h at a whole body SAR of 1.1 W/kg. Gene expression was studied in the whole brain, where the average SAR was 0.2 W/kg, by expression microarrays containing over 22,600 probe sets. Comparison of data from sham and exposed animals showed no significant difference in gene expression modulation. However, when less stringent constraints were adopted to analyze microarray results, 75 genes were found to be modulated following exposure. Forty-two probes showed fold changes ranging from 1.5 to 2.8, whereas 33 were down-regulated from 0.67- to 0.29-fold changes, but these differences in gene expression were not confirmed by real-time PCR. Under these specific limited conditions, no consistent indication of gene expression modulation in whole mouse brain was found associated to GSM 1800 MHz exposure.

(E) Paulraj R, Behari J. Single strand DNA breaks in rat brain cells exposed to microwave radiation. Mutat Res 596:76-80, 2006. (GT, LE)

This investigation concerns with the effect of low intensity microwave (2.45 and 16.5GHz, SAR 1.0 and 2.01W/kg, respectively) radiation on developing rat brain. Wistar rats (35 days old, male, six rats in each group) were selected for this study. These animals were exposed for 35 days at the above mentioned frequencies separately in two different exposure systems. After the exposure period, the rats were sacrificed and the whole brain tissue was dissected and used for study of single strand DNA breaks by micro gel electrophoresis (comet assay). Single strand DNA breaks were measured as tail length of comet. Fifty cells from each slide and two slides per animal were observed. One-way ANOVA method was adopted for statistical analysis. This study shows that the chronic exposure to these radiations cause statistically significant (p<0.001) increase in DNA single strand breaks in brain cells of rat.

(E) Pesnya DS, Romanovsky AV. Comparison of cytotoxic and genotoxic effects of plutonium-239 alpha particles and mobile phone GSM 900 radiation in the Allium cepa test. Mutat Res. 2012 Oct 8. pii: S1383-5718(12)00291-4. doi: 10.1016/j.mrgentox.2012.08.010. [Epub ahead of print] (GT)

The goal of this study was to compare the cytotoxic and genotoxic effects of plutonium-239 alpha particles and GSM 900 modulated mobile phone radiation in the Allium cepa test. Three groups of bulbs were exposed to mobile phone radiation during 0 (sham), 3 and 9hours. A positive control group was treated during 20 min with plutonium-239 alpha-radiation. Mitotic abnormalities, chromosome aberrations, micronuclei and mitotic index were analyzed. Exposure to alpha-radiation from plutonium-239 and exposure to modulated radiation from mobile phone during 3 and 9h significantly increased the mitotic index. GSM 900 mobile phone radiation as well as alpha-radiation from plutonium-239 induced both clastogenic and aneugenic effects. However, the aneugenic activity of mobile phone radiation was more pronounced. After 9 hours of exposure to mobile phone radiation, polyploid cells, three-groups metaphases, amitoses and some unspecified abnormalities were detected, which were not registered in the other experimental groups. Importantly, GSM 900 mobile phone radiation increased the mitotic index, the frequency of mitotic and chromosome abnormalities, and the micronucleus frequency in a time-dependent manner. Due to its sensitivity, the Allium cepa test can be recommended as a useful cytogenetic assay to assess cytotoxic and genotoxic effects of radiofrequency electromagnetic fields.

(NE) Qutob SS, Chauhan V, Bellier PV, Yauk CL, Douglas GR, Berndt L, Williams A, Gajda GB, Lemay E, Thansandote A, McNamee JP. Microarray gene expression profiling of a human glioblastoma cell line exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field. Radiat Res 165:636-644, 2006. (GE)

The widespread use of mobile phones has led to public concerns about the health effects associated with exposure to radiofrequency (RF) fields. The paramount concern of most persons relates to the potential of these fields to cause cancer. Unlike ionizing radiation, RF fields used for mobile telecommunications (800-1900 MHz) do not possess sufficient energy to directly damage DNA. Most rodent bioassay and in vitro genotoxicity/mutation studies have reported that RF fields at non-thermal levels have no direct mutagenic, genotoxic or carcinogenic effects.

However, some evidence has suggested that RF fields may cause detectable postexposure changes in gene expression. Therefore, the purpose of this study was to assess the ability of exposure to a 1.9 GHz pulse-modulated RF field for 4 h at specific absorption rates (SARs) of 0.1, 1.0 and 10.0 W/kg to affect global gene expression in U87MG glioblastoma cells. We found no evidence that non-thermal RF fields can affect gene expression in cultured U87MG cells relative to the nonirradiated control groups, whereas exposure to heat shock at 43 degrees C for 1 h up-regulated a number of typical stress-responsive genes in the positive control group. Future studies will assess the effect of RF fields on other cell lines and on gene expression in the mouse brain after in vivo exposure.

(E) Remondini D, Nylund R, Reivinen J, Poullotier de Gannes F, Veyret B, Lagroye I, Haro E, Trillo MA, Capri M, Franceschi C, Schlatterer K, Gminski R, Fitzner R, Tauber R, Schuderer J, Kuster N, Leszczynski D, Bersani F, Maercker C. Gene expression changes in human cells after exposure to mobile phone microwaves. *Proteomics* 6:4745-4754, 2006. (GE, CS)

Possible biological effects of mobile phone microwaves were investigated in vitro. In this study, which was part of the 5FP EU project REFLEX (Risk Evaluation of Potential Environmental Hazards From Low-Energy Electromagnetic Field Exposure Using Sensitive in vitro Methods), six human cell types, immortalized cell lines and primary cells, were exposed to 900 and 1800 MHz. RNA was isolated from exposed and sham-exposed cells and labeled for transcriptome analysis on whole-genome cDNA arrays. The results were evaluated statistically using bioinformatics techniques and examined for biological relevance with the help of different databases. NB69 neuroblastoma cells, T lymphocytes, and CHME5 microglial cells did not show significant changes in gene expression. In EA.hy926 endothelial cells, U937 lymphoblastoma cells, and HL-60 leukemia cells we found between 12 and 34 up- or down-regulated genes. Analysis of the affected gene families does not point towards a stress response. However, following microwave exposure, some but not all human cells might react with an increase in expression of genes encoding ribosomal proteins and therefore up-regulating the cellular metabolism.

(NE) Ros-Llor I, Sanchez-Siles M, Camacho-Alonso F, Lopez-Jornet P. Effect of mobile phones on micronucleus frequency in human exfoliated oral mucosal cells. *Oral Dis.* 18:786-792, 2012. (GT)

Objective: In the last two decades, the use of mobile phones has increased enormously all over the world. The controversy regarding whether radiofrequency (RF) fields exert effects upon biological systems is a concern for the general population. An evaluation is made of DNA damage and cytogenetic defects, proliferative potential, and cell death because of RF radiation emitted by mobile phones in healthy young users. Study design: This cohort study was carried out in 50 Caucasian mobile phone users. We collected two cell samples from each subject (a total of 100 cell samples), corresponding to the right and left cheek mucosa, respectively. Case histories and personal information were assessed, including age, gender, body height and weight, history of cancer, smoking and alcohol consumption, exposure to chemical carcinogens or radiation, and dietary habits. Sampling comprised cell collection from both cheeks with a cytobrush, centrifugation, slide preparation, fixation, and staining, followed by fluorescent microscopic analysis. A total of 2000 exfoliated cells were screened for nuclear abnormalities,

especially micronucleus. Results: No statistically significant changes were recorded in relation to age, gender, body mass index, or smoking status. A comparison of the results vs the control area according to the side of the face on which the mobile phone was placed, and in relation to the duration of exposure (years) to mobile phone radiation in the total 100 samples, yielded no significant differences. Conclusions: No genotoxic effects because of RF exposure were observed in relation to any of the study parameters.

(NE) Sakuma N, Komatsubara Y, Takeda H, Hirose H, Sekijima M, Nojima T, Miyakoshi J. DNA strand breaks are not induced in human cells exposed to 2.1425 GHz band CW and W-CDMA modulated radiofrequency fields allocated to mobile radio base stations. Bioelectromagnetics 27:51-57, 2006. (CT)

We conducted a large-scale in vitro study focused on the effects of low level radiofrequency (RF) fields from mobile radio base stations employing the International Mobile Telecommunication 2000 (IMT-2000) cellular system in order to test the hypothesis that modulated RF fields may act as a DNA damaging agent. First, we evaluated the responses of human cells to microwave exposure at a specific absorption rate (SAR) of 80 mW/kg, which corresponds to the limit of the average whole body SAR for general public exposure defined as a basic restriction in the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guidelines. Second, we investigated whether continuous wave (CW) and Wideband Code Division Multiple Access (W-CDMA) modulated signal RF fields at 2.1425 GHz induced different levels of DNA damage. Human glioblastoma A172 cells and normal human IMR-90 fibroblasts from fetal lungs were exposed to mobile communication frequency radiation to investigate whether such exposure produced DNA strand breaks in cell culture. A172 cells were exposed to W-CDMA radiation at SARs of 80, 250, and 800 mW/kg and CW radiation at 80 mW/kg for 2 and 24 h, while IMR-90 cells were exposed to both W-CDMA and CW radiations at a SAR of 80 mW/kg for the same time periods. Under the same RF field exposure conditions, no significant differences in the DNA strand breaks were observed between the test groups exposed to W-CDMA or CW radiation and the sham exposed negative controls, as evaluated immediately after the exposure periods by alkaline comet assays. Our results confirm that low level exposures do not act as a genotoxicant up to a SAR of 800 mW/kg.

(NE) Sakurai T, Kiyokawa T, Narita E, Suzuki Y, Taki M, Miyakoshi J. Analysis of gene expression in a human-derived glial cell line exposed to 2.45 GHz continuous radiofrequency electromagnetic fields. J Radiat Res. 52(2):185-192, 2011. (GE)

The increasing use of mobile phones has aroused public concern regarding the potential health risks of radiofrequency (RF) fields. We investigated the effects of exposure to RF fields (2.45 GHz, continuous wave) at specific absorption rate (SAR) of 1, 5, and 10 W/kg for 1, 4, and 24 h on gene expression in a normal human glial cell line, SVGp12, using DNA microarray. Microarray analysis revealed 23 assigned gene spots and 5 non-assigned gene spots as prospective altered gene spots. Twenty-two genes out of the 23 assigned gene spots were further analyzed by reverse transcription-polymerase chain reaction to validate the results of microarray, and no significant alterations in gene expression were observed. Under the experimental conditions used in this study, we found no evidence that exposure to RF fields affected gene expression in SVGp12 cells.

(NE) Sannino A, Di Costanzo G, Brescia F, Sarti M, Zeni O, Juutilainen J, Scarfi MR. 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). Radiat Res 171:743-751, 2009. (NT, IA)

Abstract Sannino, A., Di Costanzo, G., Brescia, F., Sarti, M., Zeni, O., Juutilainen, J and Scarfi, M. R. 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). Radiat Res 171, 743-751 (2009). The aim of this study was to investigate DNA damage in human dermal fibroblasts from a healthy subject and from a subject affected by Turner's syndrome that were exposed for 24 h to radiofrequency (RF) radiation at 900 MHz. The RF-radiation exposure was carried out alone or in combination with 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX), a well-known environmental mutagen and carcinogen produced during the chlorination of drinking water. Turner's syndrome fibroblasts were also exposed for a shorter time (1 h). A signal similar to that emitted by Global System for Mobile Communications (GSM) mobile phones was used at a specific absorption rate of 1 W/kg under strictly controlled conditions of temperature and dosimetry. To evaluate DNA damage after RF-radiation exposure alone, the alkaline comet assay and the cytokinesis-block micronucleus assay were used. In the combined-exposure experiments, MX was given at a concentration of 25 microM for 1 h immediately after the RF-radiation exposure, and the effects were evaluated by the alkaline comet assay. The results revealed no genotoxic and cytotoxic effects from RF radiation alone in either cell line. As expected, MX treatment induced an increase in DNA migration in the comet assay, but no enhancement of the MX-induced DNA damage was observed in the cells exposed to RF radiation.

(E) Schwarz C, Kratochvil E, Pilger A, Kuster N, Adlkofer F, Rüdiger HW. Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes. Int Arch Occup Environ Health 81:755-767, 2008. (GT, CS)

OBJECTIVE: Universal Mobile Telecommunication System (UMTS) was recently introduced as the third generation mobile communication standard in Europe. This was done without any information on biological effects and genotoxic properties of these particular high-frequency electromagnetic fields. This is disconcerting, because genotoxic effects of the second generation standard Global System for Mobile Communication have been reported after exposure of human cells in vitro. METHODS: Human cultured fibroblasts of three different donors and three different short-term human lymphocyte cultures were exposed to 1,950 MHz UMTS below the specific absorption rate (SAR) safety limit of 2 W/kg. The alkaline comet assay and the micronucleus assay were used to ascertain dose and time-dependent genotoxic effects. Five hundred cells per slide were visually evaluated in the comet assay and comet tail factor (CTF) was calculated. In the micronucleus assay 1,000 binucleated cells were evaluated per assay. The origin of the micronuclei was determined by fluorescence labeled anticentromere antibodies. All evaluations were performed under blinded conditions. RESULTS: UMTS exposure increased the CTF and induced centromere-negative micronuclei (MN) in human cultured fibroblasts in a dose and time-dependent way. Incubation for 24 h at a SAR of 0.05 W/kg generated a statistically significant rise in both CTF and MN (P = 0.02). At a SAR of 0.1 W/kg the CTF was significantly

increased after 8 h of incubation ($P = 0.02$), the number of MN after 12 h ($P = 0.02$). No UMTS effect was obtained with lymphocytes, either unstimulated or stimulated with Phytohemagglutinin. CONCLUSION: UMTS exposure may cause genetic alterations in some but not in all human cells in vitro.

(E) Sekeroğlu V, Akar A, Sekeroğlu ZA. Cytotoxic and genotoxic effects of high-frequency electromagnetic fields (GSM 1800 MHz) on immature and mature rats. Ecotoxicol Environ Saf. 80:140-144, 2012. (LE, GT, DE)

We investigated the cytogenotoxic effects of high frequency electromagnetic fields (HF-EMF) for 45 day and the effect of a recovery period of 15 day after exposure to EMF on bone marrow cells of immature and mature rats. The animals in treatment groups were exposed to 1800 MHz EMF at SAR of 0.37 W/kg and 0.49 W/kg for 2h/day for 45 day. Two recovery groups were kept for a recovery period of 15 day without EMF after exposure to HF-EMF. Two control groups for both immature and mature rats were also included. Significant differences were also observed in chromosome aberrations (CA), micronucleus (MN) frequency, mitotic index (MI) and ratio of polychromatic erythrocytes (PCEs) in all treatment groups. The cytogenotoxic damage was more remarkable in immature rats and, the recovery period did not improve this damage in immature rats. Because much higher and irreversible cytogenotoxic damage was observed in immature rats than in mature rats, further studies are needed to understand effects of EMF on DNA damage and DNA repair, and to determine safe limits for environment and human, especially for children.

(NE) Sekijima M, Takeda H, Yasunaga K, Sakuma N, Hirose H, Nojima T, Miyakoshi J. 2-GHz band CW and W-CDMA modulated radiofrequency fields have no significant effect on cell proliferation and gene expression profile in human cells. J Radiat Res. 51(3):277-284, 2010. (GE)

We investigated the mechanisms by which radiofrequency (RF) fields exert their activity, and the changes in both cell proliferation and the gene expression profile in the human cell lines, A172 (glioblastoma), H4 (neuroglioma), and IMR-90 (fibroblasts from normal fetal lung) following exposure to 2.1425 GHz continuous wave (CW) and Wideband Code Division Multiple Access (W-CDMA) RF fields at three field levels. During the incubation phase, cells were exposed at the specific absorption rates (SARs) of 80, 250, or 800 mW/kg with both CW and W-CDMA RF fields for up to 96 h. Heat shock treatment was used as the positive control. No significant differences in cell growth or viability were observed between any test group exposed to W-CDMA or CW radiation and the sham-exposed negative controls. Using the Affymetrix Human Genome Array, only a very small ($< 1\%$) number of available genes (ca. 16,000 to 19,000) exhibited altered expression in each experiment. The results confirm that low-level exposure to 2.1425 GHz CW and W-CDMA RF fields for up to 96 h did not act as an acute cytotoxicant in either cell proliferation or the gene expression profile. These results suggest that RF exposure up to the limit of whole-body average SAR levels as specified in the ICNIRP guidelines is unlikely to elicit a general stress response in the tested cell lines under these conditions.

(E) Souza LD, Cerqueira ED, Meireles JR. Assessment of nuclear abnormalities in exfoliated cells from the oral epithelium of mobile phone users. Electromagn Biol Med. 2013 May 28. [Epub ahead of print] (GE, HU)

Abstract Transmission and reception of mobile telephony signals take place through electromagnetic wave radiation, or electromagnetic radiofrequency fields, between the mobile terminal and the radio base station. Based on reports in the literature on adverse effects from exposure to this type of radiation, the objective of this study was to evaluate the genotoxic and cytotoxic potential of such exposure, by means of the micronucleus test on exfoliated cells from the oral epithelium. The sample included 45 individuals distributed in 3 groups according to the amount of time in hours per week (t) spent using mobile phones: group I, $t > 5$ h; group II, $t > 1$ h and ≤ 5 h; and group III, $t \leq 1$ h. Cells from the oral mucosa were analyzed to assess the numbers of micronuclei, broken egg structures and degenerative nuclear abnormalities indicative of apoptosis (condensed chromatin, karyorrhexis and pyknosis) or necrosis (karyolysis in addition to these changes). The occurrences of micronuclei and degenerative nuclear abnormalities did not differ between the groups, but the number of broken egg (structures that may be associated with gene amplification) was significantly greater in the individuals in group I ($p < 0.05$).

(NE) Speit G, Schütz P, Hoffmann H. Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in cultured mammalian cells are not independently reproducible. Mutat Res. 626(1-2):42-47, 2007. (GT)

Conflicting results have been published regarding the induction of genotoxic effects by exposure to radiofrequency electromagnetic fields (RF-EMF). Using the comet assay, the micronucleus test and the chromosome aberration test with human fibroblasts (ES1 cells), the EU-funded "REFLEX" project (Risk Evaluation of Potential Environmental Hazards From Low Energy Electromagnetic Field Exposure Using Sensitive in vitro Methods) reported clearly positive effects for various exposure conditions. Because of the ongoing discussion on the biological significance of the effects observed, it was the aim of the present study to independently repeat the results using the same cells, the same equipment and the same exposure conditions. We therefore exposed ES1 cells to RF-EMF (1800 MHz; SAR 2 W/kg, continuous wave with intermittent exposure) for different time periods and then performed the alkaline (pH>13) comet assay and the micronucleus test (MNT). For both tests, clearly negative results were obtained in independently repeated experiments. We also performed these experiments with V79 cells, a sensitive Chinese hamster cell line that is frequently used in genotoxicity testing, and also did not measure any genotoxic effect in the comet assay and the MNT. Appropriate measures of quality control were considered to exclude variations in the test performance, failure of the RF-EMF exposure or an evaluation bias. The reasons for the difference between the results reported by the REFLEX project and our experiments remain unclear.

(NE) Stronati L, Testa A, Moquet J, Edwards A, Cordelli E, Villani P, Marino C, Fresegna AM, Appolloni M, Lloyd D. 935 MHz cellular phone radiation. An in vitro study of genotoxicity in human lymphocytes. Int J Radiat Biol 82:339-346, 2006. (GT, IA)

Purpose: The possibility of genotoxicity of radiofrequency radiation (RFR) applied alone or in combination with x-rays was investigated in vitro using several assays on human lymphocytes. The chosen specific absorption rate (SAR) values are near the upper limit of actual energy absorption in localized tissue when persons use some cellular telephones. The purpose of the combined exposures was to examine whether RFR might act epigenetically by reducing the fidelity of repair of DNA damage caused by a well-characterized and established mutagen. Methods: Blood specimens from 14 donors were exposed continuously for 24 h to a

Global System for Mobile Communications (GSM) basic 935 MHz signal. The signal was applied at two SAR; 1 and 2 W/Kg, alone or combined with a 1-min exposure to 1.0 Gy of 250 kVp x-rays given immediately before or after the RFR. The assays employed were the alkaline comet technique to detect DNA strand breakage, metaphase analyses to detect unstable chromosomal aberrations and sister chromatid exchanges, micronuclei in cytokinesis-blocked binucleate lymphocytes and the nuclear division index to detect alterations in the speed of in vitro cell cycling. Results: By comparison with appropriate sham-exposed and control samples, no effect of RFR alone could be found for any of the assay endpoints. In addition RFR did not modify any measured effects of the x-radiation. Conclusions: This study has used several standard in vitro tests for chromosomal and DNA damage in Go human lymphocytes exposed in vitro to a combination of x-rays and RFR. It has comprehensively examined whether a 24-h continuous exposure to a 935 MHz GSM basic signal delivering SAR of 1 or 2 W/Kg is genotoxic per se or whether, it can influence the genotoxicity of the well-established clastogenic agent; x-radiation. Within the experimental parameters of the study in all instances no effect from the RFR signal was observed.

(E) Sun LX, Yao K, He JL, Lu DQ, Wang KJ, Li HW.[Effect of acute exposure to microwave from mobile phone on DNA damage and repair of cultured human lens epithelial cells in vitro.] *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing ZaZhi.* 24:465-467, 2006. [Article in Chinese] **(GT)**

OBJECTIVE: To investigate the DNA damage of human lens epithelial cells (LECs) caused by acute exposure to low-power 217 Hz modulated 1.8 GHz microwave radiation and DNA repair. **METHODS:** Cultured LECs were exposed to 217 Hz modulated 1.8 GHz microwave radiation at SAR (specific absorption rate) of 0, 1, 2, 3 and 4 W/kg for 2 hours in an sXc-1800 incubator and irradiate system. The DNA single strand breaks were detected with comet assay in sham-irradiated cells and irradiated cells incubated for varying periods: 0, 30, 60, 120 and 240 min after irradiation. Images of comets were digitized and analyzed using an Imagine-pro plus software, and the indexes used in this study were tail length (TL) and tail moment (TM). **RESULTS:** The difference in DNA-breaks between the exposure and sham exposure groups induced by 1 and 2 W/kg irradiation was not significant at every detect time ($P > 0.05$). As for the dosage of 3 and 4 W/kg there was difference in both groups immediately after irradiation ($P < 0.01$). At the time of 30 min after irradiation the difference went on at both group ($P < 0.01$). However, the difference disappeared after one hour's incubation in 3 W/kg group ($P > 0.05$), and existed in 4 W/kg group. **CONCLUSION:** No or repairable DNA damage was observed after 2 hour irradiation of 1.8 GHz microwave on LECs when SAR \leq 3 W/kg. The DNA damages caused by 4 W/kg irradiation were irreversible.

(E) Tiwari R, Lakshmi NK, Surender V, Rajesh AD, Bhargava SC, Ahuja YR. **Combinative exposure effect of radio frequency signals from CDMA mobile phones and aphidicolin on DNA integrity.** *Electromagn Biol Med* 27:418-425, 2008. **(GT, IA)**

The aim of present study is to assess DNA integrity on the effect of exposure to a radio frequency (RF) signal from Code Division Multiple Access (CDMA) mobile phones. Whole blood samples from six healthy male individuals were exposed for RF signals from a CDMA mobile phone for 1 h. Alkaline comet assay was performed to assess the DNA damage. The combinative exposure effect of the RF signals and APC at two concentrations on DNA integrity was studied. DNA repair efficiency of the samples was also studied after 2 h of exposure. The

RF signals and APC (0.2 microg/ml) alone or in synergism did not have any significant DNA damage as compared to sham exposed. However, univariate analysis showed that DNA damage was significantly different among combinative exposure of RF signals and APC at 0.2 microg/ml ($p < 0.05$) and at 2 microg/ml ($p < 0.02$). APC at 2 microg/ml concentration also showed significant damage levels ($p < 0.05$) when compared to sham exposed. DNA repair efficiency also varied in a significant way in combinative exposure sets ($p < 0.05$). From these results, it appears that the repair inhibitor APC enhances DNA breaks at 2 microg/ml concentration and that the damage is possibly repairable. Thus, it can be inferred that the in vitro exposure to RF signals induces reversible DNA damage in synergism with APC.

(E) Tkalec M, Stambuk A, Srut M, Malarić K, Klobučar GI. Oxidative and genotoxic effects of 900MHz electromagnetic fields in the earthworm *Eisenia fetida*. Ecotoxicol Environ Saf. 90:7-12, 2013. (GT, OX, WS)

Accumulating evidence suggests that exposure to radiofrequency electromagnetic field (RF-EMF) can have various biological effects. In this study the oxidative and genotoxic effects were investigated in earthworms *Eisenia fetida* exposed in vivo to RF-EMF at the mobile phone frequency (900MHz). Earthworms were exposed to the homogeneous RF-EMF at field levels of 10, 23, 41 and 120Vm(-1) for a period of 2h using a Gigahertz Transversal Electromagnetic (GTEM) cell. At the field level of 23Vm(-1) the effect of longer exposure (4h) and field modulation (80% AM 1kHz sinusoidal) was investigated as well. All exposure treatments induced significant genotoxic effect in earthworms coelomocytes detected by the Comet assay, demonstrating DNA damaging capacity of 900MHz electromagnetic radiation. Field modulation additionally increased the genotoxic effect. Moreover, our results indicated the induction of antioxidant stress response in terms of enhanced catalase and glutathione reductase activity as a result of the RF-EMF exposure, and demonstrated the generation of lipid and protein oxidative damage. Antioxidant responses and the potential of RF-EMF to induce damage to lipids, proteins and DNA differed depending on the field level applied, modulation of the field and duration of *E. fetida* exposure to 900MHz electromagnetic radiation. Nature of detected DNA lesions and oxidative stress as the mechanism of action for the induction of DNA damage are discussed.

(E) Tomruk A, Guler G, Dincel AS. The influence of 1800 MHz GSM-like signals on hepatic oxidative DNA and lipid damage in nonpregnant, pregnant, and newly born rabbits. Cell Biochem Biophys 56:39-47, 2010. (GT, OX, DE, LE)

The aim of our study is to evaluate the possible biological effects of whole-body 1800 MHz GSM-like radiofrequency (RF) radiation exposure on liver oxidative DNA damage and lipid peroxidation levels in nonpregnant, pregnant New Zealand White rabbits, and in their newly borns. Eighteen nonpregnant and pregnant rabbits were used and randomly divided into four groups which were composed of nine rabbits: (i) Group I (nonpregnant control), (ii) Group II (nonpregnant-RF exposed), (iii) Group III (pregnant control), (iv) Group IV (pregnant-RF exposed). Newborns of the pregnant rabbits were also divided into two groups: (v) Group V (newborns of Group III) and (vi) Group VI (newborns of Group IV). 1800 MHz GSM-like RF radiation whole-body exposure (15 min/day for a week) was applied to Group II and Group IV. No significant differences were found in liver 8 OHdG/10 dG levels of exposure groups (Group II and Group IV) compared to controls (Group I and Group III). However, in Group II and Group IV malondialdehyde (MDA) and ferrous oxidation in xylenol orange (FOX) levels were

increased compared to Group I ($P < 0.05$, Mann-Whitney). No significant differences were found in liver tissue of 8 OHdG/10 dG and MDA levels between Group VI and Group V ($P > 0.05$, Mann-Whitney) while liver FOX levels were found significantly increased in Group VI with respect to Group V ($P < 0.05$, Mann-Whitney). Consequently, the whole-body 1800 MHz GSM-like RF radiation exposure may lead to oxidative destruction as being indicators of subsequent reactions that occur to form oxygen toxicity in tissues.

(E) [Trivino Pardo JC](#), [Grimaldi S](#), [Taranta M](#), [Naldi I](#), [Cinti C](#). Microwave electromagnetic field regulates gene expression in T-lymphoblastoid leukemia CCRF-CEM cell line exposed to 900 MHz. [Electromagn Biol Med](#). 31(1):1-18, 2012. (GE)

Electric, magnetic, and electromagnetic fields are ubiquitous in our society, and concerns have been expressed regarding possible adverse effects of these exposures. Research on Extremely Low-Frequency (ELF) magnetic fields has been performed for more than two decades, and the methodology and quality of studies have improved over time. Studies have consistently shown increased risk for childhood leukemia associated with ELF magnetic fields. There are still inadequate data for other outcomes. More recently, focus has shifted toward Radio Frequencies (RF) exposures from mobile telephony. There are no persuasive data suggesting a health risk, but this research field is still immature with regard to the quantity and quality of available data. This technology is constantly changing and there is a need for continued research on this issue. To investigate whether exposure to high-frequency electromagnetic fields (EMF) could induce adverse health effects, we cultured acute T-lymphoblastoid leukemia cells (CCRF-CEM) in the presence of 900 MHz MW-EMF generated by a transverse electromagnetic (TEM) cell at short and long exposure times. We evaluated the effect of high-frequency EMF on gene expression and we identified functional pathways influenced by 900 MHz MW-EMF exposure.

(E) [Trosić I](#), [Pavčić I](#), [Milković-Kraus S](#), [Mladinić M](#), [Zeljezić D](#). Effect of electromagnetic radiofrequency radiation on the rats' brain, liver and kidney cells measured by comet assay. [Coll Antropol](#) 35:1259-1264, 2011. (GT)

The goal of study was to evaluate DNA damage in rat's renal, liver and brain cells after in vivo exposure to radiofrequency/microwave (Rf/Mw) radiation of cellular phone frequencies range. To determine DNA damage, a single cell gel electrophoresis/comet assay was used. Wistar rats (male, 12 week old, approximate body weight 350 g) ($N = 9$) were exposed to the carrier frequency of 915 MHz with Global System Mobile signal modulation (GSM), power density of 2.4 W/m², whole body average specific absorption rate SAR of 0.6 W/kg. The animals were irradiated for one hour/day, seven days/week during two weeks period. The exposure set-up was Gigahertz Transversal Electromagnetic Mode Cell (GTEM--cell). Sham irradiated controls ($N = 9$) were apart of the study. The body temperature was measured before and after exposure. There were no differences in temperature in between control and treated animals. Comet assay parameters such as the tail length and tail intensity were evaluated. In comparison with tail length in controls (13.5 +/- 0.7 microm), the tail was slightly elongated in brain cells of irradiated animals (14.0 +/- 0.3 microm). The tail length obtained for liver (14.5 +/- 0.3 microm) and kidney (13.9 +/- 0.5 microm) homogenates notably differs in comparison with matched sham controls (13.6 +/- 0.3 microm) and (12.9 +/- 0.9 microm). Differences in tail intensity between control and exposed animals were not significant. 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.

(NE) Valbonesi P, Franzellitti S, Piano A, Contin A, Biondi C, Fabbri E. Evaluation of HSP70 Expression and DNA damage in cells of a human trophoblast cell line exposed to 1.8 GHz amplitude-modulated radiofrequency fields. Radiat Res 169:270-279, 2008. (GT, GE)

The aim of this study was to determine whether high-frequency electromagnetic fields (EMFs) could induce cellular effects. The human trophoblast cell line HTR-8/SVneo was used as a model to evaluate the expression of proteins (HSP70 and HSC70) and genes (HSP70A, B, C and HSC70) of the HSP70 family and the primary DNA damage response after nonthermal exposure to pulse-modulated 1817 MHz sinusoidal waves (GSM-217 Hz; 1 h; SAR of 2 W/kg). HSP70 expression was significantly enhanced by heat, which was applied as the prototypical stimulus. The HSP70A, B and C transcripts were differentially expressed under basal conditions, and they were all significantly induced above basal levels by thermal stress. Conversely, HSC70 protein and gene expression was not influenced by heat. Exposing HTR-8/SVneo cells to high-frequency EMFs did not change either HSP70 or HSC70 protein or gene expression. A significant increase in DNA strand breaks was caused by exposure to HO, which was used as a positive stimulus; however, no effect was observed after exposure of cells to high-frequency EMFs. Overall, no evidence was found that a 1-h exposure to GSM-217 Hz induced a HSP70-mediated stress response or primary DNA damage in HTR-8/SVneo cells. Nevertheless, further investigations on trophoblast cell responses after exposure to GSM signals of different types and durations are needed.

(E) Valbonesi P, Franzellitti S, Bersani F, Contin A, Fabbri E. Effects of the exposure to intermittent 1.8 GHz radio frequency electromagnetic fields on HSP70 expression and MAPK signaling pathways in PC12 cells. Int J Radiat Biol. 2014 Feb 11. [Epub ahead of print] (GE, WS)

Purpose: We previously reported effects on heat shock protein 70 (HSP70) mRNA expression, a cytoprotective protein induced under stressful condition, in human trophoblast cells exposed to amplitude-modulated Global System for Mobile Communication (GSM) signals. In the present work the same experimental conditions were applied to the rat PC12 cells, in order to assess the stress responses mediated by HSP70 and by the Mitogen Activated Protein Kinases (MAPK) in neuronal-like cells, an interesting model to study possible effects of mobile phone frequencies exposure. Materials and methods: HSP70 gene expression level was evaluated by reverse transcriptase polymerase chain reaction, HSP70 protein expression and MAPK phosphorylation were assessed by Western blotting. PC12 cells were exposed for 4, 16 or 24 h to 1.8 GHz continuous wave signal (CW, carrier frequency without modulation) or to two different GSM modulation schemes, GSM-217Hz and GSM-Talk (which generates temporal changes between two different GSM signals, active during talking or listening phases respectively, thus simulating a typical conversation). Specific adsorption rate (SAR) was 2 W/kg. Results: After PC12 cells exposure to the GSM-217Hz signal for 16 or 24 h, HSP70 transcription significantly increased, whereas no effect was observed in cells exposed to the CW or GSM-Talk signals. HSP70 protein expression and three different MAPK signaling pathways were not affected by the exposure to any of the three different 1.8 GHz signals. Conclusion: The positive effect on HSP70 mRNA expression, observed only in cells exposed to the GSM-217Hz signal, is a repeatable response

previously reported in human trophoblast cells and now confirmed in PC12 cells. Further investigations towards a possible role of 1.8 GHz signal modulation are therefore advisable.

(NE) Verschaeve L, Heikkinen P, Verheyen G, Van Gorp U, Boonen F, Vander Plaetse F, Maes A, Kumlin T, Maki-Paakkanen J, Puranen L, Juutilainen J. Investigation of co-genotoxic effects of radiofrequency electromagnetic fields in vivo. Radiat Res 165:598-607, 2006. (GT, LE, IA)

We investigated the possible combined genotoxic effects of radiofrequency (RF) electromagnetic fields (900 MHz, amplitude modulated at 217 Hz, mobile phone signal) with the drinking water mutagen and carcinogen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX). Female rats were exposed to RF fields for a period of 2 years for 2 h per day, 5 days per week at average whole-body specific absorption rates of 0.3 or 0.9 W/kg. MX was given in the drinking water at a concentration of 19 µg/ml. Blood samples were taken at 3, 6 and 24 months of exposure and brain and liver samples were taken at the end of the study (24 months). DNA damage was assessed in all samples using the alkaline comet assay, and micronuclei were determined in erythrocytes. We did not find significant genotoxic activity of MX in blood and liver cells. However, MX induced DNA damage in rat brain. Co-exposures to MX and RF radiation did not significantly increase the response of blood, liver and brain cells compared to MX exposure only. In conclusion, this 2-year animal study involving long-term exposures to RF radiation and MX did not provide any evidence for enhanced genotoxicity in rats exposed to RF radiation.

(NE) Vijayalaxmi. Cytogenetic studies in human blood lymphocytes exposed in vitro to 2.45 GHz or 8.2 GHz radiofrequency radiation. Radiat Res 166, 532–538, 2006. (GT)

Peripheral blood samples collected from healthy human volunteers were exposed in vitro to 2.45 GHz or 8.2 GHz pulsed-wave radiofrequency (RF) radiation. The net forward power, average power density, mean specific absorption rate, and the temperature maintained during the 2-h exposure of the cells to 2.45 GHz or 8.2 GHz were, respectively, 21 W or 60 W, 5 mW/cm² or 10 mW/cm², 2.13 W/kg or 20.71 W/kg, and 36.9 ± 0.1°C or 37.5 ± 0.2°C. Aliquots of the same blood samples that were either sham-exposed or exposed in vitro to an acute dose of 1.5 Gy γ radiation were used as unexposed and positive controls, respectively. Cultured lymphocytes were examined to determine the extent of cytogenetic damage assessed from the incidence of chromosomal aberrations and micronuclei. Under the conditions used to perform the experiments, the levels of damage in RF-radiation-exposed and sham-exposed lymphocytes were not significantly different. Also, there were no significant differences in the response of unstimulated lymphocytes and lymphocytes stimulated with phytohemagglutinin when exposed to 8.2 GHz RF radiation. In contrast, the positive control cells that had been subjected to γ irradiation exhibited significantly more damage than RF-radiation- and sham-exposed lymphocytes.

(NE) Waldmann P, Bohnenberger S, Greinert R, Hermann-Then B, Heselich A, Klug SJ, Koenig J, Kuhr K, Kuster N, Merker M, Murbach M, Pollet D, Schadenboeck W, Scheidemann-Wesp U, Schwab B, Volkmer B, Weyer V, Blettner M. Influence of GSM Signals on Human Peripheral Lymphocytes: Study of Genotoxicity. Radiat Res. 2013 Jan 14. [Epub ahead of print] (GT)

Exposure to radiofrequency (RF) electromagnetic fields (EMF) is continuously increasing worldwide. Yet, conflicting results of a possible genotoxic effect of RF EMF continue to be discussed. In the present study, a possible genotoxic effect of RF EMF (GSM, 1,800 MHz) in human lymphocytes was investigated by a collaboration of six independent institutes (institutes a, b, c, d, e, h). Peripheral blood of 20 healthy, nonsmoking volunteers of two age groups (10 volunteers 16-20 years old and 10 volunteers 50-65 years old) was taken, stimulated and intermittently exposed to three specific absorption rates (SARs) of RF EMF (0.2 W/kg, 2 W/kg, 10 W/kg) and sham for 28 h (institute a). The exposures were performed in a setup with strictly controlled conditions of temperature and dose, and randomly and automatically determined waveguide SARs, which were designed and periodically maintained by ITIS (institute h). Four genotoxicity tests with different end points were conducted (institute a): chromosome aberration test (five types of structural aberrations), micronucleus test, sister chromatid exchange test and the alkaline comet assay (Olive tail moment and % DNA). To demonstrate the validity of the study, positive controls were implemented. The genotoxicity end points were evaluated independently by three laboratories blind to SAR information (institute c = laboratory 1; institute d = laboratory 2; institute e = laboratory 3). Statistical analysis was carried out by institute b. Methods of primary statistical analysis and rules to adjust for multiple testing were specified in a statistical analysis plan based on a data review before unblinding. A linear trend test based on a linear mixed model was used for outcomes of comet assay and exact permutation test for linear trend for all other outcomes. It was ascertained that only outcomes with a significant SAR trend found by at least two of three analyzing laboratories indicated a substantiated suspicion of an exposure effect. On the basis of these specifications, none of the nine end points tested for SAR trend showed a significant and reproducible exposure effect. Highly significant differences between sham exposures and positive controls were detected by each analyzing laboratory, thus validating the study. In conclusion, the results show no evidence of a genotoxic effect induced by RF EMF (GSM, 1,800 MHz).

(E) Wu W, Yao K, Wang KJ, Lu DQ, He JL, Xu LH, Sun WJ. [Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields.]Zhejiang Da XueXueBao Yi Xue Ban 37:34-38, 2008. [Article in Chinese] (GT, IA, OX)

OBJECTIVE: To investigate whether the exposure to the electromagnetic noise can block reactive oxygen species (ROS) production and DNA damage of lens epithelial cells induced by 1800 MHz mobile phone radiation. METHODS: The DCFH-DA method and comet assay were used respectively to detect the intracellular ROS and DNA damage of cultured human lens epithelial cells induced by 4 W/kg 1800 MHz mobile phone radiation or/and 2microT electromagnetic noise for 24 h intermittently. RESULT: 1800 MHz mobile phone radiation at 4 W/kg for 24 h increased intracellular ROS and DNA damage significantly ($P < 0.05$). However, the ROS level and DNA damage of mobile phone radiation plus noise group were not significant enhanced ($P > 0.05$) as compared to sham exposure group. Conclusion: Electromagnetic noise can block intracellular ROS production and DNA damage of human lens epithelial cells induced by 1800 MHz mobile phone radiation.

(E) Xu S, Zhong M, Zhang L, Zhou Z, Zhang W, Wang Y, Wang X, Li M, Chen Y, Chen C, He M, Zhang G, Yu Z. Exposure to 1800 MHz radiofrequency radiation induces

oxidative damage to mitochondrial DNA in primary cultured neurons. Brain Res 1311:189-196. 2010. (GT, OX)

Increasing evidence indicates that oxidative stress may be involved in the adverse effects of radiofrequency (RF) radiation on the brain. Because mitochondrial DNA (mtDNA) defects are closely associated with various nervous system diseases and mtDNA is highly susceptible to oxidative stress, the purpose of this study was to determine whether radiofrequency radiation can cause oxidative damage to mtDNA. 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 24h 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. Consistent 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 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.

(E) Xu S, Chen G, Chen C, Sun C, Zhang D, Murbach M, Kuster N, Zeng Q, Xu Z. Cell Type-Dependent Induction of DNA Damage by 1800 MHz Radiofrequency Electromagnetic Fields Does Not Result in Significant Cellular Dysfunctions. PLoS One. 8(1):e54906, 2013. (GT, CS)

BACKGROUND: Although IARC clarifies radiofrequency electromagnetic fields (RF-EMF) as possible human carcinogen, the debate on its health impact continues due to the inconsistent results. Genotoxic effect has been considered as a golden standard to determine if an environmental factor is a carcinogen, but the currently available data for RF-EMF remain controversial. As an environmental stimulus, the effect of RF-EMF on cellular DNA may be subtle. Therefore, more sensitive method and systematic research strategy are warranted to evaluate its genotoxicity. **OBJECTIVES:** To determine whether RF-EMF does induce DNA damage and if the effect is cell-type dependent by adopting a more sensitive method γ H2AX foci formation; and to investigate the biological consequences if RF-EMF does increase γ H2AX foci formation. **METHODS:** Six different types of cells were intermittently exposed to GSM 1800 MHz RF-EMF at a specific absorption rate of 3.0 W/kg for 1 h or 24 h, then subjected to immunostaining with anti- γ H2AX antibody. The biological consequences in γ H2AX-elevated cell type were further explored with comet and TUNEL assays, flow cytometry, and cell growth assay. **RESULTS:** Exposure to RF-EMF for 24 h significantly induced γ H2AX foci formation in Chinese hamster lung cells and Human skin fibroblasts (HSFs), but not the other cells. However, RF-EMF-elevated γ H2AX foci formation in HSF cells did not result in detectable DNA fragmentation, sustainable cell cycle arrest, cell proliferation or viability change. RF-EMF exposure slightly but not significantly increased the cellular ROS level. **CONCLUSIONS:** RF-EMF induces DNA damage in a cell type-dependent manner, but the elevated γ H2AX foci formation in HSF cells does not result in significant cellular dysfunctions.

(NE) Yadav AS, Sharma MK. Increased frequency of micronucleated exfoliated cells among humans exposed in vivo to mobile telephone radiations. Mutat Res.650(2):175-180, 2008. (LE, GT, HU)

The health concerns have been raised following the enormous increase in the use of wireless mobile telephones throughout the world. This investigation had been taken, with the motive to find out whether mobile phone radiations cause any in vivo effects on the frequency of micronucleated exfoliated cells in the exposed subjects. A total of 109 subjects including 85 regular mobile phone users (exposed) and 24 non-users (controls) had participated in this study. Exfoliated cells were obtained by swabbing the buccal-mucosa from exposed as well as sex-age-matched controls. One thousand exfoliated cells were screened from each individual for nuclear anomalies including micronuclei (MN), karyolysis (KL), karyorrhexis (KH), broken egg (BE) and binucleated (BN) cells. The average daily duration of exposure to mobile phone radiations is 61.26 min with an overall average duration of exposure in term of years is 2.35 years in exposed subjects along with the 9.84 \pm 0.745 micronucleated cells (MNCs) and 10.72 \pm 0.889 total micronuclei (TMN) as compared to zero duration of exposure along with average 3.75 \pm 0.774 MNC and 4.00 \pm 0.808 TMN in controls. The means are significantly different in case of MNC and TMN at 0.01% level of significance. The mean of KL in controls is 13.17 \pm 2.750 and in exposed subjects is 13.06 \pm 1.793. The value of means of KH in exposed subjects (1.84 \pm 0.432) is slightly higher than in controls (1.42 \pm 0.737). Mean frequency of broken egg is found to be more in exposed subjects (0.65 \pm 0.276) as compared to controls (0.50 \pm 0.217). Frequency of presence of more than one nucleus in a cell (binucleated) is also higher in exposed (2.72 \pm 0.374) in comparison to controls (0.67 \pm 0.231). Although there is a slight increase in mean frequency of KH, BE and BN in exposed subjects but the difference is not found statistically significant. Correlation between 0-1, 1-2, 2-3 and 3-4 years of exposure and the frequency of MNC and TMN has been calculated and found to be positively correlated.

(E) Yan JG, Agresti M, Zhang LL, Yan Y, Matloub HS. Upregulation of specific mRNA levels in rat brain after cell phone exposure. Electromagn Biol Med. 27(2):147-154, 2008. (LE, GE)

Adult Sprague-Dawley rats were exposed to regular cell phones for 6 h per day for 126 days (18 weeks). RT-PCR was used to investigate the changes in levels of mRNA synthesis of several injury-associated proteins. Calcium ATPase, Neural Cell Adhesion Molecule, Neural Growth Factor, and Vascular Endothelial Growth Factor were evaluated. The results showed statistically significant mRNA up-regulation of these proteins in the brains of rats exposed to cell phone radiation. These results indicate that relative chronic exposure to cell phone microwave radiation may result in cumulative injuries that could eventually lead to clinically significant neurological damage.

(E) Yao K, Wu W, Wang K, Ni S, Ye P, Yu Y, Ye J, Sun L. Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells. Mol Vis 14:964-969, 2008. (GT, IA, OX)

PURPOSE: The goal of this study was to investigate whether superposing of electromagnetic noise could block or attenuate DNA damage and intracellular reactive oxygen species (ROS) increase of cultured human lens epithelial cells (HLECs) induced by acute exposure to 1.8 GHz

radiofrequency field (RF) of the Global System for Mobile Communications (GSM). METHODS: An sXc-1800 RF exposure system was used to produce a GSM signal at 1.8 GHz (217 Hz amplitude-modulated) with the specific absorption rate (SAR) of 1, 2, 3, and 4 W/kg. After 2 h of intermittent exposure, the ROS level was assessed by the fluorescent probe, 2',7'-dichlorodihydrofluorescein diacetate (DCFH-DA). DNA damage to HLECs was examined by alkaline comet assay and the phosphorylated form of histone variant H2AX (γ H2AX) foci formation assay. RESULTS: After exposure to 1.8 GHz RF for 2 h, HLECs exhibited significant intracellular ROS increase in the 2, 3, and 4 W/kg groups. RF radiation at the SAR of 3 W/kg and 4 W/kg could induce significant DNA damage, examined by alkaline comet assay, which was used to detect mainly single strand breaks (SSBs), while no statistical difference in double strand breaks (DSBs), evaluated by γ H2AX foci, was found between RF exposure (SAR: 3 and 4 W/kg) and sham exposure groups. When RF was superposed with 2 μ T electromagnetic noise could block RF-induced ROS increase and DNA damage. CONCLUSIONS: DNA damage induced by 1.8 GHz radiofrequency field for 2 h, which was mainly SSBs, may be associated with the increased ROS production. Electromagnetic noise could block RF-induced ROS formation and DNA damage.

(NE) [Yildirim MS](#), [Yildirim A](#), [Zamani AG](#), [Okudan N](#). Effect of mobile phone station on micronucleus frequency and chromosomal aberrations in human blood cells. [Genet Couns](#). 21(2):243-251, 2010. (HU, LE, GT)

The use of mobile telephones has rapidly increased worldwide as well as the number of mobile phone base stations that lead to rise low level radiofrequency emissions which may in turn have possible harm for human health. The national radiation protection board has published the known effects of radio waves exposure on humans living close to mobile phone base stations. However, several studies have claimed that the base station has detrimental effects on different tissues. In this study, we aimed to evaluate the effects of mobile phone base stations on the micronucleus (MN) frequency and chromosomal aberrations on blood in people who were living around mobile phone base stations and healthy controls. Frequency of MN and chromosomal aberrations in study and control groups was 8.96 +/- 3.51 and 6.97 +/- 1.52 (p: 0.16); 0.36 +/- 0.31 and 0.75 +/- 0.61 (p: 0.07), respectively. Our results show that there was not a significant difference of MN frequency and chromosomal aberrations between the two study groups. The results claim that cellular phones and their base stations do not produce important carcinogenic changes.

(E) [Zalata, A.](#), [A. Z. El-Samanoudy](#), [D. Shaalan](#), [Y. El-Baiomy](#), and [T. Mostafa](#). In vitro effect of cell phone radiation on motility, DNA fragmentation and clusterin gene expression of sperm. *Int J Fertil Steril*, In Press. Published online ahead of print. (GT, GE, RP)

Background: Use of cellular phones that emits radiofrequency electromagnetic field (RF-EMF) has been increased exponentially and became a part of everyday life. This study aimed to investigate the effects of RF-EMF radiation emitted from cellular phones on sperm motility variables, sperm DNA fragmentation and clusterin (CLU) gene expression. Materials and Methods: 124 semen samples were grouped into; normozoospermia (N, n=26), asthenozoospermia (A, n=32), asthenoteratozoospermia (AT, n=31) and oligoasthenoteratozoospermia (OAT, n=35). Semen samples were divided into two aliquots; samples not exposed to cell phone and samples exposed to cell phone radiation (850 MHz, maximum power < 1 watt; SAR 1.46 W/kg at 10 cm distance) for 1 hr. Before and immediately

after exposure both aliquots were subjected to assessment of sperm motility, acrosin activity, sperm DNA fragmentation and CLU gene expression. Statistical differences were analyzed using paired t-student test for comparisons where $P < 0.05$ was set as significant. Results: There was significant decrease in sperm motility, sperm linear velocity, sperm linearity index, sperm acrosin activity and significant increase in sperm DNA fragmentation percent, CLU gene expression and CLU protein levels in the exposed semen samples to RF-EMF compared with non- exposed samples in OAT > AT > A > N groups ($P < 0.05$).

Conclusions: Cell phone emissions have a negative impact on exposed sperm motility indices, sperm acrosin activity, sperm DNA fragmentation and CLU gene expression especially in OAT cases.

(NE) Zeni O, Schiavoni A, Perrotta A, Forigo D, Deplano M, Scarfi MR. Evaluation of genotoxic effects in human leukocytes after in vitro exposure to 1950 MHz UMTS radiofrequency field. Bioelectromagnetics 29:177-184, 2008. (GT)

In the present study the third generation wireless technology of the Universal Mobile Telecommunication System (UMTS) signal was investigated for the induction of genotoxic effects in human leukocytes. Peripheral blood from six healthy donors was used and, for each donor, intermittent exposures (6 min RF on, 2 h RF off) at the frequency of 1950 MHz were conducted at a specific absorption rate of 2.2 W/kg. The exposures were performed in a transverse electro magnetic (TEM) cell hosted in an incubator under strictly controlled conditions of temperature and dosimetry. Following long duration intermittent RF exposures (from 24 to 68 h) in different stages of the cell cycle, micronucleus formation was evaluated by applying the cytokinesis block micronucleus assay, which also provides information on cell division kinetics. Primary DNA damage (strand breaks/alkali labile sites) was also investigated following 24 h of intermittent RF exposures, by applying the alkaline single cell gel electrophoresis (SCG)/comet assay. Positive controls were included by treating cell cultures with Mitomycin-C and methylmethanesulfonate for micronucleus and comet assays, respectively. The results obtained indicate that intermittent exposures of human lymphocytes in different stages of cell cycle do not induce either an increase in micronucleated cells, or change in cell cycle kinetics; moreover, 24 h intermittent exposures also fail to affect DNA structure of human leukocytes soon after the exposures, likely indicating that repairable DNA damage was not induced.

(E) Zhang DY, Xu ZP, Chiang H, Lu DQ, Zeng QL. [Effects of GSM 1800 MHz radiofrequency electromagnetic fields on DNA damage in Chinese hamster lung cells.] Zhonghua Yu Fang Yi Xue Za Zhi 40:149-152, 2006. [Article in Chinese] (GT)

OBJECTIVE: To study the effects of GSM 1800 MHz radiofrequency electromagnetic fields (RF EMF) on DNA damage in Chinese hamster lung (CHL) cells. METHODS: The cells were intermittently exposed or sham-exposed to GSM 1800 MHz RF EMF (5 minutes on/10 minutes off) at a special absorption rate (SAR) of 3.0 W/kg for 1 hour or 24 hours. Meanwhile, cells exposed to 2-acetaminofluorene, a DNA damage agent, at a final concentration of 20 mg/L for 2 hours were used as positive control. After exposure, cells were fixed by using 4% paraformaldehyde and processed for phosphorylated form of H2AX (gammaH2AX) immunofluorescence measurement. The primary antibody used for immunofluorescence was mouse monoclonal antibody against gammaH2AX and the secondary antibody was fluorescein

isothiocyanate (FITC)-conjugated goat anti-mouse IgG. Nuclei were counterstained with 4, 6-diamidino-2-phenylindole (DAPI). The gammaH2AX foci and nuclei were visualized with an Olympus AX70 fluorescent microscope. Image Pro-Plus software was used to count the gammaH2AX foci in each cell. For each exposure condition, at least 50 cells were selected to detect gammaH2AX foci. Cells were classified as positive when more than five foci were detected. The percentage of gammaH2AX foci positive cells was adopted as the index of DNA damage. RESULTS: The percentage of gammaH2AX foci positive cell of 1800 MHz RF EMF exposure for 24 hours (37.9 +/- 8.6)% or 2-acetylaminofluorene exposure (50.9 +/- 9.4)% was significantly higher compared with the sham-exposure (28.0 +/- 8.4)%. However, there was no significant difference between the sham-exposure and RF EMF exposure for 1 hour (31.8 +/- 8.7)%. CONCLUSION: 1800 MHz RF EMF (SAR, 3.0 W/kg) for 24 hours might induce DNA damage in CHL cells.

(E) Zhang SZ, Yao GD, Lu DQ, Chiang H, Xu ZP. [Effect of 1.8 GHz radiofrequency electromagnetic fields on gene expression of rat neurons]. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 26(8):449-452, 2008. [Article in Chinese] (GE, WS)

OBJECTIVE: To investigate the changes of gene expression in rat neuron induced by 1.8 GHz radiofrequency electromagnetic fields (RF EMF) to screen for RF EMF-responsive genes and the effect of different exposure times and modes on the gene expression in neuron. METHODS: Total RNA was extracted immediately and purified from the primary culture of neurons after intermittent exposed or sham-exposed to a frequency of 1.8 GHz RF EMF for 24 hours at an average special absorption rate (SAR) of 2 W/kg. Affymetrix Rat Neurobiology U34 array was applied to investigate the changes of gene expression in rat neuron. Differentially expressed genes (Egr-1, Mbp and Plp) were further confirmed by semi-quantitative reverse transcription polymerase chain reaction (RT PCR). The expression levels of Egr-1, Mbp and Plp were observed at different exposure times (6, 24 h) and modes (intermittent and continuous exposure). RESULTS: Among 1200 candidate genes, 24 up-regulated and 10 down-regulated genes were found by using Affymetrix microarray suite software 5.0 which are associated with multiple cellular functions (cytoskeleton, signal transduction pathway, metabolism, etc.) after functional classification. Under 24 h and 6 h intermittent exposure, Egr-1 and Plp in experiment groups showed statistic significance ($P < 0.05$) compared with the control groups, while expression of Mbp did not change significantly ($P > 0.05$). After 24 h continuous exposure, Egr-1 and Mbp in experiment groups showed statistic significance ($P < 0.05$) compared with the control group, while expression of Plp did not change significantly ($P > 0.05$). Under the same exposure mode 6 h, expression of all the 3 genes did not change significantly. Different times (6, 24 h) and modes (intermittent and continuous exposure) of exposure exerted remarkable different influences on the expression of Egr-1, Mbp, Plp genes ($P < 0.01$). CONCLUSION: The changes of many genes transcription were involved in the effect of 1.8 GHz RF EMF on rat neurons; Down-regulation of Egr-1 and up-regulation of Mbp, Plp indicated the negative effects of RF EMF on neurons; The effect of RF intermittent exposure on gene expression was more obvious than that of continuous exposure; The effect of 24 h RF exposure (both intermittent and continuous) on gene expression was more obvious than that of 6 h (both intermittent and continuous).

(E) Zhao R, Zhang S, Xu Z, Ju L, Lu D, Yao G. Studying gene expression profile of rat neuron exposed to 1800MHz radiofrequency electromagnetic fields with cDNA microassay. Toxicology 235:167-175, 2007. (GE)

A widespread use of mobile phone (MP) evokes a growing concern for their possible adverse effects on human, especially the brain. Gene expression is a unique way of characterizing how cells and organism adapt to changes in the external environment, so the aim of this investigation was to determine whether 1800 MHz radiofrequency electromagnetic fields (RF EMF) can influence the gene expression of neuron. Affymetrix Rat Neurobiology U34 array was applied to investigate the changes of gene expression in rat neuron after exposed to the pulsed RF EMF at a frequency of 1800 MHz modulated by 217 Hz which is commonly used in MP. Among 1200 candidate genes, 24 up-regulated genes and 10 down-regulated genes were identified after 24-h intermittent exposure at an average special absorption rate (SAR) of 2 W/kg, which are associated with multiple cellular functions (cytoskeleton, signal transduction pathway, metabolism, etc.) after functional classification. The results were further confirmed by quantitative real-time polymerase chain reaction (RT PCR). The present results indicated that the gene expression of rat neuron could be altered by exposure to RF EMF under our experimental conditions.

(E) Zhao TY, Zou SP, Knapp PE. Exposure to cell phone radiation up-regulates apoptosis genes in primary cultures of neurons and astrocytes. Neurosci Lett. 412(1):34-38, 2007. (GE, CS)

The health effects of cell phone radiation exposure are a growing public concern. This study investigated whether expression of genes related to cell death pathways are dysregulated in primary cultured neurons and astrocytes by exposure to a working Global System for Mobile Communication (GSM) cell phone rated at a frequency of 1900MHz. Primary cultures were exposed to cell phone emissions for 2h. We used array analysis and real-time RT-PCR to show up-regulation of caspase-2, caspase-6 and Asc (apoptosis associated speck-like protein containing a card) gene expression in neurons and astrocytes. Up-regulation occurred in both "on" and "stand-by" modes in neurons, but only in "on" mode in astrocytes. Additionally, astrocytes showed up-regulation of the Bax gene. The effects are specific since up-regulation was not seen for other genes associated with apoptosis, such as caspase-9 in either neurons or astrocytes, or Bax in neurons. The results show that even relatively short-term exposure to cell phone radiofrequency emissions can up-regulate elements of apoptotic pathways in cells derived from the brain, and that neurons appear to be more sensitive to this effect than astrocytes.

(E) Zhijian C, Xiaoxue L, Yezhen L, Shijie C, Lifen J, Jianlin L, Deqiang L, Jiliang H. Impact of 1.8-GHz radiofrequency radiation (RFR) on DNA damage and repair induced by doxorubicin in human B-cell lymphoblastoid cells. Mutat Res. 695(1-2):16-21, 2010. (GT, IA)

In the present in vitro study, a comet assay was used to determine whether 1.8-GHz radiofrequency radiation (RFR, SAR of 2W/kg) can influence DNA repair in human B-cell lymphoblastoid cells exposed to doxorubicin (DOX) at the doses of 0microg/ml, 0.05microg/ml, 0.075microg/ml, 0.10microg/ml, 0.15microg/ml and 0.20microg/ml. The combinative exposures to RFR with DOX were divided into five categories. DNA damage was detected at 0h, 6h, 12h, 18h and 24h after exposure to DOX via the comet assay, and the percent of DNA in the tail (% tail DNA) served as the indicator of DNA damage. The results demonstrated that (1) RFR could not directly induce DNA damage of human B-cell lymphoblastoid cells; (2) DOX could significantly induce DNA damage of human B-cell lymphoblastoid cells with the dose-effect

relationship, and there were special repair characteristics of DNA damage induced by DOX; (3) E-E-E type (exposure to RFR for 2h, then simultaneous exposure to RFR and DOX, and exposure to RFR for 6h, 12h, 18h and 24h after exposure to DOX) combinative exposure could obviously influence DNA repair at 6h and 12h after exposure to DOX for four DOX doses (0.075microg/ml, 0.10microg/ml, 0.15microg/ml and 0.20microg/ml) in human B-cell lymphoblastoid cells.

(NE) Zhijian C, Xiaoxue L, Yezhen L, Deqiang L, Shijie C, Lifan J, Jianlin L, Jiliang H. Influence of 1.8-GHz (GSM) radiofrequency radiation (RFR) on DNA damage and repair induced by X-rays in human leukocytes in vitro. Mutat Res. 677(1-2):100-104, 2009. (GT, IA)

In the present study, the in vitro comet assay was used to determine whether 1.8-GHz radiofrequency radiation (RFR) can influence DNA repair in human leukocytes exposed to X-rays. The specific energy absorption rate (SAR) of 2 W/kg (the current European safety limit) was applied. The leukocytes from four young healthy donors were intermittently exposed to RFR for 24 h (fields on for 5 min, fields off for 10 min), and then irradiated with X-rays at doses of 0.25, 0.5, 1.0 and 2.0 Gy. DNA damage to human leukocytes was detected using the comet assay at 0, 15, 45, 90, 150 and 240 min after exposure to X-rays. Using the comet assay, the percent of DNA in the tail (% tail DNA) served as the indicator of DNA damage; the DNA repair percentage (DRP) served as the indicator of the DNA repair speed. The results demonstrated that (1) the DNA repair speeds of human leukocytes after X-ray exposure exhibited individual differences among the four donors; (2) the intermittent exposures of 1.8-GHz RFR at the SAR of 2 W/kg for 24 h did not directly induce DNA damage or exhibit synergistic effects with X-rays on human leukocytes.

(NE) Ziemann C, Brockmeyer H, Reddy SB, Vijayalaxmi, Prihoda TJ, Kuster N, Tillmann T, Dasenbrock C. Absence of genotoxic potential of 902 MHz (GSM) and 1747 MHz (DCS) wireless communication signals: In vivo two-year bioassay in B6C3F1 mice. Int J Radiat Biol. 85(5):454-464, 2009. (GT, LE)

PURPOSE: The aim of the present investigation was to determine the incidence of micronuclei in peripheral blood erythrocytes of B6C3F1 mice that had been chronically exposed to radiofrequencies (RF) used for mobile communication. MATERIALS AND METHODS: 'Ferris wheels' were used to expose tube-restrained male and female mice to simulated environmental RF signals of the Global System for Mobile Communications (GSM, 902 MHz) or Digital Cellular System (DCS, 1747 MHz). RF signals were applied to the mice for 2 hours/day on 5 days/week for two years, at maximal whole-body-averaged specific absorption rates of 0.4, 1.3, and 4.0 W/kg body weight. Concurrent sham-exposed mice, cage controls, and positive controls injected with mitomycin C were included in this investigation. At necropsy, peripheral blood smears were prepared, and coded slides were stained using May-Grunwald-Giemsa or acridine orange. The incidence of micronuclei was recorded for each mouse in 2000 polychromatic and 2000 normochromatic erythrocytes. RESULTS: There were no significant differences in the frequency of micronuclei between RF-exposed, sham-exposed, and cage control mice, irrespective of the staining/counting method used. Micronuclei were, however, significantly increased in polychromatic erythrocytes of the positive control mice.

CONCLUSIONS: In conclusion, the data did not indicate RF-induced genotoxicity in mice after two years of exposure.

APPENDIX B - ABSTRACTS ON GENETIC EFFECTS OF EXTREMELY-LOW FREQUENCY ELECTROMAGNETIC FIELDS (2007-2014)

Below is a key to abbreviations used throughout the following list of abstracts for recent papers published since 2006 and serve as my comments to help the reader quickly identify the significance of each work. The summary sentences by each author are underlined. The list is divided into RF effects papers, and ELF effects papers.

(E- effect observed; NE- no effect observed) (LE- long term exposure; GT- genotoxic effect, e.g., DNA damage, micronucleus formation, chromosome alterations; GE- gene expression; HU- human study; OX- oxidative effects, i.e., involvement of free radicals and oxidative enzymes; IA- interaction with other factors to cause genetic effects; DE- effects on developing animals; RP- reproduction, e.g., sperm damage; EH- compared with electro-hypersensitive subjects; WS- waveform specific effect, e.g., modulation and frequency; CS- cell type specific effect).

(NE) Albert GC, McNamee JP, Marro L, Bellier PV, Prato FS, Thomas AW. Assessment of genetic damage in peripheral blood of human volunteers exposed (whole-body) to a 200 muT, 60 Hz magnetic field. Int J Radiat Biol. 85(2):144-152, 2009. (GT, IA)

AIM: To investigate the extent of damage in nucleated cells in peripheral blood of healthy human volunteers exposed to a whole-body 60 Hz, 200 microT magnetic field. **MATERIALS AND METHODS:** In this study, 10 male and 10 female healthy human volunteers received a 4 h whole-body exposure to a 200 microT, 60 Hz magnetic field. In addition, five males and five females were treated in a similar fashion, but were exposed to sham conditions. For each subject, a blood sample was obtained prior to the exposure period and aliquots were used as negative- (pre-exposure) and positive- [1.5 Gray (Gy) (60)Cobalt ((60)Co) gamma-irradiation] controls. At the end of the 4 h exposure period, a second blood sample was obtained. The extent of DNA damage was assessed in peripheral human blood leukocytes from all samples using the alkaline comet assay. To detect possible clastogenic effects, the incidence of micronuclei was assessed in phytohemagglutinin (PHA)-stimulated lymphocytes using the cytokinesis-block micronucleus assay. **RESULTS:** There was no evidence of either increased DNA damage, as indicated by the alkaline comet assay, or increased incidence of micronuclei (MN) in the magnetic field exposed group. However, an in vitro exposure of 1.5 Gy gamma-irradiation caused a significant increase in both DNA damage and MN induction. **CONCLUSIONS:** This study found no evidence that an acute, whole-body exposure to a 200 microT, 60 Hz magnetic field for 4 hours could cause DNA damage in human blood.

(E) Alcaraz M, Olmos E, Alcaraz-Saura M, Achel DG, Castillo J. Effect of long-term 50 Hz magnetic field exposure on the micronucleated polychromatic erythrocytes of mice. Electromagn Biol Med. 2013 Jun 19. [Epub ahead of print] (GT)

Abstract In recent years extremely low-frequency magnetic fields (ELF-EMF) have become widely used in human activities, leading to an increased chance of exposure to ELF-EMF. There are few reports on in vivo mammalian genotoxic effects using micronucleus (MN) assays, which generally have been used as a short-term screening system. We analyzed the possible genotoxic effect induced by long-term exposure (7, 14, 21, 28 d) of a 50 Hz ELM-MF to mice by measuring the increase in frequency of micronucleated polychromatic erythrocyte in their bone marrow (MNPCEs) and we compared it with that induced by 50 cGy of X-rays. Subsequently, we tried to reduce this chromosomal damage by administering four antioxidant substances with radioprotective capacities: dimethyl sulfoxide (DMSO), 6-n-propyl-2-thiouracil (PTU), grape-procyanidins (P) and citrus flavonoids extract (CE). The increase in micronucleated cells was higher in both physical treatments (Control < ELF-EMF ($p < 0.01$) < X-rays ($p > 0.001$)); however, the antioxidant substances only showed a genoprotective capacity against the damage induced by ionizing radiation (Ci > PTU = DMSO ($p < 0.001$) > P = CE ($p < 0.001$). The 50 Hz ELM-MF increased MNPCEs in mouse bone marrow, expressing a genotoxic capacity. Administration of antioxidant substances with radioprotective capacities known to act through the elimination of free radicals did not diminish the genotoxic effect induced by ELM-MF.

(E) Balamuralikrishnan B, Balachandar V, Kumar SS, Stalin N, Varsha P, Devi SM, Arun M, Manikantan P, Venkatesan C, Sasikala K, Dharwadkar SN. Evaluation of Chromosomal Alteration in Electrical Workers Occupationally Exposed to Low Frequency of Electro Magnetic Field (EMFs) in Coimbatore Population, India. Asian Pac J Cancer Prev. 13(6):2961-2966, 2012. (HU, LE, GT)

Extremely low frequency electromagnetic fields (EMFs) have been classified as possibly carcinogenic to humans by the International Agency for Research on Cancer. An increased number of chromosomal alterations in peripheral lymphocytes are correlated with elevated incidence of cancer. The aim of the present study was to assess occupationally induced chromosomal damage in EMF workers exposed to low levels of radiation. We used conventional metaphase chromosome aberration (CA) analysis and the micronucleus (MN) assay as biological indicators of nonionizing radiation exposure. In the present study totally 70 subjects were selected including 50 exposed and 20 controls. Informed written consent was obtained from all participants and the study was performed in accordance with the Declaration of Helsinki and the approval of the local ethical committee. A higher degree of CA and MN was observed in exposed subjects compared to controls, the frequency of CA being significantly enhanced with long years of exposure ($P < 0.05$). Moreover increase in CA and MN with age was noted in both exposed subjects and controls, but was significantly greater in the former. The results of this study demonstrated that a significant induction of cytogenetic damage in peripheral lymphocytes of workers occupationally exposed to EMFs in electric transformer and distribution stations. In conclusion, our findings suggest that EMFs possess genotoxic capability, as measured by CA and MN assays; CA analysis appeared more sensitive than other cytogenetic end-points. It can be concluded that chronic occupational exposure to EMFs may lead to an increased risk of genetic damage among electrical workers.

(E) Belyaev IY, Hillert L, Protopopova M, Tamm C, Malmgren LO, Persson BR, Selivanova G, Harms-Ringdahl M. 915 MHz microwaves and 50 Hz magnetic field affect chromatin conformation and 53BP1 foci in human lymphocytes from hypersensitive and healthy persons. *Bioelectromagnetics* 26:173-184, 2005. (GT, EH)

We used exposure to microwaves from a global system for mobile communication (GSM) mobile phone (915 MHz, specific absorption rate (SAR) 37 mW/kg) and power frequency magnetic field (50 Hz, 15 μ T peak value) to investigate the response of lymphocytes from healthy subjects and from persons reporting hypersensitivity to electromagnetic field (EMF). The hypersensitive and healthy donors were matched by gender and age and the data were analyzed blind to treatment condition. The changes in chromatin conformation were measured with the method of anomalous viscosity time dependencies (AVTD). 53BP1 protein, which has been shown to colocalize in foci with DNA double strand breaks (DSBs), was analyzed by immunostaining in situ. Exposure at room temperature to either 915 MHz or 50 Hz resulted in significant condensation of chromatin, shown as AVTD changes, which was similar to the effect of heat shock at 41 degrees C. No significant differences in responses between normal and hypersensitive subjects were detected. Neither 915 MHz nor 50 Hz exposure induced 53BP1 foci. On the contrary, a distinct decrease in background level of 53BP1 signaling was observed upon these exposures as well as after heat shock treatments. This decrease correlated with the AVTD data and may indicate decrease in accessibility of 53BP1 to antibodies because of stress-induced chromatin condensation. Apoptosis was determined by morphological changes and by apoptotic fragmentation of DNA as analyzed by pulsed-field gel electrophoresis (PFGE). No apoptosis was induced by exposure to 50 Hz and 915 MHz microwaves. In conclusion, 50 Hz magnetic field and 915 MHz microwaves under specified conditions of exposure induced comparable responses in lymphocytes from healthy and hypersensitive donors that were similar but not identical to stress response induced by heat shock.

(E) [Borhani N](#), [Rajaei F](#), [Salehi Z](#), [Javadi A](#). Analysis of DNA fragmentation in mouse embryos exposed to an extremely low-frequency electromagnetic field. [Electromagn Biol Med](#). 30(4):246-252, 2011. (GT, DE, LE)

Effects of extremely low-frequency electromagnetic fields (ELF-EMFs) on DNA damage in biological systems are still a matter of dispute. The aim of the present study was to investigate the possible effect of electromagnetic field exposure on DNA fragmentation in cells (blastomers) of mouse blastocysts. Eighty female NMRI mice were randomly divided into 2 groups of 40 animals each. The control group was left unexposed whereas the animals in the EMF-group were exposed to a 50-Hz EMF at 0.5 mT 4 h per day, 6 days a week for a duration of 2 weeks. After the 8(th) day of exposure, the female mice in both groups were superovulated (with injections of pregnant mare serum gonadotropin and human chorionic gonadotropin) and then mated overnight. At approximately 4 days after mating (102 h after the human chorionic gonadotropin treatment), blastocysts were obtained by flushing the uterus horns. The mean numbers of pregnant mice, blastocysts after flushing, blastomers within the blastocysts, and the DNA fragmentation index following staining in both groups were compared using statistical methods (SPSS, the Chi-square test, the Student's t-test and the Mann-Whitney U-test, $P < 0.05$). The results showed that the mean number of blastocysts after flushing was significantly decreased in the EMF-group compared to that of the control group ($P < 0.03$). The DNA fragmentation index was significantly increased in the EMF-group compared to control (10.53% vs. 7.14%; $P <$

0.001). However, there was no significant difference in the mean numbers of blastomers and numbers of pregnant mice between the EMF-exposed and control group. Our findings indicate that the EMF exposure in preimplantation stage could have detrimental effects on female mouse fertility and embryo development by decreasing the number of blastocysts and increasing the blastocysts DNA fragmentation.

(E) [Buldak RJ](#), [Polaniak R](#), [Buldak L](#), [Zwirska-Korczala K](#), [Skonieczna M](#), [Monsiol A](#), [Kukla M](#), [Dulawa-Buldak A](#), [Birkner E](#). Short-term exposure to 50 Hz ELF-EMF alters the cisplatin-induced oxidative response in AT478 murine squamous cell carcinoma cells. [Bioelectromagnetics](#). 2012 Apr 25. doi: 10.1002/bem.21732. [Epub ahead of print] (GT, IA, OX)

The aim of this study was to assess the influence of cisplatin and an extremely low frequency electromagnetic field (ELF-EMF) on antioxidant enzyme activity and the lipid peroxidation ratio, as well as the level of DNA damage and reactive oxygen species (ROS) production in AT478 carcinoma cells. Cells were cultured for 24 and 72 h in culture medium with cisplatin. Additionally, the cells were irradiated with 50 Hz/1 mT ELF-EMF for 16 min using a solenoid as a source of the ELF-EMF. The amount of ROS, superoxide dismutase (SOD) isoenzyme activity, glutathione peroxidase (GSH-Px) activity, DNA damage, and malondialdehyde (MDA) levels were assessed. Cells that were exposed to cisplatin exhibited a significant increase in ROS and antioxidant enzyme activity. The addition of ELF-EMF exposure to cisplatin treatment resulted in decreased ROS levels and antioxidant enzyme activity. A significant reduction in MDA concentrations was observed in all of the study groups, with the greatest decrease associated with treatment by both cisplatin and ELF-EMF. Cisplatin induced the most severe DNA damage; however, when cells were also irradiated with ELF-EMF, less DNA damage occurred. Exposure to ELF-EMF alone resulted in an increase in DNA damage compared to control cells. ELF-EMF lessened the effects of oxidative stress and DNA damage that were induced by cisplatin; however, ELF-EMF alone was a mild oxidative stressor and DNA damage inducer. We speculate that ELF-EMF exerts differential effects depending on the exogenous conditions. This information may be of value for appraising the pathophysiological consequences of exposure to ELF-EMF.

(E) Calabrò E, Condello S, Magazù S, Ientile, R. Static and 50 Hz electromagnetic fields effects on human neuronal-like cells vibration bands in the mid-infrared region. *J Electromagnetic Analysis and Applications* 3(2) 69-78, 2011. (GT)

Human neuronal-like cells were exposed to static and 50 Hz electromagnetic fields at the intensities of 2 mT and 1 mT, respectively. The effects of exposure were investigated in the mid-infrared region by means of Fourier self deconvolution spectroscopic analysis. After exposure of 3 hours to static and 50 Hz electromagnetic fields, the vibration bands of CH₂ methylene group increased significantly after both exposures, suggesting a relative increase of lipid related to conformational changes in the cell membrane due to electromagnetic fields. In addition, PO₂- stretching phosphate bands decreased after both exposures, suggesting that alteration in DNA/RNA can be occurred. In particular, exposure of 3 hours to 50 Hz electromagnetic fields produced significant increases in β -sheet contents in amide I, and around the 1740 cm⁻¹ band assigned to non-hydrogen-bonded ester carbonyl stretching mode, that can be

related to unfolding processes of proteins structure and cells death. Further exposure up to 18 hours to static magnetic field produced an increase in β -sheet contents as to α -helix components of amide I region, as well.

(E) [Celikler S](#), [Aydemir N](#), [Vatan O](#), [Kurtuldu S](#), [Bilaloglu R](#). A biomonitoring study of genotoxic risk to workers of transformers and distribution line stations. [Int J Environ Health Res](#). 19(6):421-430, 2009. (GT, HU)

A cytogenetic monitoring study was carried out on a group of workers from transformer and distribution line stations in the Bursa province of Turkey, to investigate the genotoxic risk of occupational exposure to extremely low frequency electric (ELF) and magnetic fields (EMF). Cytogenetic analysis, namely chromosomal aberrations (CAs) and micronucleus (MN) tests were performed on a strictly selected group of 55 workers and compared to 17 controls. CA and MN frequencies in electrical workers appeared significantly higher than in controls ($p < 0.001$, 0.05 , respectively). The frequency of CA in exposed groups were significantly enhanced with the years of exposure ($p < 0.01$). The effect of smoking on the level of CA and MN was not significant in the control and exposure groups. The results of this study demonstrated that a significant induction of cytogenetic damage in peripheral lymphocytes of workers engaged to occupational exposure to ELMF in electric transformer and distribution stations.

(E) [Chen GD](#), [Lu DQ](#), [Jiang H](#), [Xu ZP](#). [Effects of 50 Hz magnetic fields on gene expression in MCF-7 cells]. [Zhejiang Da Xue Xue Bao Yi Xue Ban](#). 37(1):15-22, 2008. [Article in Chinese] (GT, GE)

OBJECTIVE: To investigate whether 50 Hz magnetic fields (MF) can change the gene expression profile in MCF-7 cells and to screen MF responsive genes. **METHODS:** In vitro cultured MCF-7 cells were continuously exposed or sham-exposed to 0.4 mT of 50 Hz MF for 24 hours. Affymetrix Human Genome Genechips (U133A) were applied to analyze gene expression profiles in MF exposed and sham-exposed MCF-7 cells and the data were processed with Genechip data analysis software MAS 5.0 and DMT 3.0. Real-time RT-PCR assay was employed to examine the differentially expressed genes. **RESULT:** Thirty differentially expressed genes were screened with 100 % consistency change calls in the MF exposed MCF-7 cells. Six independent real-time RT-PCR analyses showed that SCNN1A, METTL3 and GPR137B were slightly but statistically significantly changed in MCF-7 cells after exposure to 50 Hz MF ($P < 0.05$), while other analyzed genes exhibited slight up-and down-fluctuations in expressions and no increase or decrease in each gene expression reached statistical significance ($P > 0.05$). **CONCLUSION:** The present study identified three 50 Hz MF responsive genes in MCF-7 cells and the biological consequences of expression changes in these MF responsive genes need to be further investigated. 0.4 mT 50 Hz MF exposure for longer duration might induce DNA double-strand breaks in human lens epithelial cells in vitro.

(NE) [Chen G](#), [Lu D](#), [Chiang H](#), [Leszczynski D](#), [Xu Z](#). Using model organism *Saccharomyces cerevisiae* to evaluate the effects of ELF-MF and RF-EMF exposure on global gene expression. [Bioelectromagnetics](#). 33(7):550-560, 2012. (GE)

The potential health hazard of exposure to electromagnetic fields (EMF) continues to cause public concern. However, the possibility of biological and health effects of exposure to EMF remains controversial and their biophysical mechanisms are unknown. In the present study, we used *Saccharomyces cerevisiae* to identify genes responding to extremely low frequency magnetic fields (ELF-MF) and to radiofrequency EMF (RF-EMF) exposures. The yeast cells were exposed for 6 h to either 0.4 mT 50 Hz ELF-MF or 1800 MHz RF-EMF at a specific absorption rate of 4.7 W/kg. Gene expression was analyzed by microarray screening and confirmed using real-time reverse transcription-polymerase chain reaction (RT-PCR). We were unable to confirm microarray-detected changes in three of the ELF-MF responsive candidate genes using RT-PCR ($P > 0.05$). On the other hand, out of the 40 potential RF-EMF responsive genes, only the expressions of structural maintenance of chromosomes 3 (SMC3) and aquaporin 2 (AQY2 (m)) were confirmed, while three other genes, that is, halotolerance protein 9 (HAL9), yet another kinase 1 (YAK1) and one function-unknown gene (open reading frame: YJL171C), showed opposite changes in expression compared to the microarray data ($P < 0.05$). In conclusion, the results of this study suggest that the yeast cells did not alter gene expression in response to 50 Hz ELF-MF and that the response to RF-EMF is limited to only a very small number of genes. The possible biological consequences of the gene expression changes induced by RF-EMF await further investigation.

(E) Cho S, Lee Y, Lee S, Choi YJ, Chung HW. Enhanced cytotoxic and genotoxic effects of gadolinium following ELF-EMF irradiation in human lymphocytes. Drug Chem Toxicol. 2014 Jan 30. [Epub ahead of print] (GT, IA)

Gadolinium (Gd) and its chelated derivatives are widely utilized for various industrial and medical purposes, particularly as a contrast agent for magnetic resonance imaging (MRI). There are many studies of Gd nephrotoxicity and neurotoxicity, whereas research on cyto- and genotoxicity in normal human lymphocytes is scarce. It is important to investigate the effect of extremely low-frequency electromagnetic fields (ELF-EMF) on Gd toxicity, as patients are co-exposed to Gd and ELF-EMF generated by MRI scanners. We investigated the cytotoxicity and genotoxicity of Gd and the possible enhancing effect of ELF-EMF on Gd toxicity in cultured human lymphocytes by performing a micronuclei (MN) assay, trypan blue dye exclusion, single cell gel electrophoresis, and apoptosis analyses using flow cytometry. Isolated lymphocytes were exposed to 0.2-1.2 mM of Gd only or in combination with a 60-Hz ELF-EMF of 0.8-mT field strength. Exposing human lymphocytes to Gd resulted in a concentration- and time-dependent decrease in cell viability and an increase in MN frequency, single strand DNA breakage, apoptotic cell death, and ROS production. ELF-EMF (0.8 mT) exposure also increased cell death, MN frequency, olive tail moment, and apoptosis induced by Gd treatment alone. These results suggest that Gd induces DNA damage and apoptotic cell death in human lymphocytes and that ELF-EMF enhances the cytotoxicity and genotoxicity of Gd.

(E) Cho YH, Jeon HK, Chung HW. Effects of extremely low-frequency electromagnetic fields on delayed chromosomal instability induced by bleomycin in normal human fibroblast cells. J Toxicol Environ Health A. 70(15-16):1252-1258, 2007. (GT, IA)

This study was carried out to examine the interaction of extremely low-frequency electromagnetic fields (ELF-EMF) on delayed chromosomal instability by bleomycin (BLM) in

human fibroblast cells. A micronucleus-centromere assay using DNA probes for chromosomes 1 and 4 was performed and a 60-Hz ELF-EMF of 0.8 mT field strength was applied either alone or with BLM throughout the culture period. The frequencies of micronuclei (MN) and aneuploidy were analyzed at 28, 88, and 240 h after treatment with BLM. The coexposure of cells to BLM and ELF-EMF led to a significant increase in the frequencies of MN and aneuploidy compared to the cells treated with BLM alone. No difference was observed between field-exposed and sham-exposed control cells. The frequency of MN induced by BLM was increased at 28 h, and further analysis showed a persistent increase up to 240 h, but the new levels were not significantly different from the level at 28 h. BLM increased the frequencies of aneuploidy at 28, 88, and 240 h, and significantly higher frequency of aneuploidy was observed in the cells analyzed at 240 h compared to the cells examined at 28 h. No interaction of ELF-EMF on delayed chromosomal instability by BLM was observed. Our results suggest that ELF-EMF enhances the cytotoxicity of BLM. BLM might induce delayed chromosomal instability, but no effect of ELF-EMF was observed on the BLM-induced delayed chromosomal instability in fibroblast cells.

(E) Collard JF, Lazar C, Nowé A, Hinsenkamp M. Statistical validation of the acceleration of the differentiation at the expense of the proliferation in human epidermal cells exposed to extremely low frequency electric fields. *Prog Biophys Mol Biol.* 111(1):37-45, 2013. (GE)

An acceleration of differentiation at the expense of proliferation is observed in our previous publications and in the literature after exposure of various biological models to low frequency and low-amplitude electric and electromagnetic fields. This observation is related with a significant modification of genes expression. We observed and compared over time this modification. This study use microarray data obtained on epidermis cultures harvested from human abdominoplasty exposed to ELF electric fields. This protocol is repeated with samples collected on three different healthy patients. The sampling over time allows comparison of the effect of the stimulus at a given time with the evolution of control group. After 4 days, we observed a significant difference of the genes expression between control (D4C) and stimulated (D4S) ($p < 0.05$). On the control between day 4 and 7, we observed another group of genes with significant difference ($p < 0.05$) in their expression. We identify the common genes between these two groups and we select from them those expressing no difference between stimulate at 4 days (D4S) and control after 7 days (D7C). The same analysis was performed with D4S-D4C-D12C and D7S-D7C-D12C. The lists of genes which follow this pattern show acceleration in their expressions under stimulation appearing on control at a later time. In this list, genes such as DKK1, SPRR3, NDRG4, and CHEK1 are involved in cell proliferation or differentiation. Numerous other genes are also playing a function in mitosis, cell cycle or in the DNA replication transcription and translation.

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(E) Cuccurazzu B, Leone L, Podda MV, Piacentini R, Riccardi E, Ripoli C, Azzena GB, Grassi C. Exposure to extremely low-frequency (50 Hz) electromagnetic fields enhances adult hippocampal neurogenesis in C57BL/6 mice. *Exp Neurol.* 226(1):173-182, 2010. (LE, GE, DE)

Throughout life, new neurons are continuously generated in the hippocampus, which is therefore a major site of structural plasticity in the adult brain. We recently demonstrated that extremely low-frequency electromagnetic fields (ELFEFs) promote the neuronal differentiation of neural stem cells in vitro by up-regulating Ca(v)1-channel activity. The aim of the present study was to determine whether 50-Hz/1 mT ELFEF stimulation also affects adult hippocampal neurogenesis in vivo, and if so, to identify the molecular mechanisms underlying this action and its functional impact on synaptic plasticity. ELFEF exposure (1 to 7 h/day for 7 days) significantly enhanced neurogenesis in the dentate gyrus (DG) of adult mice, as documented by increased numbers of cells double-labeled for 5-bromo-deoxyuridine (BrdU) and double cortin. Quantitative RT-PCR analysis of hippocampal extracts revealed significant ELFEF exposure-induced increases in the transcription of pro-neuronal genes (Mash1, NeuroD2, Hes1) and genes encoding Ca(v)1.2 channel α (1C) subunits. Increased expression of NeuroD1, NeuroD2 and Ca(v)1 channels was also documented by Western blot analysis. Immunofluorescence experiments showed that, 30 days after ELFEF stimulation, roughly half of the newly generated immature neurons had survived and become mature dentate granule cells (as shown by their immunoreactivity for both BrdU and NeuN) and were integrated into the granule cell layer of the DG. Electrophysiological experiments demonstrated that the new mature neurons influenced hippocampal synaptic plasticity, as reflected by increased long-term potentiation. Our findings show that ELFEF exposure can be an effective tool for increasing in vivo neurogenesis, and they could lead to the development of novel therapeutic approaches in regenerative medicine.

(E) [Di Campli E](#), [Di Bartolomeo S](#), [Grande R](#), [Di Giulio M](#), [Cellini L](#). Effects of extremely low-frequency electromagnetic fields on *Helicobacter pylori* biofilm. [Curr Microbiol](#). **60(6):412-418, 2010. (GE)**

The aim of this work was to investigate the effects of exposure to extremely low-frequency electromagnetic fields (ELF-EMF) both on biofilm formation and on mature biofilm of *Helicobacter pylori*. Bacterial cultures and 2-day-old biofilm of *H. pylori* ATCC 43629 were exposed to ELF-EMF (50 Hz frequency-1 mT intensity) for 2 days to assess their effect on the cell adhesion and on the mature biofilm detachment, respectively. All the exposed cultures and the respective sham exposed controls were studied for: the cell viability status, the cell morphological analysis, the biofilm mass measurement, the genotypic profile, and the luxS and amiA gene expression. The ELF-EMF acted on the bacterial population during the biofilm formation displaying significant differences in cell viability, as well as, in morphotypes measured by the prevalence of spiral forms (58.41%) in respect to the controls (33.14%), whereas, on mature biofilm, no significant differences were found when compared to the controls. The measurement of biofilm cell mass was significantly reduced in exposed cultures in both examined experimental conditions. No changes in DNA patterns were recorded, whereas a modulation in amiA gene expression was detected. An exposure to ELF-EMF of *H. pylori* biofilm induces phenotypic changes on adhering bacteria and decreases the cell adhesion unbalancing the bacterial population therefore reducing the *H. pylori* capability to protect itself.

(E) [Dominici L](#), [Villarini M](#), [Fatigoni C](#), [Monarca S](#), [Moretti M](#). Genotoxic hazard evaluation in welders occupationally exposed to extremely low-frequency magnetic fields (ELF-MF). [Int J Hyg Environ Health](#). **215(1):68-75, 2011. (GT, HU)**

Electric arc welding is known to involve considerable exposure to extremely low-frequency magnetic fields (ELF-MF). A cytogenetic monitoring study was carried out in a group of welders to investigate the genotoxic risk of occupational exposure to ELF-MF. This study assessed individual occupational exposure to ELF-MF using a personal magnetic-field dosimeter, and the cytogenetic effects were examined by comparing micronuclei (MN) and sister chromatid exchange (SCE) frequencies in the lymphocytes of the exposed workers with those of non-exposed control subjects (blood donors) matched for age and smoking habit. Cytogenetic analyses were carried out on 21 workers enrolled from two different welding companies in Central Italy and compared to 21 controls. Some differences between the groups were observed on analysis of SCE and MN, whereas replication indices in the exposed were found not to differ from the controls. In particular, the exposed group showed a significantly higher frequency of MN (group mean \pm SEM: 6.10 \pm 0.39) compared to the control group (4.45 \pm 0.30). Moreover, the increase in MN is associated with a proportional increase in ELF-MF exposure levels with a dose-response relationship. A significant decrease in SCE frequency was observed in exposed subjects (3.73 \pm 0.21) compared to controls (4.89 \pm 0.12). The hypothesis of a correlation between genotoxic assays and ELF-MF exposure value was partially supported, especially as regards MN assay. Since these results are derived from a small-scale pilot study, a larger scale study should be undertaken.

(E) [Du XG, Xu SS, Chen Q, Lu DQ, Xu ZP, Zeng QL.](#) [Effects of 50 Hz magnetic fields on DNA double-strand breaks in human lens epithelial cells]. [Zhejiang Da Xue Xue Bao Yi Xue Ban.](#) 37(1):9-14, 2008. [Article in Chinese] **(GT)**

OBJECTIVE: To investigate the effects of 50 Hz magnetic fields (MF) on DNA double-strand breaks in human lens epithelial cells (hLECs). **METHODS:** The cultured human lens epithelial cells were exposed to 0.4 mT 50 Hz MF for 2 h, 6 h, 12 h, 24 h and 48 h. Cells exposed to 4-nitroquinoline-1-oxide, a DNA damage agent, at a final concentration of 0.1 micromol/L for 1 h were used as positive controls. After exposure, cells were fixed with 4 % paraformaldehyde and for H2AX (gamma H2AX) immunofluorescence measurement. gamma H2AX foci were detected at least 200 cells for each sample. Cells were classified as positive when more than three foci per cell were observed. Mean values of foci per cell and percentage of foci positive cells were adopted as indexes of DNA double-strand breaks. **RESULT:** The mean value of foci per cell and the percentage of gamma H2AX foci positive cells in 50 Hz MF exposure group for 24 h were (2.93 +/-0.43) and (27.88 +/-2.59)%, respectively, which were significantly higher than those of sham-exposure group [(1.77 +/-0.37) and (19.38+/-2.70)%, P <0.05], and the mean value of foci per cell and the percentage of gamma H2AX foci positive cells in 50 Hz MF exposure group for 48 h were (3.14 +/-0.35) and (31.00 +/-3.44)%, which were significantly higher than those of sham-exposure group (P <0.01). However there was no significant difference between 50 Hz MF exposure groups for 2 h, 6 h, 12 h and sham-exposure group for above two indexes (P >0.05). **CONCLUSION:** 0.4 mT 50 Hz MF exposure for longer duration might induce DNA double-strand breaks in human lens epithelial cells in vitro.

(E) [El-Bialy NS, Rageh MM.](#) Extremely low-frequency magnetic field enhances the therapeutic efficacy of low-dose cisplatin in the treatment of Ehrlich carcinoma. [Biomed Res Int.](#) 2013;2013:189352. doi: 10.1155/2013/189352. Epub 2013 Jan 14. **(GT, IA)**

The present study examines the therapeutic efficacy of the administration of low-dose cisplatin (cis) followed by exposure to extremely low-frequency magnetic field (ELF-MF), with an average intensity of 10 mT, on Ehrlich carcinoma in vivo. The cytotoxic and genotoxic actions of this combination were studied using comet assay, mitotic index (MI), and the induction of micronucleus (MN). Moreover, the inhibition of tumor growth was also measured. Treatment with cisplatin and ELF-MF (group A) increased the number of damaged cells by 54% compared with 41% for mice treated with cisplatin alone (group B), 20% for mice treated by exposure to ELF-MF (group C), and 9% for the control group (group D). Also the mitotic index decreased significantly for all treated groups ($P < 0.001$). The decrement percent for the treated groups (A, B, and C) were 70%, 65%, and 22%, respectively, compared with the control group (D). Additionally, the rate of tumor growth at day 12 was suppressed significantly ($P < 0.001$) for groups A, B, and C with respect to group (D). These results suggest that ELF-MF enhanced the cytotoxic activity of cisplatin and potentiate the benefit of using a combination of low-dose cisplatin and ELF-MF in the treatment of Ehrlich carcinoma.

(E) Erdal N, Gürgül S, Celik A. Cytogenetic effects of extremely low frequency magnetic field on Wistar rat bone marrow. [Mutat Res.](#) 630(1-2):69-77, 2007. (GT, LE)

In this study, the genotoxic and cytotoxic potential of extremely low frequency magnetic fields (ELF-MF) was investigated in Wistar rat tibial bone marrow cells, using the chromosomal aberration (CA) and micronucleus (MN) test systems. In addition to these test systems, we also investigated the mitotic index (MI), and the ratio of polychromatic erythrocytes (PCEs) to normochromatic erythrocytes (NCEs). Wistar rats were exposed to acute (1 day for 4h) and long-term (4h/day for 45 days) to a horizontal 50Hz, 1mT uniform magnetic field generated by a Helmholtz coil system. Mitomycin C (MMC, 2mg/kg BW) was used as positive control. Results obtained by chromosome analysis do not show any statistically significant differences between the negative control and both acute and long-term ELF-MF exposed samples. When comparing the group mean CA of long-term exposure with the negative control and acute exposure, the group mean of the long-term exposed group was higher, but this was not statistically significant. However, the mean micronucleus frequency of the longer-term exposed group was considerably higher than the negative control and acutely exposed groups. This difference was statistically significant ($p < 0.01$). The results of the MI in bone marrow showed that the averages of both A-MF and L-MF groups significantly decreased when compared to those in the negative control ($p < 0.001$ and $p < 0.01$, respectively). No significant differences were found between the group mean MI of A-MF exposure with L-MF. We found that the average of PCEs/NCEs ratios of A-MF exposed group was significantly lower than the negative control and L-MF exposed groups ($p < 0.001$ and $p < 0.01$, respectively). In addition, the group mean of the PCEs/NCEs ratios of L-MF was significantly lower than negative control ($p < 0.01$). We also found that the MMC treated group showed higher the number of CA and the frequency of MN formation when compared to those in all other each groups (p -values of all each groups < 0.01) and also MMC treated group showed lower MI and the PCEs/NCEs ratios when compared to those in all other each groups (p -values of all groups < 0.01). These observations indicate the in vivo susceptibility of mammals to the genotoxicity potential of ELF-MF.

(E) Fedrowitz M, Löscher W. Gene expression in the mammary gland tissue of female Fischer 344 and Lewis rats after magnetic field exposure (50 Hz, 100 μ T) for 2 weeks. [Int J](#)

[Radiat Biol.](#) 88(5):425-429, 2012. **(GE, LE)** See also: Fedrowitz [M](#), [Hass R](#), [Löscher W](#). Effects of 50 Hz magnetic field exposure on the stress marker α -amylase in the rat mammary gland. [Int J Radiat Biol.](#) 88(7):556-564, 2012.

PURPOSE: The issue of whether exposure to environmental power-frequency magnetic fields (MF) has impact on breast cancer development still remains equivocal. Previously, we observed rat strain differences in the MF response of breast tissue, so that the genetic background plays a role in MF effects. The present experiment aimed to elucidate candidate genes involved in MF effects by comparison of MF-susceptible Fischer 344 (F344) rats and MF-insensitive Lewis rats. **MATERIALS AND METHODS:** Female F344 and Lewis rats were exposed to MF (50 Hz, 100 μ T) for two weeks, and a whole genome microarray analysis in the mammary gland tissue was performed. **RESULTS:** A remarkably decreased α -amylase gene expression, decreases in carbonic anhydrase 6 and lactoperoxidase, both relevant for pH regulation, and an increased gene expression of cystatin E/M, a tumor suppressor, were observed in MF-exposed F344, but not in Lewis rats. **CONCLUSION:** The MF-exposed F344 breast tissue showed alterations in gene expression, which were absent in Lewis and may therefore be involved in the MF-susceptibility of F344. Notably α -amylase might serve as a promising target to study MF effects, because first experiments indicate that MF exposure alters the functionality of this enzyme in breast tissue.

(E) [Focke F](#), [Schuermann D](#), [Kuster N](#), [Schär P](#). DNA fragmentation in human fibroblasts under extremely low frequency electromagnetic field exposure. [Mutat Res.](#) 683(1-2):74-83, 2010. **(GT)**

Extremely low frequency electromagnetic fields (ELF-EMFs) were reported to affect DNA integrity in human cells with evidence based on the Comet assay. These findings were heavily debated for two main reasons; the lack of reproducibility, and the absence of a plausible scientific rationale for how EMFs could damage DNA. Starting out from a replication of the relevant experiments, we performed this study to clarify the existence and explore origin and nature of ELF-EMF induced DNA effects. Our data confirm that intermittent (but not continuous) exposure of human primary fibroblasts to a 50 Hz EMF at a flux density of 1 mT induces a slight but significant increase of DNA fragmentation in the Comet assay, and we provide first evidence for this to be caused by the magnetic rather than the electric field. Moreover, we show that EMF-induced responses in the Comet assay are dependent on cell proliferation, suggesting that processes of DNA replication rather than the DNA itself may be affected. Consistently, the Comet effects correlated with a reduction of actively replicating cells and a concomitant increase of apoptotic cells in exposed cultures, whereas a combined Fpg-Comet test failed to produce evidence for a notable contribution of oxidative DNA base damage. Hence, ELF-EMF induced effects in the Comet assay are reproducible under specific conditions and can be explained by minor disturbances in S-phase processes and occasional triggering of apoptosis rather than by the generation of DNA damage.

(E) [Frisch P](#), [Li GC](#), [McLeod K](#), [Laramee CB](#). Induction of heat shock gene expression in RAT1 primary fibroblast cells by ELF electric fields. [Bioelectromagnetics.](#) 34(5):405-413, 2013. **(GE)**

Recent studies have demonstrated that the Ku70 gene fragment can be placed in the anti-sense orientation under the control of a heat-inducible heat shock protein 70 (HSP70) promoter and activated through heat shock exposure. This results in attenuation of the Ku70 protein expression, inhibiting cellular repair processes, and sensitizing the transfected cells to exposures such as the ionizing radiation exposures used clinically. However, achieving the tissue temperatures necessary to thermally induce the HSP70 response presents significant limitations to the clinical application of this strategy. Previous findings suggest an alternative approach to inducing a heat shock response, specifically through the use of extremely low frequency (ELF) electrical field stimulation. To further pursue this approach, we investigated HSP70 responses in transfected rat primary fibroblast (RAT1) cells exposed to 10 Hz electric fields at intensities of 20-500 V/m. We confirmed that low frequency electric fields can induce HSP70 heat shock expression, with peak responses obtained at 8 h following a 2 h field exposure. However, the approximate threefold increase in expression is substantially lower than that obtained using thermal stimulation, raising questions of the clinical utility of the response.

(E) [Giorgi G](#), [Marcantonio P](#), [Bersani F](#), [Gavoci E](#), [Del Re B](#). Effect of extremely low frequency magnetic field exposure on DNA transposition in relation to frequency, wave shape and exposure time. [Int J Radiat Biol](#). 87(6):601-608, 2011. (GT, WS)

PURPOSE: To examine the effect of extremely low frequency magnetic field (ELF-MF) exposure on transposon (Tn) mobility in relation to the exposure time, the frequency and the wave shape of the field applied. **MATERIALS AND METHODS:** Two Escherichia coli model systems were used: (1) Cells unable to express β -galactosidase (LacZ(-)), containing a mini-transposon Tn10 element able to give ability to express β -galactosidase (LacZ(+)) upon its transposition; therefore in these cells transposition activity can be evaluated by analysing LacZ(+) clones; (2) cells carrying Fertility plasmid (F(+)), and a Tn5 element located on the chromosome; therefore in these cells transposition activity can be estimated by a bacterial conjugation assay. Cells were exposed to sinusoidal (SiMF) or pulsed-square wave (PMF) magnetic fields of various frequencies (20, 50, 75 Hz) and for different exposure times (15 and 90 min). **RESULTS:** Both mini-Tn10 and Tn5 transposition decreased under SiMF and increased under PMF, as compared to sham exposure control. No significant difference was found between frequencies and between exposure times. **CONCLUSIONS:** ELF-MF exposure affects transposition activity and the effects critically depend on the wave shape of the field, but not on the frequency and the exposure time, at least in the range observed.

(E) [Heredia-Rojas JA](#), [Rodríguez de la Fuente AO](#), [Alcocer González JM](#), [Rodríguez-Flores LE](#), [Rodríguez-Padilla C](#), [Santovo-Stephano MA](#), [Castañeda-Garza E](#), [Taméz-Guerra RS](#). Effect of 60 Hz magnetic fields on the activation of hsp70 promoter in cultured INER-37 and RMA E7 cells. [In Vitro Cell Dev Biol Anim](#). 46(9):758-63, 2010. (GE)

It has been reported that 50-60 Hz magnetic fields (MF) with flux densities ranging from microtesla to millitesla are able to induce heat shock factor or heat shock proteins in various cells. In this study, we investigated the effect of 60 Hz sinusoidal MF at 8 and 80 μ T on the expression of the luciferase gene contained in a plasmid labeled as electromagnetic field-plasmid (pEMF). This gene construct contains the specific sequences previously described for the

induction of hsp70 expression by MF, as well as the reporter for the luciferase gene. The pEMF vector was transfected into INER-37 and RMA E7 cell lines that were later exposed to either MF or thermal shock (TS). Cells that received the MF or TS treatments and their controls were processed according to the luciferase assay system for evaluate luciferase activity. An increased luciferase gene expression was observed in INER-37 cells exposed to MF and TS compared with controls ($p < 0.05$), but MF exposure had no effect on the RMA E7 cell line.

(NE) [Huwiler SG](#), [Beyer C](#), [Fröhlich J](#), [Hennecke H](#), [Egli T](#), [Schürmann D](#), [Rehrauer H](#), [Fischer HM](#). Genome-wide transcription analysis of *Escherichia coli* in response to extremely low-frequency magnetic fields. [Bioelectromagnetics](#). 2012 Feb 13. doi: 10.1002/bem.21709. [Epub ahead of print] (GE)

The widespread use of electricity raises the question of whether or not 50 Hz (power line frequency in Europe) magnetic fields (MFs) affect organisms. We investigated the transcription of *Escherichia coli* K-12 MG1655 in response to extremely low-frequency (ELF) MFs. Fields generated by three signal types (sinusoidal continuous, sinusoidal intermittent, and power line intermittent; all at 50 Hz, 1 mT) were applied and gene expression was monitored at the transcript level using an Affymetrix whole-genome microarray. Bacterial cells were grown continuously in a chemostat (dilution rate $D = 0.4 \text{ h}^{-1}$) fed with glucose-limited minimal medium and exposed to 50 Hz MFs with a homogenous flux density of 1 mT. For all three types of MFs investigated, neither bacterial growth (determined using optical density) nor culturable counts were affected. Likewise, no statistically significant change (fold-change > 2 , $P \leq 0.01$) in the expression of 4,358 genes and 714 intergenic regions represented on the gene chip was detected after MF exposure for 2.5 h (1.4 generations) or 15 h (8.7 generations). Moreover, short-term exposure (8 min) to the sinusoidal continuous and power line intermittent signal neither affected bacterial growth nor showed evidence for reliable changes in transcription. In conclusion, our experiments did not indicate that the different tested MFs (50 Hz, 1 mT) affected the transcription of *E. coli*.

(NE) [Jin YB](#), [Kang GY](#), [Lee JS](#), [Choi JI](#), [Lee JW](#), [Hong SC](#), [Myung SH](#), [Lee YS](#). Effects on micronuclei formation of 60-Hz electromagnetic field exposure with ionizing radiation, hydrogen peroxide, or c-Myc overexpression. [Int J Radiat Biol](#). 88(4):374-380, 2012. (GT, IA)

PURPOSE: Epidemiological studies have demonstrated a possible correlation between exposure to extremely low-frequency magnetic fields (ELF-MF) and cancer. However, this correlation has yet to be definitively confirmed by epidemiological studies. The principal objective of this study was to assess the effects of 60 Hz magnetic fields in a normal cell line system, and particularly in combination with various external factors, via micronucleus (MN) assays. **MATERIALS AND METHODS:** Mouse embryonic fibroblast NIH3T3 cells and human lung fibroblast WI-38 cells were exposed for 4 h to a 60 Hz, 1 mT uniform magnetic field with or without ionizing radiation (IR, 2 Gy), H_2O_2 (100 μM) and cellular myelocytomatosis oncogene (c-Myc) activation. **RESULTS:** The results obtained showed no significant differences between the cells exposed to ELF-MF alone and the unexposed cells. Moreover, no synergistic effects were observed when ELF-MF was combined with IR, H_2O_2 , and c-Myc

activation. **CONCLUSIONS:** Our results demonstrate that ELF-MF did not enhance MN frequency by IR, H₂O₂ and c-Myc activation.

(NE) Jin YB, Choi SH, Lee JS, Kim JK, Lee JW, Hong SC, Myung SH, Lee YS. Absence of DNA damage after 60-Hz electromagnetic field exposure combined with ionizing radiation, hydrogen peroxide, or c-Myc overexpression. Radiat Environ Biophys. 2013 Dec 5. [Epub ahead of print] (GT, IA)

The principal objective of this study was to assess the DNA damage in a normal cell line system after exposure to 60 Hz of extremely low frequency magnetic field (ELF-MF) and particularly in combination with various external factors, via comet assays. NIH3T3 mouse fibroblast cells, WI-38 human lung fibroblast cells, L132 human lung epithelial cells, and MCF10A human mammary gland epithelial cells were exposed for 4 or 16 h to a 60-Hz, 1 mT uniform magnetic field in the presence or absence of ionizing radiation (IR, 1 Gy), H₂O₂ (50 μM), or c-Myc oncogenic activation. The results obtained showed no significant differences between the cells exposed to ELF-MF alone and the unexposed cells. Moreover, no synergistic or additive effects were observed after 4 or 16 h of pre-exposure to 1 mT ELF-MF or simultaneous exposure to ELF-MF combined with IR, H₂O₂ or c-Myc activation.

(E) Jouni FJ, Abdolmaleki P, Ghanati F. Oxidative stress in broad bean (Vicia faba L.) induced by static magnetic field under natural radioactivity. Mutat Res. 741(1-2):116-121, 2012. (LE, GT, OX, IA)

The investigation was performed to evaluate the influence of the static magnetic field on oxidative stress in Vicia faba cultivated in soil from high background natural radioactivity in Iran. Soil samples were collected from Ramsar, Iran where the annual radiation absorbed dose from background radiation is substantially higher than 20 mSv/year. The soil samples were then divided into 2 separate groups including high and low natural radioactivity. The plants were continuously exposed to static magnetic field of 15 mT for 8 days, each 8h/day. The results showed that in the plants cultivated in soils with high background natural radioactivity and low background natural radioactivity the activity of antioxidant enzymes as well as flavonoid content were lower than those of the control. Treatment of plants with static magnetic field showed similar results in terms of lowering of antioxidant defense system and increase of peroxidation of membrane lipids. Accumulation of ROS also resulted in chromosomal aberration and DNA damage. This phenomenon was more pronounced when a combination of natural radiation and treatment with static magnetic field was applied. The results suggest that exposure to static magnetic field causes accumulation of reactive oxygen species in V. faba and natural radioactivity of soil exaggerates oxidative stress.

(E) Kim J, Ha CS, Lee HJ, Song K. Repetitive exposure to a 60-Hz time-varying magnetic field induces DNA double-strand breaks and apoptosis in human cells. Biochem Biophys Res Commun. 400(4):739-744, 2010. (GT)

We investigated the effects of extremely low frequency time-varying magnetic fields (MFs) on human normal and cancer cells. Whereas a single exposure to a 60-Hz time-varying MF of 6 mT for 30min showed no effect, repetitive exposure decreased cell viability. This decrease was

accompanied by phosphorylation of γ -H2AX, a common DNA double-strand break (DSB) marker, and checkpoint kinase 2 (Chk2), which is critical to the DNA damage checkpoint pathway. In addition, repetitive exposure to a time-varying MF of 6 mT for 30 min every 24 h for 3 days led to p38 activation and induction of apoptosis in cancer and normal cells. Therefore, these results demonstrate that repetitive exposure to MF with extremely low frequency can induce DNA DSBs and apoptosis through p38 activation. These results also suggest the need for further evaluation of the effects of repetitive exposure to environmental time-varying MFs on human health.

(E) Kim J, Yoon Y, Yun S, Park GS, Lee HJ, Song K. Time-varying magnetic fields of 60 Hz at 7 mT induce DNA double-strand breaks and activate DNA damage checkpoints without apoptosis. Bioelectromagnetics, 33(5):383-393, 2012. (GT, WS)

The potential genotoxic effect of a time-varying magnetic field (MF) on human cells was investigated. Upon continuous exposure of human primary fibroblast and cervical cancer cells to a 60 Hz MF at 7 mT for 10-60 min, no significant change in cell viability was observed. However, deoxyribonucleic acid (DNA) double-strand breaks (DSBs) were detected, and the DNA damage checkpoint pathway was activated in these cells without programmed cell death (called apoptosis). The exposure of human cells to a 60 Hz MF did not induce intracellular reactive oxygen species (ROS) production, suggesting that the observed DNA DSBs are not directly caused by ROS. We also compared the position and time dependency of DNA DSBs with numerical simulation of MFs. The Lorentz force and eddy currents in these experiments were numerically calculated to investigate the influence of each factor on DNA DSBs. The DNA DSBs mainly occurred at the central region, where the MF was strongest, after a 30-min exposure. After 90 min, however, the amount of DNA DSBs increased rapidly in the outer regions, where the eddy current and Lorentz force were strong.

(NE) Kirschenlohr H, Ellis P, Hesketh R, Metcalfe J. Gene Expression Profiles in White Blood Cells of Volunteers Exposed to a 50 Hz Electromagnetic Field. Radiat Res. 178(3): 138-149, 2012. (GE, HU)

Consistent and independently replicated laboratory evidence to support a causative relationship between environmental exposure to extremely low-frequency electromagnetic fields (EMFs) at power line frequencies and the associated increase in risk of childhood leukemia has not been obtained. In particular, although gene expression responses have been reported in a wide variety of cells, none has emerged as robust, widely replicated effects. DNA microarrays facilitate comprehensive searches for changes in gene expression without a requirement to select candidate responsive genes. To determine if gene expression changes occur in white blood cells of volunteers exposed to an ELF-EMF, each of 17 pairs of male volunteers age 20-30 was subjected either to a 50 Hz EMF exposure of $62.0 \pm 7.1 \mu\text{T}$ for 2 h or to a sham exposure ($0.21 \pm 0.05 \mu\text{T}$) at the same time (11:00 a.m. to 13:00 p.m.). The alternative regime for each volunteer was repeated on the following day and the two-day sequence was repeated 6 days later, with the exception that a null exposure ($0.085 \pm 0.01 \mu\text{T}$) replaced the sham exposure. Five blood samples (10 ml) were collected at 2 h intervals from 9:00 to 17:00 with five additional samples during the exposure and sham or null exposure periods on each study day. RNA samples were pooled for the same time on each study day for the group of 17 volunteers that were subjected to the

ELF-EMF exposure/sham or null exposure sequence and were analyzed on Illumina microarrays. Time courses for 16 mammalian genes previously reported to be responsive to ELF-EMF exposure, including immediate early genes, stress response, cell proliferation and apoptotic genes were examined in detail. No genes or gene sets showed consistent response profiles to repeated ELF-EMF exposures. A stress response was detected as a transient increase in plasma cortisol at the onset of either exposure or sham exposure on the first study day. The cortisol response diminished progressively on subsequent exposures or sham exposures, and was attributable to mild stress associated with the experimental protocol.

(E) [Koyama S](#), [Sakurai T](#), [Nakahara T](#), [Miyakoshi J](#). Extremely low frequency (ELF) magnetic fields enhance chemically induced formation of apurinic/aprimidinic (AP) sites in A172 cells. [Int J Radiat Biol.](#) 84(1):53-59, 2008. (GT, IA)

PURPOSE: To detect the effects of extremely low frequency (ELF) magnetic fields, the number of apurinic/aprimidinic (AP) sites in human glioma A172 cells was measured following exposure to ELF magnetic fields. **MATERIALS AND METHODS:** The cells were exposed to an ELF magnetic field alone, to genotoxic agents (methyl methane sulfonate (MMS) and hydrogen peroxide (H₂O₂)) alone, or to an ELF magnetic field with the genotoxic agents. After exposure, DNA was extracted, and the number of AP sites was measured. **RESULTS:** There was no difference in the number of AP sites between cells exposed to an ELF magnetic field and sham controls. With MMS or H₂O₂ alone, the number of AP sites increased with longer treatment times. Exposure to an ELF magnetic field in combination with the genotoxic agents increased AP-site levels compared with the genotoxic agents alone. **CONCLUSIONS:** Our results suggest that the number of AP sites induced by MMS or H₂O₂ is enhanced by exposure to ELF magnetic fields at 5 millitesla (mT). This may occur because such exposure can enhance the activity or lengthen the lifetime of radical pairs.

(E) [Lee JW](#), [Kim MS](#), [Kim YJ](#), [Choi YJ](#), [Lee Y](#), [Chung HW](#). Genotoxic effects of 3 T magnetic resonance imaging in cultured human lymphocytes. [Bioelectromagnetics.](#) 32(7):535-542, 2011. (GT)

The clinical and preclinical use of high-field intensity (HF, 3 T and above) magnetic resonance imaging (MRI) scanners have significantly increased in the past few years. However, potential health risks are implied in the MRI and especially HF MRI environment due to high-static magnetic fields, fast gradient magnetic fields, and strong radiofrequency electromagnetic fields. In this study, the genotoxic potential of 3 T clinical MRI scans in cultured human lymphocytes in vitro was investigated by analyzing chromosome aberrations (CA), micronuclei (MN), and single-cell gel electrophoresis. Human lymphocytes were exposed to electromagnetic fields generated during MRI scanning (clinical routine brain examination protocols: three-channel head coil) for 22, 45, 67, and 89 min. We observed a significant increase in the frequency of single-strand DNA breaks following exposure to a 3 T MRI. In addition, the frequency of both CAs and MN in exposed cells increased in a time-dependent manner. The frequencies of MN in lymphocytes exposed to complex electromagnetic fields for 0, 22, 45, 67, and 89 min were 9.67, 11.67, 14.67, 18.00, and 20.33 per 1000 cells, respectively. Similarly, the frequencies of CAs in lymphocytes exposed for 0, 45, 67, and 89 min were 1.33, 2.33, 3.67, and 4.67 per 200 cells,

respectively. These results suggest that exposure to 3 T MRI induces genotoxic effects in human lymphocytes.

(E) Leone L, Fusco S, Mastrodonato A, Piacentini R, Barbati SA, Zaffina S, Pani G, Podda MV, Grassi C. Epigenetic Modulation of Adult Hippocampal Neurogenesis by Extremely Low-Frequency Electromagnetic Fields. Mol Neurobiol. 2014 Feb 16. [Epub ahead of print] (GE)

Throughout life, adult neurogenesis generates new neurons in the dentate gyrus of hippocampus that have a critical role in memory formation. Strategies able to stimulate this endogenous process have raised considerable interest because of their potential use to treat neurological disorders entailing cognitive impairment. We previously reported that mice exposed to extremely low-frequency electromagnetic fields (ELFEFs) showed increased hippocampal neurogenesis. Here, we demonstrate that the ELFEF-dependent enhancement of hippocampal neurogenesis improves spatial learning and memory. To gain insights on the molecular mechanisms underlying ELFEFs' effects, we extended our studies to an in vitro model of neural stem cells (NSCs) isolated from the hippocampi of newborn mice. We found that ELFEFs enhanced proliferation and neuronal differentiation of hippocampal NSCs by regulation of epigenetic mechanisms leading to pro-neuronal gene expression. Upon ELFEF stimulation of NSCs, we observed a significant enhancement of expression of the pro-proliferative gene hairy enhancer of split 1 and the neuronal determination genes NeuroD1 and Neurogenin1. These events were preceded by increased acetylation of H3K9 and binding of the phosphorylated transcription factor cAMP response element-binding protein (CREB) on the regulatory sequence of these genes. Such ELFEF-dependent epigenetic modifications were prevented by the Ca_v1-channel blocker nifedipine, and were associated with increased occupancy of CREB-binding protein (CBP) to the same loci within the analyzed promoters. Our results unravel the molecular mechanisms underlying the ELFEFs' ability to improve endogenous neurogenesis, pointing to histone acetylation-related chromatin remodeling as a critical determinant. These findings could pave the way to the development of novel therapeutic approaches in regenerative medicine.

(E) Li SS, Zhang ZY, Yang CJ, Lian HY, Cai P. Gene expression and reproductive abilities of male *Drosophila melanogaster* subjected to ELF-EMF exposure. Mutat Res. 758(1-2):95-103, 2013. (GE, LE, RP)

Extremely low frequency electromagnetic field (ELF-EMF) exposure is attracting increased attention as a possible disease-inducing factor. The in vivo effects of short-term and long-term ELF-EMF exposure on male *Drosophila melanogaster* were studied using transcriptomic analysis for preliminary screening and QRT-PCR for further verification. Transcriptomic analysis indicated that 439 genes were up-regulated and 874 genes were down-regulated following short-term exposures and that 514 genes were up-regulated and 1206 genes were down-regulated following long-term exposures (expression >2- or <0.5-fold, respectively). In addition, there are 238 up-regulated genes and 598 down-regulated genes in the intersection of short-term and long-term exposure (expression >2- or <0.5-fold). The DEGs (differentially expressed genes) in *D. melanogaster* following short-term exposures were involved in metabolic processes, cytoskeletal organization, mitotic spindle organization, cell death, protein modification and proteolysis. Long-term exposure led to changes in expression of genes involved in metabolic

processes, response to stress, mitotic spindle organization, aging, cell death and cellular respiration. In the intersection of short-term and long-term exposure, a series of DEGs were related to apoptosis, aging, immunological stress and reproduction. To check the ELF-EMF effects on reproduction, some experiments on male reproduction ability were performed. Their results indicated that short-term ELF-EMF exposure may decrease the reproductive ability of males, but long-term exposures had no effect on reproductive ability. Down-regulation of ark gene in the exposed males suggests that the decrease in reproductive capacity may be induced by the effects of ELF-EMF exposure on spermatogenesis through the caspase pathway. QRT-PCR analysis confirmed that jra, ark and decay genes were down regulated in males exposed for 1 Generation (1G) and 72 h, which suggests that apoptosis may be inhibited in vivo. ELF-EMF exposure may have accelerated cell senescence, as suggested by the down-regulation of both cat and jra genes and the up-regulation of hsp22 gene. Up-regulation of totA and hsp22 genes during exposure suggests that exposed flies might induce an in vivo immune response to counter the adverse effects encountered during ELF-EMF exposure. Down-regulation of cat genes suggests that the partial oxidative protection system might be restrained, especially during short-term exposures. This study demonstrates the bioeffects of ELF-EMF exposure and provides evidence for understanding the in vivo mechanisms of ELF-EMF exposure on male *D. melanogaster*.

(E) [Lupke M](#), [Frahm J](#), [Lantow M](#), [Maercker C](#), [Remondini D](#), [Bersani F](#), [Simkó M](#). Gene expression analysis of ELF-MF exposed human monocytes indicating the involvement of the alternative activation pathway. [Biochim Biophys Acta](#). 1763(4):402-12, 2006. (GE)

This study focused on the cell activating capacity of extremely low frequency magnetic fields (ELF-MF) on human umbilical cord blood-derived monocytes. Our results confirm the previous findings of cell activating capacity of ELF-MF (1.0 mT) in human monocytes, which was detected as an increased ROS release. Furthermore, gene expression profiling (whole-genome cDNA array Human Unigene RZPD-2) was performed to achieve a comprehensive view of involved genes during the cell activation process after 45 min ELF-MF exposure. Our results indicate the alteration of 986 genes involved in metabolism, cellular physiological processes, signal transduction and immune response. Significant regulations could be analyzed for 5 genes (expression >2- or <0.5-fold): IL15RA (Interleukin 15 receptor, alpha chain), EPS15R (Epidermal growth factor receptor pathway substrate 15 - like 1), DNMT3A (Hypothetical protein MGC16121), DNMT3A (DNA (cytosine-5) methyltransferase 3 alpha), and one gene with no match to known genes, DKFZP586J1624. Real-time RT-PCR analysis of the kinetic of the expression of IL15RA, and IL10RA during 45 min ELF-MF exposure indicates the regulation of cell activation via the alternative pathway, whereas the delayed gene expression of FOS, IL2RA and the melatonin synthesizing enzyme HIOMT suggests the suppression of inflammatory processes. Accordingly, we suggest that ELF-MF activates human monocytes via the alternative pathway.

(E) [Luukkonen J](#), [Liimatainen A](#), [Höytö A](#), [Juutilainen J](#), [Naarala J](#). Pre-exposure to 50 Hz magnetic fields modifies menadione-induced genotoxic effects in human SH-SY5Y neuroblastoma cells. [PLoS One](#). 2011 Mar 23;6(3):e18021. (GT, IA)

BACKGROUND: Extremely low frequency (ELF) magnetic fields (MF) are generated by power lines and various electric appliances. They have been classified as possibly carcinogenic

by the International Agency for Research on Cancer, but a mechanistic explanation for carcinogenic effects is lacking. A previous study in our laboratory showed that pre-exposure to ELF MF altered cancer-relevant cellular responses (cell cycle arrest, apoptosis) to menadione-induced DNA damage, but it did not include endpoints measuring actual genetic damage. In the present study, we examined whether pre-exposure to ELF MF affects chemically induced DNA damage level, DNA repair rate, or micronucleus frequency in human SH-SY5Y neuroblastoma cells. **METHODOLOGY/PRINCIPAL FINDINGS:** Exposure to 50 Hz MF was conducted at 100 μ T for 24 hours, followed by chemical exposure for 3 hours. The chemicals used for inducing DNA damage and subsequent micronucleus formation were menadione and methyl methanesulphonate (MMS). Pre-treatment with MF enhanced menadione-induced DNA damage, DNA repair rate, and micronucleus formation in human SH-SY5Y neuroblastoma cells. Although the results with MMS indicated similar effects, the differences were not statistically significant. No effects were observed after MF exposure alone. **CONCLUSIONS:** The results confirm our previous findings showing that pre-exposure to MFs as low as 100 μ T alters cellular responses to menadione, and show that increased genotoxicity results from such interaction. The present findings also indicate that complementary data at several chronological points may be critical for understanding the MF effects on DNA damage, repair, and post-repair integrity of the genome.

(E) Luukkonen J, Liimatainen A, Juutilainen J, Naarala J. Induction of genomic instability, oxidative processes, and mitochondrial activity by 50Hz magnetic fields in human SH-SY5Y neuroblastoma cells. Mutat Res. 760:33-41, 2014. (GT, OX, IA)

Epidemiological studies have suggested that exposure to 50Hz magnetic fields (MF) increases the risk of childhood leukemia, but there is no mechanistic explanation for carcinogenic effects. In two previous studies we have observed that a 24-h pre-exposure to MF alters cellular responses to menadione-induced DNA damage. The aim of this study was to investigate the cellular changes that must occur already during the first 24h of exposure to MF, and to explore whether the MF-induced changes in DNA damage response can lead to genomic instability in the progeny of the exposed cells. In order to answer these questions, human SH-SY5Y neuroblastoma cells were exposed to a 50-Hz, 100- μ T MF for 24h, followed by 3-h exposure to menadione. The main finding was that MF exposure was associated with increased level of micronuclei, used as an indicator of induced genomic instability, at 8 and 15d after the exposures. Other delayed effects in MF-exposed cells included increased mitochondrial activity at 8d, and increased reactive oxygen species (ROS) production and lipid peroxidation at 15d after the exposures. Oxidative processes (ROS production, reduced glutathione level, and mitochondrial superoxide level) were affected by MF immediately after the exposure. In conclusion, the present results suggest that MF exposure disturbs oxidative balance immediately after the exposure, which might explain our previous findings on MF altered cellular responses to menadione-induced DNA damage. Persistently elevated levels of micronuclei were found in the progeny of MF-exposed cells, indicating induction of genomic instability.

(E) Ma Q, Deng P, Zhu G, Liu C, Zhang L, Zhou Z, Luo X, Li M, Zhong M, Yu Z, Chen C, Zhang Y. Extremely low-frequency electromagnetic fields affect transcript levels of

neuronal differentiation-related genes in embryonic neural stem cells. PLoS One. 2014 Mar 3;9(3):e90041. doi: 10.1371/journal.pone.0090041. eCollection 2014. (GE)

Previous studies have reported that extremely low-frequency electromagnetic fields (ELF-EMF) can affect the processes of brain development, but the underlying mechanism is largely unknown. The proliferation and differentiation of embryonic neural stem cells (eNSCs) is essential for brain development during the gestation period. To date, there is no report about the effects of ELF-EMF on eNSCs. In this paper, we studied the effects of ELF-EMF on the proliferation and differentiation of eNSCs. Primary cultured eNSCs were treated with 50 Hz ELF-EMF; various magnetic intensities and exposure times were applied. Our data showed that there was no significant change in cell proliferation, which was evaluated by cell viability (CCK-8 assay), DNA synthesis (Edu incorporation), average diameter of neurospheres, cell cycle distribution (flow cytometry) and transcript levels of cell cycle related genes (P53, P21 and GADD45 detected by real-time PCR). When eNSCs were induced to differentiation, real-time PCR results showed a down-regulation of Sox2 and up-regulation of Math1, Math3, Ngn1 and Tuj1 mRNA levels after 50 Hz ELF-EMF exposure (2 mT for 3 days), but the percentages of neurons (Tuj1 positive cells) and astrocytes (GFAP positive cells) were not altered when detected by immunofluorescence assay. Although cell proliferation and the percentages of neurons and astrocytes differentiated from eNSCs were not affected by 50 Hz ELF-EMF, the expression of genes regulating neuronal differentiation was altered. In conclusion, our results support that 50 Hz ELF-EMF induce molecular changes during eNSCs differentiation, which might be compensated by post-transcriptional mechanisms to support cellular homeostasis.

(E) [Mairs RJ](#), [Hughes K](#), [Fitzsimmons S](#), [Prise KM](#), [Livingstone A](#), [Wilson L](#), [Baig N](#), [Clark AM](#), [Timpson A](#), [Patel G](#), [Folkard M](#), [Angerson WJ](#), [Boyd M](#). Microsatellite analysis for determination of the mutagenicity of extremely low-frequency electromagnetic fields and ionising radiation in vitro. [Mutat Res.](#) 626(1-2):34-41, 2007. (GT, IA)

Extremely low-frequency electromagnetic fields (ELF-EMF) have been reported to induce lesions in DNA and to enhance the mutagenicity of ionising radiation. However, the significance of these findings is uncertain because the determination of the carcinogenic potential of EMFs has largely been based on investigations of large chromosomal aberrations. Using a more sensitive method of detecting DNA damage involving microsatellite sequences, we observed that exposure of UVW human glioma cells to ELF-EMF alone at a field strength of 1 mT (50 Hz) for 12 h gave rise to 0.011 mutations/locus/cell. This was equivalent to a 3.75-fold increase in mutation induction compared with unexposed controls. Furthermore, ELF-EMF increased the mutagenic capacity of 0.3 and 3 Gy gamma-irradiation by factors of 2.6 and 2.75, respectively. These results suggest not only that ELF-EMF is mutagenic as a single agent but also that it can potentiate the mutagenicity of ionising radiation. Treatment with 0.3 Gy induced more than 10 times more mutations per unit dose than irradiation with 3 Gy, indicating hypermutability at low dose.

(E) [Mariucci G](#), [Villarini M](#), [Moretti M](#), [Taha E](#), [Conte C](#), [Minelli A](#), [Aristei C](#), [Ambrosini MV](#). Brain DNA damage and 70-kDa heat shock protein expression in CD1 mice exposed to extremely low frequency magnetic fields. [Int J Radiat Biol.](#) 86(8):701-710, 2010. (GT, LE)

PURPOSE: The question of whether exposure to extremely low frequency magnetic fields (ELF-MF), may contribute to cerebral cancer and neurodegeneration is of current interest. In this study we investigated whether exposure to ELF-MF (50 Hz-1 mT) harms cerebral DNA and induces expression of 70-kDa heat shock protein (hsp70). **MATERIALS AND METHODS:** CD1 mice were exposed to a MF (50 Hz-1 mT) for 1 or 7 days (15 h/day) and sacrificed either at the end of exposure or after 24 h. Unexposed and sham-exposed mice were used as controls. Mouse brains were dissected into cerebral cortex-striatum, hippocampus and cerebellum to evaluate primary DNA damage and hsp70 gene expression. Food intake, weight gain, and motor activity were also evaluated. **RESULTS:** An increase in primary DNA damage was detected in all cerebral areas of the exposed mice sacrificed at the end of exposure, as compared to controls. DNA damage, as can be evaluated by the comet assay, appeared to be repaired in mice sacrificed 24 h after a 7-day exposure. Neither a short (15 h) nor long (7 days) MF-exposure induced hsp70 expression, metabolic and behavioural changes. **CONCLUSIONS:** These results indicate that in vivo ELF-MF induce reversible brain DNA damage while they do not elicit the stress response.

(E) [Markkanen A](#), [Juutilainen J](#), [Naarala J](#). Pre-exposure to 50 Hz magnetic fields modifies menadione-induced DNA damage response in murine L929 cells. [Int J Radiat Biol.](#) 84(9):742-751, 2008. (IA)

PURPOSE: Effects on DNA damage response were investigated in murine L929 cells exposed to 50 Hz magnetic fields (MF) with or without ultraviolet B (UVB, wavelength 280-320 nm) radiation or menadione (MQ). **MATERIALS AND METHODS:** Cells were exposed to MF at 100 or 300 microT combined with MQ (150 microM, 1 hour) or UVB radiation (160 J/m²) using various exposure schedules. The samples were stained with propidium iodide (PI) and analysed by flow cytometer for cell cycle stages. Apoptotic cells were defined as sub G(1) events. **RESULTS:** In cells first exposed to 100 microT MF for 24 h, the response to subsequent MQ treatment was significantly altered so that the proportion of sub G(1) cells was decreased and the proportion of cells in the G(2)/M phase was increased. When a 300 microT MF was used, also the proportion of cells in the G(1) phase was decreased. MF exposures after MQ treatment did not alter responses to MQ. No effects were found from MF exposure alone or from MF combined with UVB radiation. **CONCLUSIONS:** The results strengthen previous findings suggesting that pre-exposure to MF can alter cellular responses to other agents, and indicate that MF as low as 100 microT has measurable impacts on cancer-relevant cellular processes such as DNA-damage.

(NE) Mizuno K, Narita E, Yamada M, Shinohara N, Miyakoshi J. ELF magnetic fields do not affect cell survival and DNA damage induced by ultraviolet B. [Bioelectromagnetics.](#) 35(2):108-115, 2014. (GT, IA)

We investigated whether extremely low frequency (ELF) magnetic field exposure has modification effects on cell survival after ultraviolet B (UV-B) irradiation and on repair process of DNA damage induced by UV-B irradiation in WI38VA13 subcloned 2RA and XP2OS(SV) cells. The ELF magnetic field exposure was conducted using a Helmholtz coil-based system that was designed to generate a sinusoidal magnetic field at 5 mT and 60 Hz. Cell survival was assessed by WST assay after UV-B irradiation at 20-80 J/m² , ELF magnetic field exposure for

24 h, followed by incubation for 48 h. DNA damage was assessed by quantification of cyclobutane pyrimidine dimer formation and 6-4 photoproduct formation using ELISA after UV-B irradiation at 20-80 J/m² followed by ELF magnetic field exposure for 24 h. No significant changes were observed in cell survival between ELF magnetic field and sham exposures. Similarly, DNA damage induced by UV-B irradiation did not change significantly following ELF magnetic field exposure. Our results suggest that ELF magnetic field exposure at 5 mT does not have modification effect on cell survival after UV-B irradiation and on repair process of DNA damage induced by UV-B irradiation.

(E) Nikolova T, Czyz J, Rolletschek A, Blyszczuk P, Fuchs J, Jovtchev G, Schuderer J, Kuster N, Wobus AM. Electromagnetic fields affect transcript levels of apoptosis-related genes in embryonic stem cell-derived neural progenitor cells. ASEB J 19(12):1686-1688, 2005. (GT, GE)

Mouse embryonic stem (ES) cells were used as an experimental model to study the effects of electromagnetic fields (EMF). ES-derived nestin-positive neural progenitor cells were exposed to extremely low frequency EMF simulating power line magnetic fields at 50 Hz (ELF-EMF) and to radiofrequency EMF simulating the Global System for Mobile Communication (GSM) signals at 1.71 GHz (RF-EMF). Following EMF exposure, cells were analyzed for transcript levels of cell cycle regulatory, apoptosis-related, and neural-specific genes and proteins; changes in proliferation; apoptosis; and cytogenetic effects. Quantitative RT-PCR analysis revealed that ELF-EMF exposure to ES-derived neural cells significantly affected transcript levels of the apoptosis-related bcl-2, bax, and cell cycle regulatory "growth arrest DNA damage inducible" GADD45 genes, whereas mRNA levels of neural-specific genes were not affected. RF-EMF exposure of neural progenitor cells resulted in down-regulation of neural-specific Nurr1 and in up-regulation of bax and GADD45 mRNA levels. Short-term RF-EMF exposure for 6 h, but not for 48 h, resulted in a low and transient increase of DNA double-strand breaks. No effects of ELF- and RF-EMF on mitochondrial function, nuclear apoptosis, cell proliferation, and chromosomal alterations were observed. We may conclude that EMF exposure of ES-derived neural progenitor cells transiently affects the transcript level of genes related to apoptosis and cell cycle control. However, these responses are not associated with detectable changes of cell physiology, suggesting compensatory mechanisms at the translational and posttranslational level.

(NE) Okudan N, Celik I, Salbacak A, Cicekcibasi AE, Buyukmumcu M, Gökbel H. Effects of long-term 50 Hz magnetic field exposure on the micro nucleated polychromatic erythrocyte and blood lymphocyte frequency and argyrophilic nucleolar organizer regions in lymphocytes of mice. Neuro Endocrinol Lett. 31(2):208-214, 2010. (GT)

OBJECTIVES: We aimed to investigate the effects of weak extremely low frequency electromagnetic fields (ELF-EMFs) on the nucleus size, the silver staining nucleolar organizer regions (AgNORs), the frequency of micro nucleated peripheral blood lymphocytes (MPBLs) and the micro nucleated polychromatic erythrocytes (MPCEs). **METHODS:** One hundred and twenty Swiss albino mice were equally divided into 6 groups. The study groups were exposed to 1, 2, 3, 4 and 5 microT 50 Hz-EMFs for 40 days. Micronucleus number (MN) per PBL was determined. **RESULTS:** ELF-EMF exposure caused a nonlinear decline of nucleus area. A sharp drop occurred in AgNOR area of 1 microT group, and following it gained an insignificantly higher level than that of the control group. The field did not change mean AgNOR

numbers per nucleus of the groups. Relative AgNOR area had the highest level in 1 microT-exposure group, and the level was quite similar to that of the 5 microT-exposure group. The remaining groups had significantly lower values quite similar to that of the control level. The field exposure at any intensity did not affect significantly the frequency of either MPBLs or MPCEs. The number of MN per PBL in the 4 and 5 microT-exposure groups were significantly higher than those of the lower intensity exposure groups. The males in 4 microT-exposure group displayed the highest MN number per PBL, whereas values changed in a nonlinear manner.

CONCLUSIONS: The results of the present study suggest that ≤ 5 microT intensities of 50 Hz EMFs did not cause genotoxic effect on the mouse.

(E) Panagopoulos DJ, Karabarbounis A, Lioliouis C. ELF alternating magnetic field decreases reproduction by DNA damage induction. Cell Biochem Biophys. 67(2):703-16, 2013. (LE, GT, RP)

In the present experiments, the effect of 50-Hz alternating magnetic field on *Drosophila melanogaster* reproduction was studied. Newly eclosed insects were separated into identical groups of ten males and ten females and exposed to three different intensities of the ELF magnetic field (1, 11, and 21 G) continuously during the first 5 days of their adult lives. The reproductive capacity was assessed by the number of F1 pupae according to a well-defined protocol of ours. The magnetic field was found to decrease reproduction by up to 4.3%. The effect increased with increasing field intensities. The decline in reproductive capacity was found to be due to severe DNA damage (DNA fragmentation) and consequent cell death induction in the reproductive cells as determined by the TUNEL assay applied during early and mid-oogenesis (from germarium to stage 10) where physiological apoptosis does not occur. The increase in DNA damage was more significant than the corresponding decrease in reproductive capacity (up to ~7.5%). The TUNEL-positive signal denoting DNA fragmentation was observed exclusively at the two most sensitive developmental stages of oogenesis: the early and mid-oogenesis checkpoints (i.e. region 2a/2b of the germarium and stages 7-8 just before the onset of vitellogenesis)-in contrast to exposure to microwave radiation of earlier work of ours in which the DNA fragmentation was induced at all developmental stages of early and mid-oogenesis. Moreover, the TUNEL-positive signal was observed in all three types of egg chamber cells, mainly in the nurse and follicle cells and also in the oocyte, in agreement with the microwave exposure of our earlier works. According to previous reports, cell death induction in the oocyte was observed only in the case of microwave exposure and not after exposure to other stress factors as toxic chemicals or food deprivation. Now it is also observed for the first time after ELF magnetic field exposure. Finally, in contrast to microwave exposure of previous experiments of ours in which the germarium checkpoint was found to be more sensitive than stage 7-8, in the magnetic field exposure of the present experiments the mid-oogenesis checkpoint was found to be more sensitive than the germarium.

(E) Rageh MM, El-Gebaly RH, El-Bialy NS. Assessment of genotoxic and cytotoxic hazards in brain and bone marrow cells of newborn rats exposed to extremely low-frequency magnetic field. J Biomed Biotechnol. 2012;2012:716023. (LE, GT, DE, OX)

The present study aimed to evaluate the association between whole body exposure to extremely low frequency magnetic field (ELF-MF) and genotoxic , cytotoxic hazards in brain and bone

marrow cells of newborn rats. Newborn rats (10 days after delivery) were exposed continuously to 50 Hz, 0.5 mT for 30 days. The control group was treated as the exposed one with the sole difference that the rats were not exposed to magnetic field. Comet assay was used to quantify the level of DNA damage in isolated brain cells. Also bone marrow cells were flushed out to assess micronucleus induction and mitotic index. Spectrophotometric methods were used to measure the level of malondialdehyde (MDA) and the activity of glutathione (GSH) and superoxide dismutase (SOD). The results showed a significant increase in the mean tail moment indicating DNA damage in exposed group ($P < 0.01, 0.001, 0.0001$). Moreover ELF-MF exposure induced a significant ($P < 0.01, 0.001$) four folds increase in the induction of micronucleus and about three folds increase in mitotic index ($P < 0.0001$). Additionally newborn rats exposed to ELF-MF showed significant higher levels of MDA and SOD ($P < 0.05$). Meanwhile ELF-MF failed to alter the activity of GSH. In conclusion, the present study suggests an association between DNA damage and ELF-MF exposure in newborn rats.

(E) Reyes-Guerrero G, Guzmán C, García DE, Camacho-Arroyo I, Vázquez-García M. Extremely low-frequency electromagnetic fields differentially regulate estrogen receptor-alpha and -beta expression in the rat olfactory bulb. Neurosci Lett. 471(2):109-13, 2010. (GE)

Recently, the effects of extremely low-frequency electromagnetic fields (ELF EMF) on biological systems have been extensively investigated. In this report, the influence of ELF EMF on olfactory bulb (OB) estrogen receptor-alpha (ER alpha) mRNA and -beta (ER beta) mRNA expression was studied by RT-PCR in adult female and male rats. Results reveal for the first time that ELF EMF exerted a biphasic effect on female OB ER beta mRNA gene expression, which increased during diestrus and decreased during estrus. We did not observe any influence of ELF EMF on female OB ER alpha mRNA expression. Our data demonstrate a fluctuating pattern of ER-alpha and -beta mRNA expression in the female OB throughout the phases of the estrous cycle in non-ELF EMF-exposed animals. Thus the highest ER alpha expression was observed in diestrus and the lowest in proestrus. The pattern of ER beta mRNA was less variable, the lowest expression was observed in diestrus. ER-alpha mRNA and -beta mRNA expression level in the male OB did not exhibit any variation either in ELF EMF-exposed or non-ELF EMF-exposed animals. In summary, ELF EMF modulate ER beta gene expression in the OB of female adult rats but not in males.

(E) Ruiz-Gómez MJ, Sendra-Portero F, Martínez-Morillo M. Effect of 2.45 mT sinusoidal 50 Hz magnetic field on *Saccharomyces cerevisiae* strains deficient in DNA strand breaks repair. Int J Radiat Biol. 86(7):602-611, 2010. (GT)

PURPOSE: To investigate whether extremely-low frequency magnetic field (MF) exposure produce alterations in the growth, cell cycle, survival and DNA damage of wild type (wt) and mutant yeast strains. **MATERIALS AND METHODS:** wt and high affinity DNA binding factor 1 (hdf1), radiation sensitive 52 (rad52), rad52 hdf1 mutant *Saccharomyces cerevisiae* strains were exposed to 2.45 mT, sinusoidal 50 Hz MF for 96 h. MF was generated by a pair of Helmholtz coils. During this time the growth was monitored by measuring the optical density at 600 nm and cell cycle evolution were analysed by microscopic morphological analysis. Then, yeast survival was assayed by the drop test and DNA was extracted and electrophoresed.

RESULTS: A significant increase in the growth was observed for rad52 strain ($P = 0.005$, Analysis of Variance [ANOVA]) and close to significance for rad52 hdf1 strain ($P = 0.069$, ANOVA). In addition, the surviving fraction values obtained for MF-exposed samples were in all cases less than for the controls, being the P value obtained for the whole set of MF-treated strains close to significance ($P = 0.066$, Student's t -test). In contrast, the cell cycle evolution and the DNA pattern obtained for wt and the mutant strains were not altered after exposure to MF.

CONCLUSIONS: The data presented in the current report show that the applied MF (2.45 mT, sinusoidal 50 Hz, 96 h) induces alterations in the growth and survival of *S. cerevisiae* strains deficient in DNA strand breaks repair. In contrast, the MF treatment does not induce alterations in the cell cycle and does not cause DNA damage.

(E) Sarimov R, Alipov ED, Belyaev IY. Fifty hertz magnetic fields individually affect chromatin conformation in human lymphocytes: dependence on amplitude, temperature, and initial chromatin state. *Bioelectromagnetics*. 32(7):570-579, 2011. (GT)

Effects of magnetic field (MF) at 50 Hz on chromatin conformation were studied by the method of anomalous viscosity time dependence (AVTD) in human lymphocytes from two healthy donors. MF within the peak amplitude range of 5-20 μ T affected chromatin conformation. These MF effects differed significantly between studied donors, and depended on magnetic flux density and initial condensation of chromatin. While the initial state of chromatin was rather stable in one donor during one calendar year of measurements, the initial condensation varied significantly in cells from another donor. Both this variation and the MF effect depended on temperature during exposure. Despite these variations, the general rule was that MF condensed the relaxed chromatin and relaxed the condensed chromatin. Thus, in this study we show that individual effects of 50 Hz MF exposure at peak amplitudes within the range of 5-20 μ T may be observed in human lymphocytes in dependence on the initial state of chromatin and temperature.

(E) Tiwari R, Lakshmi NK, Bhargava SC, Ahuja YR. Epinephrine, DNA integrity and oxidative stress in workers exposed to extremely low-frequency electromagnetic fields (ELF-EMFs) at 132 kV substations. *Electromagn Biol Med*. 2014 Jan 24. [Epub ahead of print] (LE, GT, HU, OX)

There is apprehension about widespread use of electrical and electromagnetic gadgets which are supposed to emit electromagnetic radiations. Reports are controversy. These electromagnetic fields (EMFs) have considerable effect on endocrine system of exposed subjects. This study was focused to assess the possible bioeffects of extremely low-frequency (ELF)-EMFs on epinephrine level, DNA damage and oxidative stress in subjects occupationally exposed to 132 kV high-voltage substations. The blood sample of 142 exposed subjects and 151 non-exposed individuals was analyzed. Plasma epinephrine was measured by enzyme-linked immunosorbent assay, DNA damage was studied by alkaline comet assay along with oxidative stress. Epinephrine levels of sub-groups showed mean concentration of 75.22 ± 1.46 , 64.43 ± 8.26 and 48.47 ± 4.97 for high, medium and low exposed groups, respectively. DNA damage ranged between 1.69 μ m and 9.91 μ m. The oxidative stress levels showed significant increase. The individuals employed in the live-line procedures were found to be vulnerable for EM stress with altered epinephrine concentrations, DNA damage and increased oxidative stress.

(E) Udroi I, Cristaldi M, Ieradi LA, Bedini A, Giuliani L, Tanzarella C. Clastogenicity and aneuploidy in newborn and adult mice exposed to 50 Hz magnetic fields. Int J Radiat Biol. 82(8):561-567, 2006. (GT, DE, LE)

PURPOSE: To detect possible clastogenic and aneugenic properties of a 50 Hz, 650 μ T magnetic field. **MATERIALS AND METHODS:** The micronucleus test with CREST (Calcinosis, Raynaud's phenomenon, Esophageal dysmotility, Sclerodactyly, Telangiectasia) antibody staining was performed on liver and peripheral blood sampled from newborn mice exposed to an ELF (Extremely Low Frequency) magnetic field during the whole intra-uterine life (21 days), and on bone marrow and peripheral blood sampled from adult mice exposed to the same magnetic field for the same period. **RESULTS:** Data obtained in newborn mice show a significant increase in micronuclei frequencies. In absolute terms, most of the induced micronuclei were CREST-negative (i.e., formed by a chromosome fragment). However, in relative terms, ELF exposure caused a two-fold increase in CREST-negative micronuclei and a four-fold increase in CREST-positive micronuclei (i.e., formed by a whole chromosome). No significant effect was recorded on exposed adults. **CONCLUSIONS:** These findings suggest the need for investigation of aneugenic properties of ELF magnetic fields in order to establish a possible relationship to carcinogenesis.

(NE) Verschaeve L, Anthonissen R, Grudniewska M, Wudarski J, Gevaert L, Maes A. Genotoxicity investigation of ELF-magnetic fields in Salmonella typhimurium with the sensitive SOS-based VITOTOX test. Bioelectromagnetics. 32(7):580-584, 2011. (GT, IA)

We performed a genotoxicity investigation of extremely low-frequency (ELF) magnetic fields (MFs, 50 Hz, 100 and 500 μ T, 1 and 2 h exposure) alone and in combination with known chemical mutagens using the VITOTOX test. This test is a very sensitive reporter assay of Salmonella typhimurium bacteria based on the SOS response. Our study showed that ELF-MFs do not induce SOS-based mutagenicity in S. typhimurium bacteria and do not show any synergetic effect when combined with chemical mutagens.

(E) Villarini M, Ambrosini MV, Moretti M, Dominici L, Taha E, Piobbico D, Gambelunghe C, Mariucci G. Brain hsp70 expression and DNA damage in mice exposed to extremely low frequency magnetic fields: a dose-response study. Int J Radiat Biol. 89(7):562-570, 2013. (LE, GT)

Purpose: To determine whether a dose-response relationship exists among exposure to extremely low frequency magnetic fields (ELF-MF) at different densities and 70-kDa heat shock protein (hsp70) expression and DNA damage in mouse brain. **Materials and Methods:** Male CD1 mice were exposed to ELF-MF (50 Hz; 0.1, 0.2, 1 or 2 mT) for 7 days (15 hours/day) and sacrificed either at the end of exposure or after 24 h. Hsp70 expression was determined in cerebral cortex-striatum, hippocampus and cerebellum by real-time reverse-transcriptase polymerase chain reaction (RT-PCR) and western blot analysis. Primary DNA damage was evaluated in the same tissues by comet assay. Sham-exposed mice were used as controls. **Results:** No changes in both hsp70 mRNA and corresponding protein occurred following exposure to ELF-MF, except for a weak increase in the mRNA in hippocampus of exposed mice to 0.1 mT ELF-MF. Only mice exposed to 1 or 2 mT and sacrificed immediately after exposure presented DNA strand

breaks higher than controls in all the cerebral areas; such DNA breakage reverted to baseline in the mice sacrificed 24 h after exposure. Conclusions: These data show that high density ELF-MF only induce reversible brain DNA damage while they do not affect hsp70 expression.

(E) [Wahab MA](#), [Podd JV](#), [Rapley BI](#), [Rowland RE](#). Elevated sister chromatid exchange frequencies in dividing human peripheral blood lymphocytes exposed to 50 Hz magnetic fields. [Bioelectromagnetics](#). 28(4):281-288, 2007. (GT, WS)

The in vitro cytomolecular technique, sister chromatid exchange (SCE), was applied to test the clastogenic potentiality of extremely low frequency (ELF) electromagnetic fields (EMFs) on human peripheral blood lymphocytes (HPBLs). SCE frequencies were scored in dividing peripheral blood lymphocytes (PBLs) from six healthy male blood donors in two rounds of experiments, R1 and R2, to determine reproducibility. Lymphocyte cultures in the eight experiments conducted in each round were exposed to 50 Hz sinusoidal (continuous or pulsed) or square (continuous or pulsed) MFs at field strengths of 1 microT or 1 mT for 72 h. A significant increase in the number of SCEs/cell in the grouped experimental conditions compared to the controls was observed in both rounds. The highest SCE frequency in R1 was 10.03 for a square continuous field, and 10.39 for a square continuous field was the second highest frequency in R2. DNA crosslinking at the replication fork is proposed as a model which could explain the mechanistic link between ELF EMF exposure and increased SCE frequency.

(E) Wang Z, Sarje A, Che PL, Yarema KJ. Moderate strength (0.23-0.28 T) static magnetic fields (SMF) modulate signaling and differentiation in human embryonic cells. BMC Genomics. 10:356, 2009. (GE)

BACKGROUND: Compelling evidence exists that magnetic fields modulate living systems. To date, however rigorous studies have focused on identifying the molecular-level biosensor (e.g., radical ion pairs or membranes) or on the behavior of whole animals leaving a gap in understanding how molecular effects are translated into tissue-wide and organism-level responses. This study begins to bridge this gulf by investigating static magnetic fields (SMF) through global mRNA profiling in human embryonic cells coupled with software analysis to identify the affected signaling pathways. **RESULTS:** Software analysis of gene expression in cells exposed to 0.23-0.28 T SMF showed that nine signaling networks responded to SMF; of these, detailed biochemical validation was performed for the network linked to the inflammatory cytokine IL-6. We found the short-term (<24 h) activation of IL-6 involved the coordinate up-regulation of toll-like receptor-4 (TLR4) with complementary changes to NEU3 and ST3GAL5 that reduced ganglioside GM3 in a manner that augmented the activation of TLR4 and IL-6. Loss of GM3 also provided a plausible mechanism for the attenuation of cellular responses to SMF that occurred over longer exposure periods. Finally, SMF-mediated responses were manifest at the cellular level as morphological changes and biochemical markers indicative of pre-oligodendrocyte differentiation. **CONCLUSION:** This study provides a framework describing how magnetic exposure is transduced from a plausible molecular biosensor (lipid membranes) to cell-level responses that include differentiation toward neural lineages. In addition, SMF provided a stimulus that uncovered new relationships - that exist even in the absence of magnetic fields - between gangliosides, the time-dependent regulation of IL-6 signaling by these glycosphingolipids, and the fate of embryonic cells.

(NE) Williams PA, Ingebretsen RJ, Dawson RJ. 14.6 mT ELF magnetic field exposure yields no DNA breaks in model system Salmonella, but provides evidence of heat stress protection. Bioelectromagnetics. 27(6):445-450, 2006. (GT)

In this study, we demonstrate that common extremely low frequency magnetic field (MF) exposure does not cause DNA breaks in this Salmonella test system. The data does, however, provide evidence that MF exposure induces protection from heat stress. Bacterial cultures were exposed to MF (14.6 mT 60 Hz field, cycled 5 min on, 10 min off for 4 h) and a temperature-matched control. Double- and single-stranded DNA breaks were assayed using a recombination event counter. After MF or control exposure they were grown on indicator plates from which recombination events can be quantified and the frequency of DNA strand breaks deduced. The effect of MF was also monitored using a recombination-deficient mutant (recA). The results showed no significant increase in recombination events and strand breaks due to MF. Evidence of heat stress protection was determined using a cell viability assay that compared the survival rates of MF exposed and control cells after the administration of a 10 min 53 degrees C heat stress. The control cells exhibited nine times more cell mortality than the MF exposed cells. This Salmonella system provides many mutants and genetic tools for further investigation of this phenomenon.

(E) Yokus B, Akdag MZ, Dasdag S, Cakir DU, Kizil M. Extremely low frequency magnetic fields cause oxidative DNA damage in rats. Int J Radiat Biol. 84(10):789-795, 2008. (GT)

PURPOSE: To detect the genotoxic effects of extremely low frequency (ELF) -magnetic fields (MF) on oxidative DNA base modifications [8-hydroxyguanine (8-OH-Gua), 2,6-diamino-4-hydroxy-5-formamidopyrimidine (FapyGua) and 4,6-diamino-5-formamidopyrimidine (FapyAde)] in rat leucocytes, measured following exposure to ELF-MF. **MATERIALS AND METHODS:** After exposure to ELF-MF (50 Hz, 100 and 500 microT, for 2 hours/day during 10 months), DNA was extracted, and measurement of DNA lesions was achieved by gas chromatography/mass spectrometry (GC/MS) and liquid chromatography/mass spectrometry (LC/MS). **RESULTS:** Levels of FapyAde, FapyGua and 8OHdG in DNA were increased by both 100 microT and 500 microT ELF-MF as compared to a cage-control and a sham group; however, statistical significance was observed only in the group exposed to 100 microT. **CONCLUSION:** This is the first study to report that ELF-MF exposure generates oxidatively induced DNA base modifications which are mutagenic in mammalian cells, such as FapyGua, FapyAde and 8-OH-Gua, in vivo. This may explain previous studies showing DNA damage and genomic instability. These findings support the hypothesis that chronic exposure to 50-Hz MF may be potentially genotoxic. However, the intensity of ELF-MF has an important influence on the extent of DNA damage.

(E) Yoon HE, Lee JS, Myung SH, Lee YS. Increased γ -H2AX by exposure to a 60-Hz magnetic fields combined with ionizing radiation, but not hydrogen peroxide, in non-tumorigenic human cell lines. Int J Radiat Biol. 2014 Jan 28. [Epub ahead of print] (GT, IA)

Purpose: Genotoxic effects have been considered the gold standard to determine if an environmental factor is a carcinogen, but the currently available data for extremely low

frequency time-varying magnetic fields (ELF-MFs) remain controversial. As an environmental stimulus, the effect of ELF-MF on cellular DNA may be subtle. Therefore, a more sensitive method and systematic research strategy are warranted to evaluate genotoxicity. Materials and methods: We investigated the effect of ELF-MFs in combination with ionizing radiation (IR) or H₂O₂ on the DNA damage response of expression of phosphorylated H2AX (γ -H2AX) and production of γ -H2AX foci in non-tumorigenic human cell systems consisting of human lung fibroblast WI38 cells and human lung epithelial L132 cells. Results: Exposure to a 60-Hz, 2 mT ELF-MFs for 6 h produced increased γ -H2AX expression, as well as γ -H2AX foci production, a common DNA double-strand break (DSB) marker. However, exposure to a 1 mT ELF-MFs did not have the same effect. Moreover, 2 mT ELF-MFs exposure potentiated the expression of γ -H2AX and γ -H2AX foci production when combined with IR, but not when combined with H₂O₂. Conclusions: ELF-MFs could affect the DNA damage response and, in combination with different stimuli, provide different effects on γ -H2AX.



MEMORANDUM

TO: The Town of Montreat Board of Commissioners

CC: Ben Blackburn (Interim Town Manager), Angela Murphy (Town Clerk)

FROM: Kayla DiCristina (Zoning Administrator)

SUBJECT: French Broad MPO Unified Planning Work Program Call for Projects

DATE: December 8, 2022

ATTACHMENTS: Correspondence with Biltmore Forest Town Manager & Federal Highway Administration's Course on Bicycle and Pedestrian Safety Lesson 11

On November 2, 2022, the French Broad River Metropolitan Planning Organization (FBRMPO), of which Montreat is a part of, announced a Call for Projects under the Unified Planning Work Program (UPWP). Per the FBRMPO's website...

"The Unified Planning Work Program (UPWP) is a framework for the planning activities and efforts that the FBRMPO will undertake for the fiscal year. Serving as both a budget and guide, the UPWP is adopted on an annual basis in accordance with Federal Highway Administration (FHWA) and Federal Transit Administration (FTA) guidelines. The UPWP illustrates the funding allocations from federal transportation planning funds, including highway and transit programs. An example of a few items outlined in the UPWP include project prioritization coordination, bicycle and pedestrian planning, special studies, and public involvement. The UPWP is the MPO's budget for planning activities through the fiscal year, including in-house planning activities and special studies undertaken in the region using MPO planning funds."

The Call for Projects under the UPWP is a funding opportunity for short-range transportation planning activities throughout the FBRMPO region for Fiscal Year 2023. There is no budget limit, but the program contains \$200,000.00 of available funds for the region. This program requires applicants (i.e. The Town of Montreat) to provide a 20% cost match for the overall project cost. In other words, if the project is budgeted at \$20,000.00, the Town of Montreat would be required to

provide \$2,000.00 in funds to meet the cost share requirement. While the Board of Commissioners does not need to formally commit to this cost share at the time of application, the deadline to submit the local match commitment letter is July 1, 2023. Applications are due December 19, 2022 and projects will be announced in the summer or fall of 2023.

On November 3, 2022, Mayor Tim Helms contacted the Planning and Zoning Department and the Interim Town Manager to see if there was a desire or need to seek funding for a traffic study. The Mayor went on to say that subjects such as speed and increasing traffic amounts continue to surface within the Town. The Zoning Administrator responded with a recommendation to discuss with the full Board of Commissioners pursuing a transportation study limited to examining traffic calming for NC-9/Assembly Drive and a parking study focused on NC-9/Assembly Drive, Lookout Road, and the portion of Greybeard Trail within the municipal limits.

The Zoning Administrator met with the Chief of Police to discuss the current state of speeding and safety concerns on NC-9/Assembly Drive. The Chief of Police shared anecdotal observations with the Zoning Administrator and stated that the recent purchase of a new flashing speed sign may indicate an increase in speeding and reduction of pedestrian safety on NC-9/Assembly Drive, particularly at the Town's entrance gate. Traffic calming, per the Federal Highway Administration's Course on Bicycle and Pedestrian Safety Lesson 11, is a term used to describe a collection of methods intended to slow cars as they move through commercial and residential neighborhoods. While the fundamental goal of traffic calming is to reduce the speed of cars, additional benefits may appear from the implementation of traffic calming measures. For example, traffic calming measures may improve the pedestrian environment or the "feel" of the street while also increasing pedestrian safety. An examination of traffic calming measures for NC-9/Assembly Drive would investigate the current state of the road to ascertain existing issues and concerns via an examination of existing information and public participation, analyze this information, identify opportunities for improvement, and provide a list of recommendations to calm traffic in this area.

The inclusion of a parking study in this project with the goal of addressing the concerns of volume and availability of parking may also be beneficial to the Town. The Zoning Administrator recommends the parking study focus on NC-9/Assembly Drive, Lookout Road, and the portion of Greybeard Trail within the municipal limits as, anecdotally, these areas appear to be hot spots for parking concerns. A parking study intends to examine the existing parking system via an examination of existing information and public participation and develop a series of

recommendations for improvements to existing infrastructure and strategies for handling increased demand. As a note, the recommendations provided as a part of the traffic calming and parking studies are not binding. Instead, these recommendations serve as possible pathways the Town may take to address the identified issues in the studied areas in the future when feasible.

In 2022, the Town of Biltmore Forest, with the assistance of a consultant, developed the Town of Biltmore Forest Transportation Study. This plan is an example of a transportation study which includes the examination and development of recommendations for traffic calming and parking improvements. As of the writing of this memo, the Town of Biltmore Forest implemented an all-way stop at the intersection of Vanderbilt/Busbee Roads, which has been a huge success, and is continuing to implement recommendations identified in their plan. While the Town of Montreat's study will be more limited in scope, this plan provides a local example of what a study of this nature may generate for the Town. A link to the Town of Biltmore Forest Transportation Study can be found

here:

https://www.biltmoreforest.org/sites/default/files/uploads/documents/surveys/biltmore_forest_transportation_study_3.4.22_final.pdf.

To pursue an application for a transportation study focused on traffic calming and parking, the Board of Commissioners must direct the Zoning Administrator to prepare an application to the UPWP on behalf of the Town of Montreat at the December 8, 2022 Board of Commissioners meeting. The Zoning Administrator recommends a maximum request of \$25,000.00 with a required cost share of \$2,500.00, as this is approximately what the Town of Biltmore Forest paid for the Town of Biltmore Forest Transportation Study. As stated previously, the Board of Commissioners does not need to formally commit to this cost share at the time of application, but will need to provide a commitment letter to the FBRMPO no later than July 1, 2023. Should the application be awarded to the Town of Montreat, the Zoning Administrator will request that FBRMPO staff manage the project and acquire consulting services to prepare the transportation study. There is no cost associated with management of the project by FBRMPO staff. The Zoning Administrator will serve in a supporting role to the consultant and FBRMPO staff throughout the duration of this project. In sum, the Zoning Administrator requests direction from the Board of Commissioners on submitting an application to the FBRMPO UPWP Call for Projects for a transportation study limited in scope to traffic calming for NC-9/Assembly Drive and parking for NC-9/Assembly Drive, Lookout Road, and Greybeard Trail within the municipal limits.

RE: Traffic Calming Study Question

Jonathan Kanipe <jkanipe@biltmoreforest.org>

Tue 11/8/2022 2:56 PM

To: Kayla DiCristina <kayla@landofsky.org>; Harry Buckner Public Works Director <hbuckner@biltmoreforest.org>

Cc: Tristan Winkler <Tristan@landofsky.org>

 2 attachments (1 MB)

Biltmore Forest Town-Wide Traffic Study Proposal_(JMTE)_9.17.21.pdf; SURVEY for PRINT (Biltmore Forest Transportation Study) 11.5.21.pdf;

Be Advised: This email originated from outside Land of Sky

Hi Tristan and Kayla,

We're happy to help! We performed our traffic (transportation) study the end of 2021-early 2022, and presented to our Board in March 2022. The Board adopted the plan as part of our overall comprehensive plan in May 2022. We used J.M. Teague for this work. The full cost was just over \$20,000. I have attached the scope we agreed upon in September 2021 and you can see exactly what we were looking for and how we structured it. As a note, we did revise the price down somewhat and reduced some of the scope from our initial meetings – but what you'll see in this scope is what we really needed.

The big thing for Biltmore Forest was being proactive in traffic management and having a plan (based largely on citizen feedback) that we could utilize when making decisions. The survey component was huge for us and provided us a great opportunity to tackle some otherwise “tough” issues – which we're still working on today. Of the items mentioned, we have implemented an all-way stop at Vanderbilt/Busbee Roads (after the Board determined the feasibility for a roundabout there was not doable) and that has been a huge success. We've had no complaints at all about that change. We are currently striping many of the thoroughfares in town to provide defined lanes of travel – with the idea that this will help with some speeding complaints as well as specifically dictating where pedestrians need to walk. Otherwise, we're working on some of the other changes but have not moved much more through them as yet.

The full report is a huge file, but you can access it from our website [here](#). I've attached our survey questions and the verbatim responses are listed in the full report. Feel free to let me know if we can help with anything else.

Jonathan Kanipe
Town Manager
Town of Biltmore Forest
(828) 274-0824 // jkanipe@biltmoreforest.org
<http://www.biltmoreforest.org>

All email correspondence to and from this address is subject to public review under the NC Public Records Law.

From: Kayla DiCristina <kayla@landofsky.org>
Sent: Tuesday, November 08, 2022 1:55 PM
To: Jonathan Kanipe <jkanipe@biltmoreforest.org>; Harry Buckner <hbuckner@biltmoreforest.org>
Cc: Tristan Winkler <Tristan@landofsky.org>
Subject: Re: Traffic Calming Study Question

WARNING: This email originated from outside of the Town of Biltmore Forest Network.

Tristan, thanks for connecting us!

Good afternoon Harry and Jonathan,

Any information you can share is much appreciated. I'd also be curious to know how many of the recommendations have been implemented and what the effect has been so far. Thanks in advance!

Best,

Kayla DiCristina, AICP

(*For inquiries regarding the Town of Montreat, please see below)

Regional Planner | Economic and Community Development

Land of Sky Regional Council

339 New Leicester Hwy., Suite 140 • Asheville, NC 28806



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***Town of Montreat:** *Inquiries regarding the Town of Montreat are answered in the order they are received during Montreat office hours every Tuesday through Thursday 8:00 am through 5:00 pm. For assistance, please call 828-669-8002, ext. 3030, or e-mail zoning@townofmontreat.org.*

This institution is an equal opportunity provider and employer. All email correspondence to and from this address is subject to public review under the NC Public Records Law.

From: Tristan Winkler <Tristan@landofsky.org>
Sent: Tuesday, November 8, 2022 1:42 PM
To: Manager Jonathan Kanipe <jkanipe@biltmoreforest.org>; Harry Buckner Public Works Director <hbuckner@biltmoreforest.org>
Cc: Kayla DiCristina <kayla@landofsky.org>
Subject: Traffic Calming Study Question

Harry and Jonathan,

I hope this email finds you both well.

The Town of Montreat is interested in pursuing funding for a traffic calming study and are trying to put together a potential budget for the project- would you all be willing to share the budget for the study done in Biltmore Forest? Kayla DiCristina is cc'd and is helping Montreat put together a potential application.

Thank you!

Tristan Winkler
 French Broad River MPO Director
 Land of Sky Regional Council
 He/Him/His
 828.251.7454
 847.997.7328 (cell)
Tristan@landofsky.org



All email correspondence to and from this address is subject to public review under the NC Public Records Law.

L E S S O N 11

Traffic Calming

11.1 Purpose

Traffic calming is a traffic management approach that evolved in Europe and is now being implemented in many U.S. cities. The following definition is quoted from *An Illustrated Guide to Traffic Calming* by Hass Klau (1990):

“Traffic calming is a term that has emerged in Europe to describe a full range of methods to slow cars, but not necessarily ban them, as they move through commercial and residential neighborhoods. The benefit for pedestrians and bicyclists is that cars now drive at speeds that are safer and more compatible to walking and bicycling. There is, in fact, a kind of equilibrium among all of the uses of a street, so no one mode can dominate at the expense of another.”

This chapter explores the principle of traffic calming and provides a variety of studies, design details, and photographs of areas where traffic calming has been effectively used in the United States and in Europe. Along with the advantages of traffic calming, the text describes mistakes that practitioners have sometimes made in implementing traffic-calming techniques.

11.2 Traffic-Calming Objectives

The most fundamental traffic-calming goal is to reduce the speed of vehicular movement. With reduction of speed, the following objectives can be realized:

1. **Improved “feel” of the street.**
This objective calls for increased community involvement in and “ownership” of the street. If people feel more comfortable on the street, they are more likely to walk or bicycle there and to



engage in other street-oriented activities with their neighbors. A key aspect of achieving this objective is reducing the perceived threat of danger from motor traffic.

2. Enhanced aesthetic values and a sense of nature.

Several traffic-calming techniques, such as street landscaping, pedestrian amenities, and reclamation of roadway areas can serve as community open space. Not only do these techniques make the neighborhood more attractive, but they also break up long, uninterrupted street vistas conducive to speeding and convey the message that “this is a pedestrian place.”

3. Reduced crime.

It’s harder to make a speedy getaway if a fleeing felon has to deal with speed humps, woonerfs, and traffic circles. It’s harder to get away without being spotted if there are “eyes on the street” – if the street is a positive, community focus.

4. Equitable balance among transportation modes.

With reduced motorist speeds, safety is improved. Pedestrians and bicyclists have more time to detect and avoid motor vehicles. Traffic



Traffic-calming devices are used to break up long uninterrupted street vistas that encourage speeding.

calming sends the message that “motor vehicles don’t exclusively OWN the roadway” – that other modes have equal rights. Studies that evaluate traffic-calming improvements show increased levels of walking, bicycling, and transit use following installation.

5. Increased safety/decreased severity of injury in traffic crashes. With reduced speeds comes a significant reduction in the number and severity of crashes involving motor vehicles. Traffic-calming

facility evaluations uniformly show fewer crashes, fewer fatalities, and less severe injuries.

6. Improved air quality and noise levels.

Slower moving vehicles make less noise and, generally, emit fewer pollutants.

7. Decreased fuel consumption.

With more trips made by walking, bicycling, and transit, and with slower traffic speeds, fuel consumption reductions of 10 to 12 percent have been reported.

8. Continued accommodation of motor vehicle traffic.

An important objective is the continued accommodation of motor vehicle traffic. Although traffic calming shifts the balance among travel

modes, this shift should not result in severely restricted traffic volumes or in shifting traffic problems from the traffic-calmed area to other streets.



11.3 Traffic-Calming Issues

When any new traffic management approach is introduced, issues, concerns, and questions are bound to arise. Design decisions related to traffic can have far-reaching consequences. Lives, economic well-being, and urban livability are directly affected.

Professional engineers, planners, government, and the public all are aware of and sensitive to proposals for changes in the traffic environment. Roadway congestion, air quality, traffic safety, street crimes, and the high cost of new improvements are among the most-widely debated issues in America today. New design ideas are, and should be, subjected to rigorous testing and evaluation before being accepted as part of the standard engineering and transportation planning tool kit. Traffic calming is not a panacea for urban transportation woes, but it can have significant benefits in many situations.



Traffic calming can be termed as engineering and other physical measures designed to control traffic speeds and encourage driving behavior appropriate to the

In considering the application of traffic-calming techniques, what specific issues are likely to arise? The discussion on the following pages focuses on traffic-calming issues. (Note: Studies and statistics referenced are cited in FHWA Case Study Nos. 19 and 20, *National Bicycling and Walking Study*.)

1. Traffic safety.

The Issue: Encouraging people to walk, play, and bicycle in and next to the streets is just asking for trouble. They will have a false sense of security and accidents will increase. They will develop bad habits that may increase their when they leave the neighborhood.

Comment: Traffic-calming measures have been implemented in many European cities. In the Netherlands and Germany, extensive research has been conducted to evaluate the safety and impact of traffic-calming techniques and devices.

2. Impact on traffic volumes, distribution, and operations.

The Issue: Traffic calming will never work on anything except very low-volume residential streets. It will substantially reduce the amount of traffic that a street can handle efficiently and this is counterproductive. We need to move vehicles, not restrict them. Furthermore, if we slow traffic on one street, the traffic will simply be diverted to another street. The net result will be increased congestion and more problems overall.

Comment: A 5-year German Federal Government evaluation of traffic calming and follow-up research found:

- Little change in overall traffic volumes.
- Reduction in average vehicle speeds by almost 50 percent.
- Average increase in motorist trip time of only 33 seconds.

3. Lack of proven design standards.

The Issue: There are no uniform, accepted, and legally defensible standards to follow. If we want to try traffic calming, where can we get specific information about design?

Comment: Many U.S. cities are now developing and testing design guidelines for traffic-calming improvements. Although uniform, national standards have yet to evolve, valuable experience is being gained. The list of references at the end of this lesson provides a starting point for further exploration of specific design approaches.

4. Liability.

The Issue: These traffic-calming ideas may be accepted in Europe, but they haven't really been tried here. Are we opening the door to all kinds of legal problems if somebody crashes on a traffic circle or a speed table and sues us?

Comment: When considering the use of any new design approach, concerns about liability can be



Emergency vehicle access should always be considered when incorporating traffic-calming measures.

addressed somewhat through performance of “due diligence” on the part of the engineer, planner, or other professionals involved in the design. Research into the experiences of other U.S. cities, European standards, and evaluation studies should be thorough and followed up with a first-hand look if possible. Construction of a pilot project or other testing of proposed designs can benefit, as can ongoing and systematic evaluation of the improvements once installed.

5. Emergency and service vehicle access.

The Issue: Construction of speed bumps, neck-downs, medians, and traffic circles will increase response times for emergency vehicles and may restrict access for garbage trucks, delivery vans, and other large vehicles.

Comment: Studies in Berkley and Palo Alto, CA, show that traffic management measures (e.g., traffic diverters, bicycle boulevards) have not impaired police or fire emergency response times.

- The Seattle Engineering Department works closely with its Fire Department to design and field-test traffic circles on a site-specific basis to ensure good emergency access.

6. Impacts on bicycling.

The Issue: Pavement texturing, speed tables, wider sidewalks, “bulb-outs” at corners and similar improvements may make things better for pedestrians, but may have a negative impact on bicycling.

Comment: A 5-year German Federal Government evaluation of traffic calming and follow-up research found doubling of bicycle use over a 4-year period.

- Implementation of traffic management strategies in the downtown area of the Dutch City of Groningen contributed to a substantial increase in bicycling and walking. Bicycle use is now well over 50 percent of all trips.
- Studies of traffic-calming areas in Japan show increases in both bicycle and pedestrian traffic volumes along most routes.

(Note: *Cyclists and Traffic Calming*, a Technical Note publication of the Cyclists Touring Club (see references, end of lesson), includes extensive information on adapting traffic-calming techniques for bicycling.

11.4 Traffic-Calming Devices

Traffic calming has many potential applications, especially in residential neighborhoods and small commercial centers. Traffic-calming devices can be grouped within the following general categories:

- Bumps, humps, and other raised pavement areas.
- Reducing street area where motor traffic is given priority.
- Street closures.
- Traffic diversion.
- Surface texture and visual devices.
- Parking treatments.

Frequently, a combination of traffic-calming devices is used. Examples of such combinations will be discussed briefly, including:

- The woonerf.
- Entry treatments across intersections.
- Shared surfaces.
- Bicycle boulevards.
- Slow streets.

- Channelization changes.
- Traffic calming on a major road.
- Modified intersection design.

1. Bumps, humps, and other raised pavement areas.

This category includes all traffic-calming devices raised above pavement level. Drivers must slow down when they cross these devices or suffer an uncomfortable KER-BUMP or (KER-BUMP-KER-BUMP), running the risk of spilled coffee and a severe jolt to their tailbones. Although people often gripe about the inconvenience of having to slow down for these devices, they don't have much choice. Their effectiveness at slowing traffic cannot be disputed. They are sometimes referred to as "Silent Policemen."

Included in this category are:

- Speed bumps.
- Speed humps.
- Raised crosswalks.
- Raised intersections.

The following are brief descriptions of each, with definitions, comments, and examples:

Speed Bumps

A speed bump is a raised area in the roadway pavement surface extending transversely across the travel way, generally with a height of 3 to 6 inches and a length of 1 to 3 feet.

Design Considerations:

- Most effective if used in a series at 300- to 500- foot spacing.
- Typically used on private property for speed control – parking lots, apartment complexes, private streets, and driveways.
- Speed bumps are not conducive to bicycle travel, so they should be used carefully.



Speed bumps can be combined with curb extensions and a winding street alignment. Signing and pavement markings should clearly identify the bump.

Speed Humps

A speed hump (or "road hump") is a raised area in the roadway pavement surface extending transversely across the roadway. Speed humps normally have a minimum height of 3 to 4 inches and a travel length of approximately 12 feet, although these dimensions may vary. In some cases, the speed hump may raise the roadway surface to the height of the adjacent curb for a short distance.

The humps can be round or flat-topped. The flat-topped configuration is sometimes called a "speed table." Humps can

either extend the full width of the road, curb-to-curb, or be cut back at the sides to allow bicycles to pass and facilitate drainage.

Design Considerations:

- If mid-block pedestrian crossings exist or are planned, they can be coordinated with speed hump installation since vehicle speeds will be lowest at the hump to negotiate ramps or curbs between the sidewalk and the street.
- The hump must be visible at night.
- Speed humps should be located to avoid conflict with underground utility access to boxes, vaults, and sewers.



Speed humps slow traffic speeds on residential streets.

- Speed humps should not be constructed at driveway locations.
- Speed humps may be constructed on streets without curbs, but steps should be taken to prevent circumnavigation around the humps in these situations.
- Adequate signing and marking of each speed hump is essential to warn roadway users of the hump's presence and guide their subsequent movements.
- Speed humps should not be installed in street sections where transit vehicles must transition between the travel lane and curbside stop. To the extent possible, speed humps should be located to ensure that transit vehicles can traverse the hump perpendicularly.
- A single hump acts as only a point speed control. To reduce speeds along an extended section of street, a series of humps is usually needed. Typically, speed humps are spaced at between 300 and 600 feet apart.

Example:

Bellevue, Washington has installed speed humps in residential neighborhoods (labeled as speed “bumps” below, although broader than the typical speed bump). The City uses a 12-foot-wide hump, 3 inches high at the center. The design allows for little



Raised crosswalks can both slow motor traffic and give pedestrians a continuous-level surface at the crossing. Changes in texture and color help define the edges of the crossing.

or no discomfort at speeds of 15 to 25 mph, but will cause discomfort at higher speeds. The humps are marked clearly, distinguishing them from crosswalks. White reflectors enhance nighttime visibility.

Bellevue found that the speed humps reduced traffic speeds and volumes. The humps, in general, received strong public support, and residents favored their permanent installation.

The following concerns were raised regarding the speed hump installation:

- Concern about restricted access and increased response time for emergency vehicles. The Bellevue Fire Department asked that the humps be installed on primary emergency access routes.
- Concern about aesthetics of signing and markings at the traffic humps. Residents raising the concerns, however, felt that the speed reductions compensated for the appearance of the humps.
- Concern about the effectiveness of the humps in reducing motor vehicle speeds along the length of a street, not at just two or three points. The distance between speed humps was found to have an impact on traffic speeds. The City found that maximum spacing should be approximately 500 feet.

The Bellevue Department of Public Works concluded that speed humps were effective speed-control measures on residential streets and recommended their use be continued. The table on the following page summarizes “before” and “after” data related to the Bellevue speed humps:

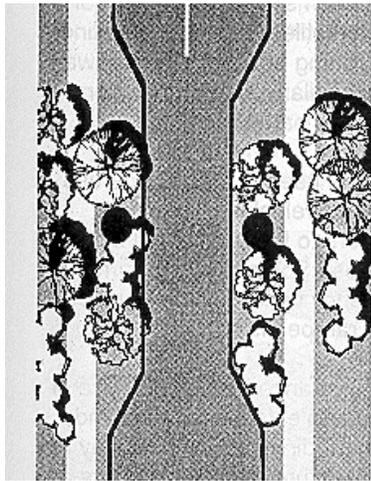
Raised Crosswalks

Raised crosswalks are essentially broad, flat-topped speed humps that coincide with pedestrian crosswalks at street intersections. The crosswalks are raised above the level of the roadway to slow traffic, enhance crosswalk visibility, and make the crossing easier for pedestrians who may have difficulty stepping up and down curbs.

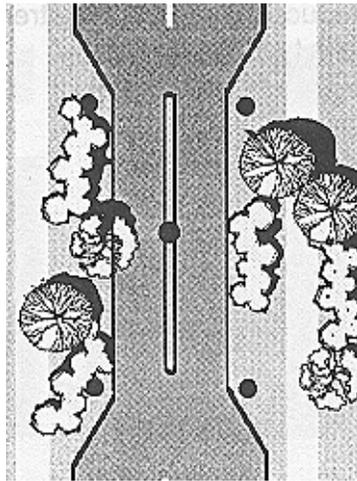
Table 2. Bellevue Speed Humps Findings

LOCATION	STREET TYPE/ WIDTH	# OF HUMPS	HUMP SPACING	SPEED LIMIT	Before:		After:	
					85TH % SPEED	VPD	85TH % SPEED	VPD
Somerset Drive SE	Two-way, 40 feet wide local residential neighborhood street	2	340'	25 mph	39 mph	795	27 mph	541 (VPD increased to 746 when the hump was reduced from 3/4" to 3")
Highland Drive SE	Two-way, 35 feet wide neighborhood collector	3	220'	25 mph	36 mph	1,700	25 mph	No change because no alternative route exists
166th/162nd Avenue SE	Two-way, 36 feet wide local residential street; walk to school route	2	600'	25 mph	37 mph	655	24 mph	.017
		2	580'	25 mph	37 mph	472	27 mph	.017
SE 63rd Street	Two-way, 35 feet wide local residential street temporarily serving as a connection between two minor arterials	2	1,000'	25 mph	36 mph	2,456	27 mph	2,802
		3	500'					
Yarrow Bay neighborhood	Primarily a neighborhood connector	2	400		39 mph	3,685	25 mph	2,931
						1,641		1,653

Source: FHWA Case Study No. 19.



ONE-LANE SLOW POINT



TWO-LANE SLOW POINT

Design Considerations:

- Ramps should not exceed a maximum gradient of 16 percent.
- Raised and/or textured surfaces can be used to alert drivers to the need for particular care.
- Distinctive surfacing helps reinforce the concept of a “calmed” area and thus plays a part in reducing vehicle speeds.
- Distinctive surfacing materials should be skid-resistant, particularly on inclines.

Design Considerations:

- Can be constructed of brick, concrete block, colored asphalt or cement, with ramps striped for better visibility.
- Raised crosswalks are applicable:
 - (1) On roadways with vehicular speeds perceived as being incompatible with the adjacent residential land uses.
 - (2) Where there is a significant number of pedestrian crossings.
 - (3) In conjunction with other traffic-calming devices, particularly entry treatments.
 - (4) On two-lane or fewer residential streets classified as either “local streets” or neighborhood collector streets.”
 - (5) On roadways with 85th percentile speeds less than 45 mph.

- Ramps should be clearly marked to enable bicyclists to identify and anticipate them, particularly under conditions of poor visibility.
- Care must be taken so the visually impaired have adequate cues to identify the roadway’s location (e.g., tactile strips). Color contrasts will aid those who are partially sighted.

2. Reducing street area where motor traffic is given priority.

This category of traffic-calming techniques includes all those that reduce the area of the street designated exclusively for motor vehicle travel. “Reclaimed” space is typically used for landscaping, pedestrian amenities, and parking.

Discussed here are:

- Slow points.
- Medians.
- Curb extensions.
- Corner radius treatment.
- Narrow traffic lanes.

Slow Points (neck-downs, traffic throttles, pinch points)

Slow points narrow a two-way road over a short distance, forcing motorists to slow and, in some cases, to merge into a single lane. Sometimes these are used in conjunction with a speed table and coincident with a pedestrian crossing. The following are advantages and disadvantages of both one-lane and two-lane slow points:

Intersection Humps/Raised Intersections

Intersection humps raise the roadway at the intersection, forming a type of “plateau” across the intersection, with a ramp on each approach. The plateau is at curb level and can be enhanced through the use of distinctive surfacing such as pavement coloring, brickwork, or other pavements. In some cases, the distinction between roadway and sidewalk surfaces is blurred. If this is done, physical obstructions such as bollards or planters should be considered, restricting the area to which motor vehicles have access.

(1) One-lane slow point.

One-lane slow points restrict traffic flow to one lane. This lane must accommodate motor traffic in both travel directions. Passage through the slow point can be either straight through or angled.

Advantages:

- Vehicle speed reduced.
- Most effective when used in a series.
- Imposes minimal inconvenience to local traffic.
- Pedestrians have a reduced crossing distance, greater safety.

Disadvantages:

- Reduced sight distances if landscaping is not low and trimmed.
- Contrary to driver expectations of unobstructed flow.
- Can be hazardous for drivers and bicyclists if not designed and maintained properly.
- Opposing drivers arriving simultaneously can create confrontation.

(2) Two-lane slow point.

Two-lane slow points narrow the roadway while providing one travel lane in each direction.

Advantages:

- Only a minor inconvenience to drivers.
- Regulates parking and protects parked vehicles as the narrowing can help stop illegal parking.
- Pedestrian crossing distances reduced.
- Space for landscaping provided.

Disadvantages:

- Not very effective in slowing vehicles or diverting through traffic.
- Only partially effective as a visual obstruction.

Design Considerations:

- Where slow points have been used in isolation as speed control measures, bicyclists have felt squeezed as motorists attempt to overtake them at the narrowing. Not all bicyclists have the confidence to position themselves in the middle of the

road to prevent overtaking on the approach to and passage through the narrow area.

- To reduce the risk of bicyclists' being squeezed, slow points should generally be used in conjunction with other speed control devices such as speed tables at the narrowing. Slower moving drivers will be more inclined to allow bicyclists through before trying to pass. Where bicycle flows are high, consideration should be given to a separate right-of-way for bicyclists past the narrow area.
- A textured surface such as brick or pavers may be used to emphasize pedestrian crossing movement. Substituting this for the normal roadway surface material may also help to impress upon motorists that lower speeds are intended.
- Such measures should not confuse pedestrians with respect to the boundary of the roadway area over which due care should still be taken. In particular, where a road is raised to the level of the adjacent sidewalk, this can cause problems for those with poor sight. However, a tactile strip may help blind people in distinguishing between the roadway and the sidewalk; similarly, a color variation will aid those who are partially sighted.
- Slow points can be used to discourage use of the street by large vehicles. They can, however, be barriers to fire trucks and other emergency



This traffic-calming measure uses a landscaped median to narrow the travel lanes.

vehicles. Some designs permit access by emergency vehicles by means of lockable posts or ramped islands.

- Slow points can enhance the appearance of the street. For example, landscaped islands can be installed, intruding into the roadway to form a narrow “gate” through which drivers must pass. Landscaping enhances the neighborhood’s sense of nature and provides a visual break in views along the street.



This median provides a diagonal waiting area for bicyclists, including a railing to hold onto.

- Slow points are generally only sanctioned where traffic flows are less than 4,000 to 5,000 vehicles per day. Above this level, considerable delays will occur during peak periods.
- Clear signing should indicate traffic flow priorities.

Slow Point Examples:

Medians

Medians are islands located along the roadway centerline, separating opposing directions of traffic movement. They can be either raised or flush with the level of the roadway surface. They can be expressed as painted pavement markings, raised concrete platforms, landscaped areas, or any of a variety of other design forms. Medians can provide special facilities to accommodate pedestrians and bicyclists, especially at crossings of major roadways.

Design Considerations:

- Medians are most valuable on major, multi-lane roads that present safety problems for bicyclists and pedestrians wishing to cross. The minimum central refuge width for safe use by those with wheelchairs, bicycles, baby buggies, etc. is 1.6 meters (2 meters is desirable).
- Where medians are used as pedestrian and bicyclist refuges, internally illuminated bollards are suggested on the medians to facilitate quick and easy identification.

- Used in isolation, roadway medians do not have a significant impact in reducing vehicle speeds. For the purpose of slowing traffic, medians are generally used in conjunction with other devices, such as curb extensions or roadway lane narrowing.

Several caveats apply:

- To achieve meaningful speed reductions, the travel lane width reduction must be substantial and visually obvious. The slowing, however, is temporary; as soon as the roadway widens again, traffic resumes its normal speed.
- Bicyclists have been put at risk of being squeezed where insufficient room has been left between a central median and the adjacent curb. Experience shows that most drivers are unlikely to hold back in such instances to let bicyclists go through first. This threat is particularly serious on roads with high proportions of heavy vehicles.
- The contradiction between the need to reduce the roadway width sufficiently to lower motorist speeds, while at the same time leaving enough room for bicyclists to ride safely, must be addressed. This may be achieved by reducing the roadway width to the minimum necessary for a bicyclist and a motorist to pass safely (i.e., 3.5 meters).

There are three suggestions:

- Introducing color or texture changes to the road surface material around the refuge area reminds motorist that a speed reduction is intended.
- White striping gives a visual impression that vehicles are confined to a narrower roadway than that created by the physical obstruction — adjacent areas exist that vehicles can run over, but these are not generally apparent to approaching drivers.
- In some cases, provide an alternate, cut-through route for the bicyclists.

Curb Extensions

The sidewalk and/or landscaped area on one or both sides of the road is extended to reduce the roadway to a single lane or minimum-width double lane. By reducing crossing distances, sidewalk widening is used to facilitate easier and safer pedestrian movement.

Reducing roadway width results in vehicle speed reductions. When curb extensions are used at intersections, the resultant tightened radii ensure that vehicles negotiating the intersection do so at slow speeds.

Design Considerations:

- Can be installed either at intersections or mid-block.
- May be used in conjunction with other traffic-calming devices.
- Curb extensions are limited only to the degree that they extend into the travelway. Curb extensions cannot impede or restrict the operation of the roadway.
- Successful bicycle facilities need a clear separation from sidewalk and street pavement, with adequate distances from parked cars to avoid opening doors. Cross-traffic should be slowed to allow bicyclists better continuity and safety.
- Narrowing certain streets can, at the same time, create safer bicycle facilities, but care should be taken that bicyclists are not squeezed by overtaking vehicles where the road narrows. Encouraging motorists to let the bicyclists through first by using complementary traffic-calming techniques such as speed tables and cautionary signing or leaving sufficient room for both to pass safely at the narrowing would be appropriate measures.
- If it is expected that a motorist should be able to pass a bicyclist, the minimum desirable width is 3.5 meters.



A 7-foot radius allows for a slow and safe turn. As the radius increases, so does the speed of the vehicle.

- Curb extensions can be employed to facilitate bicycle movement where a segregated multi-use trail crosses a busy street.

Corner Radius Treatment

Corner radii of intersection curbs are reduced, forcing turning vehicles to slow down. Efforts to accommodate trucks and other large vehicles have historically led to increased corner radii at intersections.

The following results have been observed:

- Large vehicles (trucks, vans, etc.) turn the corners easily.
- Other vehicles turn faster than with a reduced radius corner.
- Pedestrian crossing distances are increased by up to 4 feet, depending on the radius.
- Pedestrian safety is decreased, due to higher speeds.
- The sharper turns that result from the reduced radii require motorists to reduce speed, increasing the time available to detect and take appropriate actions related to pedestrians at the crossing.

Advantage:

- Can result in increased safety for pedestrians by reducing crossing distances and slowing the speed of turning vehicles.

Disadvantages:

- May result in wide swings in turning movements of large vehicles.



The design of street closures should provide specific parking areas to discourage obstruction of bicycle and pedestrian traffic.

- May affect response times for emergency vehicles.

Design Consideration:

- To slow traffic, a corner radius of approximately 7 feet is recommended.

Narrow Traffic Lanes

Especially in residential areas, wide streets may not be necessary or desirable. Wide traffic lanes encourage faster motor vehicle speeds. Consideration should be given to the review of cross-sections for all street classifications to determine whether roadway lane widths can be reduced (within AASHTO guidelines) so more area can be dedicated to bicycle and pedestrian use and associated traffic-calming facilities.

Advantages:

- Additional area for landscaping, and pedestrian facilities.
- Reduced vehicle speeds and increased safety.

Disadvantage:

- On-street parking may be restricted.

Design Consideration:

- Cross-section approaching the reduced-width street should also be slowed.

Example: City of Portland, Skinny Street Program
The City of Portland requires most newly constructed residential streets to be 20 or 26 feet wide, depending

on neighborhood on-street parking needs. In the past, residential streets were required to be as wide as 32 feet. To achieve a variety of benefits, the City reduced residential street widths. The City's Fire Bureau participated in the development of this standard to ensure access for emergency vehicles.

3. Street closures.

Three types of street closures are described in the following discussion:

- Complete street closures.
- Partial street closures.
- Driveway links.

(Caution: Street closures must be considered in an area-wide context or traffic problems may simply shift to another nearby street).

Complete Street Closures

Street closures, generally on residential streets, can prohibit through-traffic movement or prevent undesirable turns. Street closures may be appropriate where large volumes of through-traffic or "short-cut" maneuvers create unsafe conditions in a residential environment.

Design Considerations:

- Where proposals are likely to lead to a reduction in access, prior consultation with residents at early stages of planning and design is especially important to minimize opposition.
- The benefits of exempting bicyclists should be carefully considered in all cases.
- Bicycle gaps should be designed to minimize the risk of obstruction by parked vehicles. Painting a bicycle symbol and other directional markings on the road in front of the bicycle gap has proven to be effective.
- Bollards can reduce the parking obstruction.
- Bollards should be lighted or reflectorized to be visible at night.

- The design of bicycle gaps should permit good visibility of adjacent roads.
- Signing should acknowledge the continued route as a through one for bicyclists.
- Clearly defined parking can reduce the problem of parked cars blocking the closure and bicycle gap.
- Police and fire departments should be consulted early in the design process to determine emergency access requirements. Often, removable bollards, crash gates, and card or key-operated gates can satisfy these requirements, combined with parking restrictions. A 20-foot-wide clear path is needed for emergency access.
- Tree planting, benches, and textured paving can enhance appearance.
- Street closures are recommended only after full consideration of all expected turning and reversing movements, including those of refuse trucks, fire trucks, and other large vehicles.

Partial Street Closures

Access to or from a street is prohibited at one end, with a no-entry sign and barrier restricting traffic in one direction. The street remains two-way, but access from the closed end is permitted only for bicyclists and pedestrians.

Design Considerations:

- Bicycle and pedestrian exemptions should be provided as a general rule, designed to minimize the likelihood of obstruction by parked vehicles.
- All signing should acknowledge the continued existence of the route as a through one for bicyclists and pedestrians.

Driveway Links

A driveway link is a partial street closure, where the street character is significantly changed so it appears to be a private drive. Typically, the

roadway is narrowed and defined with textured or colored paving. A ribbon curb or landscaping may be used to delineate roadway edges. “Reclaimed” roadway area is converted to pedestrian facilities and landscaping.

This is a very effective method of changing the initial impression of the street. If done right, drivers will not be able to see through. It appears as a road closure, yet allows through traffic.

The driveway link can provide access to small groups of homes and is especially applicable to planned residential developments. The “go slow” feel of the driveway link is enhanced by design standards that eliminate vertical curb and gutter and use a relatively narrow driveway cross-section. A ribbon curb may be used to protect roadway edges.

4. Traffic diversion.

Traffic diversion is one of the most widely applied traffic-calming concepts. It includes all devices that cause motor vehicles to slow and change direction to travel around a physical barrier. Physical barriers used to divert traffic in this fashion can range from traffic circles to trees planted in the middle of the road. The discussion that follows provides information on: traffic diverters, traffic circles, chicanes, and “tortuous” street alignments as traffic-calming devices.

Traffic Diverters

Traffic diverters are physical barriers installed at intersections that restrict motor vehicle movements in



Diagonal road closures/diverters limit vehicular access, but allow emergency vehicles to enter through removable bollards.

selected directions. The diverters may be designed to prevent right- or left-hand turns, to block straight-ahead travel and force turns to the right or left, or create a “T” intersection. In all cases, paths, cut-throughs, or other provisions should be made to allow bicyclists and pedestrians access across the closure.

Traffic diverters can take many forms. Here are two examples:

(1) Diagonal road closure/diversion.

Straight-through traffic movements are prohibited. Motorists are diverted in one direction only.

Advantages:

- Through-traffic is eliminated.



The splitter islands should be raised and landscaped to prevent left-turning vehicles from taking a short cut to avoid driving around the outside of the island.

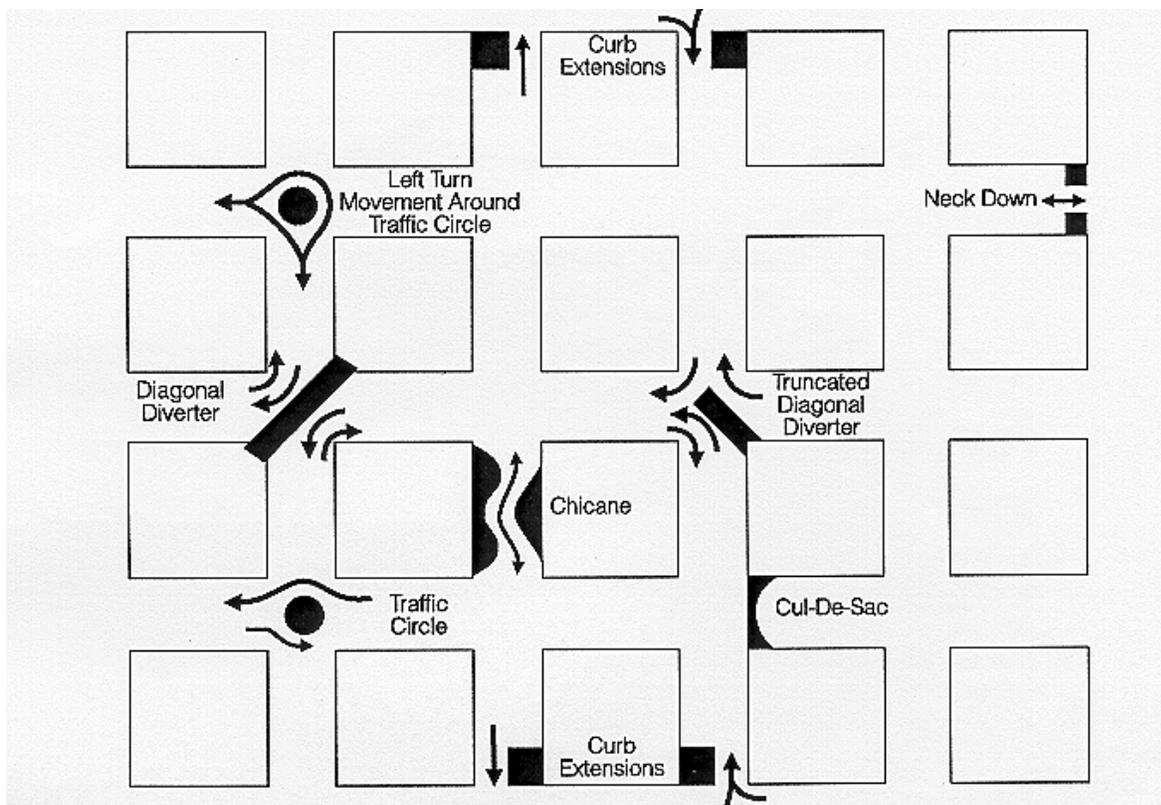
- Area for landscaping is provided.
- Conflicts are reduced.
- Pedestrian safety is increased.
- Can include a bicycle pathway connection.

Disadvantages:

- Will inconvenience residents in gaining access to their properties.
- May inhibit access by emergency vehicles unless street names are changed.
- Will move through traffic to other streets if not back to the arterial.

Example:

Eugene, Oregon has used diagonal diverters with positive community response. Eugene installs the



Example of an integrated traffic-calming plan.

diverters on a temporary basis to get neighborhood feedback before making a permanent installation. Two types of diagonal diverters are used — some are landscaped, while others are just guardrails. Both types have openings for bicycles. These have been supported by nearby residents.

Seattle installed truncated diagonal diverters, which allow right-turn movements around one end of the diverter. The Engineering Department found that these diverters were disruptive to neighborhood traffic and has focused instead on installation of traffic circles to control neighborhood traffic problems. Problems experienced with diverters included: (1) travel time and distance increased for all users; (2) local residents were diverted to other streets; (3) visitors and delivery services were often confused and delayed; and (4) emergency vehicle response times were potentially increased.

(2) Turning-movement diverters.

This type of diverter is designed to prevent cut-through traffic at the intersection of a neighborhood street with a major street or collector. It prevents straight-through movements and allows right turns only into and out of the neighborhood.

Advantages:

- Effective at discouraging cut-through traffic.
- Relatively low cost.
- Creates sense of neighborhood entry and identity.

Disadvantages:

- Limits resident access. Should be installed as part of overall neighborhood circulation improvements to ensure reasonable convenience for residents.
- Motorists may try to override the diverter to make prohibited turns unless vertical curbs, barriers, landscaping, or other means are used to discourage such actions.



Traffic circles can be designed to accommodate large vehicles and emergency access without undue restrictions.

Traffic Circles

Small traffic circles (center island approximately 4 meters in diameter) can be used as traffic-calming devices at intersections, reducing vehicle speeds. A roundabout is a channelized intersection at which all traffic moves counterclockwise around a central traffic island. These islands may be painted or domed, mountable elements may be curbed, and may include landscaping or other improvements.

Advantages:

- Crashes reduced by 50 to 90 percent when compared to two-way and four-way stop signs and other traffic signs by reducing the number of conflict points at intersections.
- Effective in reducing motor vehicle speeds. Success, however, depends on the central island being sufficiently visible and the approach lanes engineered to deflect vehicles, preventing overrun of the island. Overrunnable roundabouts on straight roads are less likely to produce the desired speed reduction.

Roundabout Accident Study

In 1989, a survey of crashes at mini-roundabouts examined years of crash data for 447 sites in England, Wales, and Scotland.

Key survey findings were:

- Mini-roundabouts were most commonly used on streets with speed limits of 30 mph or less.



Where possible, cyclists should be provided with cycle slips which enable them to bypass speed humps.

- Mini-roundabouts were found to have a far lower overall accident rate than that of signalized intersections with equivalent speed limits.
- Looking only at crashes involving bicycles, the study showed that four-arm mini-roundabouts have about the same involvement rate (accidents per million vehicles of that type entering the intersection) as do conventional, four-legged, signalized intersections.

Comparative Accident Rates:

Signalized intersections:

2.65 accidents/intersection/year
34 accidents per 100 million vehicles
20% resulted in serious or fatal injury

Roundabouts:

0.83 accidents/intersection/year
20 accidents per 100 million vehicles
19% resulted in serious or fatal injury

Both types of intersections compared have 30-mph speed limits and are four-legged intersections.

Splitter islands are the islands placed within a leg of the roundabout, separating entering and exiting traffic and designed to deflect entering traffic. They are designed to prevent hazardous, wrong-way turning movements.

These islands are important design elements and should be provided as a matter of routine, wherever feasible. Without splitter islands, left-turning

motorists have a tendency to shortcut the turn to avoid driving around the outside of the central island. The islands should, preferably, be raised and landscaped. If this is not possible, painted island markings should be provided.

Design Considerations:

- Roundabouts should preferably have sufficiently raised and highly visible centers to ensure that motorists use them, rather than overrunning.
- Clear signing is essential.

- Complementary speed reduction measures such as road humps on the approach to roundabouts can improve safety.
- The design of roundabouts must ensure that bicyclists are not squeezed by other vehicles negotiating the feature. Yet, where possible, adequate deflection must be incorporated on each approach to enforce appropriate entry speeds for motor vehicles.

Example: Seattle Neighborhood Traffic Control Program

The Seattle Engineering Department (SED) has experimented since the 1960's with a variety of neighborhood traffic control devices. The major emphasis of the SED Neighborhood Traffic Control Program is installing traffic circles (roundabouts) at residential street intersections. City staff report that about 30 circles are built each year. A total of approximately 400 circles have been installed to date. Each circle costs about \$5,000 to \$6,000.

In Seattle, a traffic circle is an island built in the middle of a residential street intersection. Each circle is custom-fitted to the intersection's geometry; every circle is designed to allow a single-unit truck to maneuver around the circle without running over it. A 2-foot concrete apron is built around the outside edge of the circle to accommodate larger trucks. Large trucks, when maneuvering around the circle, may run over the apron. The interior section of the circle is usually landscaped.

SED coordinates the design and construction of each circle with the Seattle Fire Department and school bus companies.

Traffic circles are installed at the request of citizens and community groups. Because there are more requests than funding to build them, SED has created a system for evaluating and ranking the requests. Before a request can be evaluated, a petition requesting a circle must be signed by 60 percent of the residents within a one-block radius of the proposed location. Then, the intersection's collision history, traffic volume, and speeds are studied.

Chicanes

Chicanes are barriers placed in the street that require drivers to slow down and drive around them. The barriers may take the form of landscaping, street furniture, parking bays, curb extensions, or other devices.

The Seattle Engineering Department has experimented with chicanes for neighborhood traffic control. It has found chicanes to be an effective means of reducing speed and traffic volumes at specific locations under certain circumstances. A demonstration project at two sets of chicanes showed:

- Reduction of traffic volumes on the demonstration streets.
- Little increase in traffic on adjacent residential streets.
- Reduced motor speeds and collisions.
- Strong support for permanent installation of chicanes by residents (68 percent).

Design Considerations:

- Consideration should be given to safe bicycle travel. Bicycle bypasses and signs to indicate directional priority are suggested.
- A reduction in sight lines should not be used in isolation to reduce speeds, as alone, this could be

potentially dangerous. A reduction in sight lines may be appropriate to avoid excessive land taking or as a reinforcing measure only where other physical features are employed that reduce speed.

- Chicanes offer a good opportunity to make environmental improvements through planting. However, preference should be given to low-lying or slow-growing shrubs to minimize maintenance and ensure good visibility.
- Measures should be employed to ensure that chicanes are clearly visible at night.
- Where full closure or speed humps are not feasible, chicanes may be used to reduce traffic speeds. Many different layouts are possible, including staggered parking (on alternating sides of the road).

Tortuous Roads

Roads can be designed to meander or jog sharply, slowing traffic and limiting views to discourage speeding. This technique can incorporate use of cul-de-sacs and courtyards.

Design Considerations:

Tortuous roads are generally planned as part of the design stage of a new road layout, rather than being superimposed on an existing layout. The siting of buildings is used to create a meandering road.



These pavement markings at a median refuge not only delineate the crossing for motorists, but also cue pedestrians about the location of the roadway edge.

- Designers should be aware of the need for accessibility to residential properties, both in terms of servicing and the needs of the individual. Tortuous roads will prove to be unpopular if they severely restrict accessibility.
- Where traffic is deliberately diverted onto a tortuous route — to avert town center congestion, for example — consideration should be given to maintaining as direct a route as possible for bicyclists.
- Tortuous roads (a.k.a. serpentine) are under study, but have not yet been approved for use in Portland. If approved, they would be limited for use on two-lane or fewer residential streets.
- Road design is limited by AASHTO standards for transition taper lengths.
- This traffic-calming device may require significant parking removal and should be used where parking removal is not an issue.

5. Surface texture and visual devices.

This category of traffic-calming devices includes signing, pavement marking, colored and textured pavement treatments, and rumble strips. These devices provide visual and audible cues about the traffic-calmed area. Colors and textures that contrast with those prevailing along the roadway alert



Pavement treatments can be applied to the entire traffic-calmed area or limited to specific street uses. The texture or color should be a noticeable contrast to the approaching roadways if speeds are to be reduced.

motorists to the need for alertness, much as conspicuous materials increase bicyclist and pedestrian visibility. Signs and pavement markings also provide information about applicable regulations, warnings, and directions.

Signing and Pavement Markings

Installation of directional, warning, and informational signs and pavement markings should conform to MUTCD guidelines, as applicable. Traffic-calming devices may be new to many people in the United States and the signs and markings will help minimize confusion and traffic conflicts.

Design Considerations:

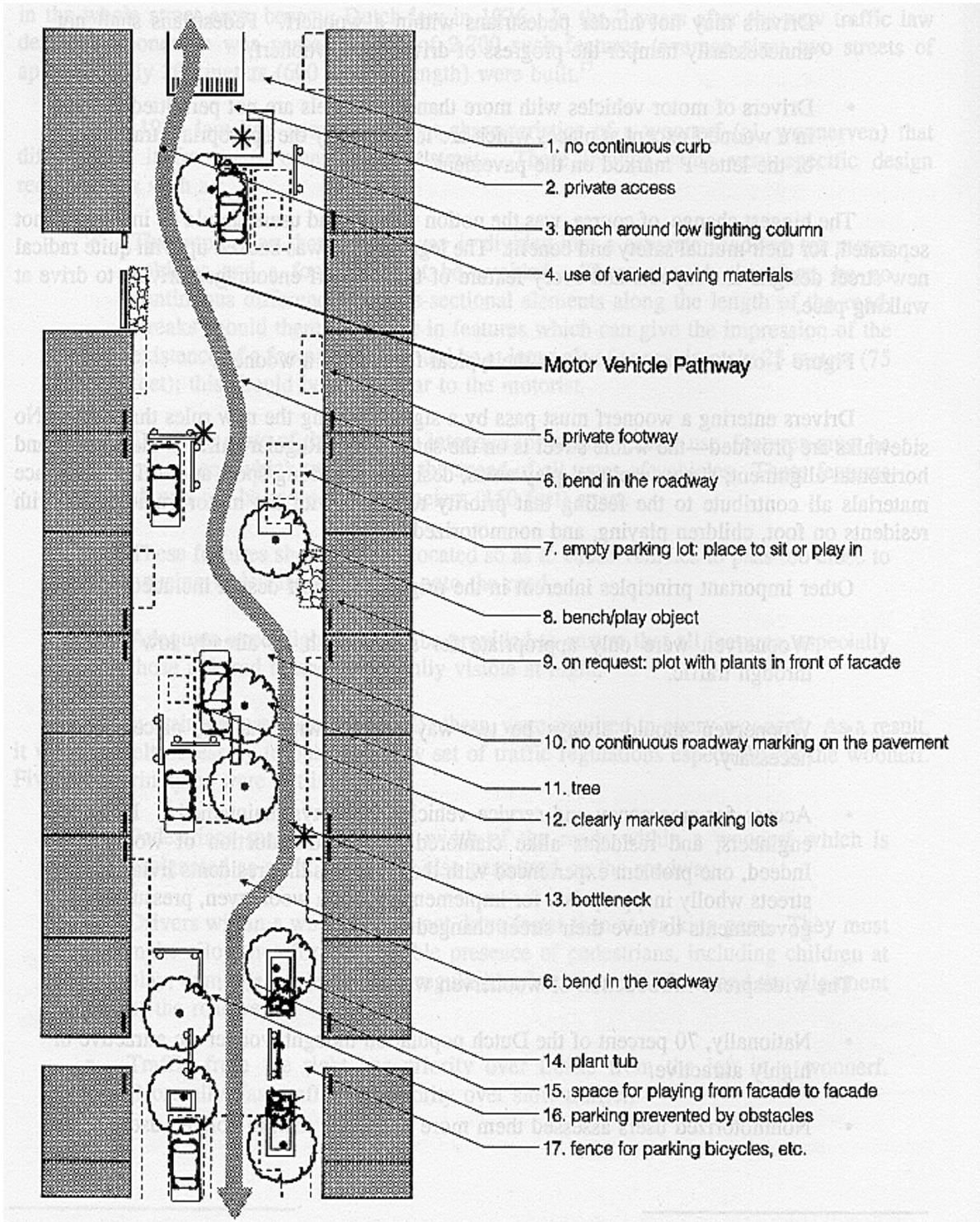
- A part of the sign/pavement marking approach to mitigating traffic in residential areas includes painting of stripes/lines on the roadway and other patterns that are designed to have a psychological impact on drivers. Although such patterns are basically intended to slow vehicles rather than reduce traffic, they should make passage over residential streets less desirable than if the roadway were untreated, in effect, encouraging the use of alternative routes.
- Many of the patterns tried have had only marginal success. In a few cases, the average speed increased slightly. A pattern that is successful is that of painting transverse bands.

Painted lines are applied to the road at decreasing intervals approaching an intersection or “slow-down” point. They are intended to give the impression of increasing speed and motorists react by slowing down.

Pavement Texturing and Coloring

The use of paving materials such as brick, cobbles, concrete pavers, or other materials that create variation in color and texture reinforces the identity of the area as a traffic-restricted zone.

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Model of a "woonerf."



The distinction between sidewalks and roadway is blurred in woonerfs.

1. The woonerf.

A woonerf (or “living yard”) combines many of the traffic-calming devices just discussed to create a street where pedestrians have priority and the line between “motor vehicle space” and “pedestrian (or living) space” is deliberately blurred (see the model of a woonerf). The street is designed so motorists are forced to slow down and exercise caution. Drivers, the Dutch say, do not obey speed limit signs, but they do respect the design of the street.

Design Considerations:

- The choice of materials should ensure that they do not pose a danger or deterrent to bicyclists. Cobbles present special difficulties, particularly for vehicles with narrow wheels and without the benefit of suspension. Such treatment is particularly discouraging for bicyclists on steep slopes, making it harder to maintain momentum when riding uphill. Thus, as a general rule, cobbles should not be employed. Similarly, pavers with chamfered edges impair a bicyclist’s stability and should be avoided.
- The color and texture of the street surface are important aspects of the attractiveness of many residential streets. The variation from asphalt or concrete paving associated by most people with “automobile territory” signals to the motorist that he or she has crossed into a different, residential zone where pedestrians and bicyclists can be expected to have greater priority.

Putting the Design Techniques to Work: Selected Examples of Traffic Calming

Most traffic-calmed streets utilize a combination of the devices just discussed. The following are some examples: the woonerf, entry treatments, shared streets, and other techniques (bicycle boulevards, modified street design, modified intersection design, channelization changes, traffic calming on a major road, slow streets, transit streets, and pedestrian zones).

The woonerf (plural — woonerven) is a concept that emerged in the 1970’s as increased emphasis was given by planners to residential neighborhoods. People recognized that many residential streets were unsafe and unattractive and that the streets, which took up a considerable amount of land area, were used for nothing but motor vehicle access and parking. Most of the time, the streets were empty, creating a “no-man’s land” separating the homes from one another.

The Dutch, in particular, experimented extensively with street design concepts in which there was no segregation between motorized and non-motorized traffic and in which pedestrians had priority.

A law passed in 1976 provided 14 strict “design rules” for woonerfs and resulted in construction of 2,700 such features in the following seven years.

The woonerven were closely evaluated, with the following findings:

- Injury accidents were reduced by 50 percent.
- Vehicle speeds were reduced to an average of 8 to 15 mph (13 to 25 km/h).
- Nationally, 70 percent of the Dutch population thought woonerven to be attractive or highly attractive.
- Non-motorized users assessed woonerven more positively than motorized users.

- Feedback from residents living on woonerfs was very positive. They appreciated the low traffic volumes and absence of cut-through traffic, but considered the larger play areas and other improvements to the street environment to be even more important benefits.

Woonerf Design Principles:

Following evaluation of the woonerven, the Dutch law was amended (July 1988) to allow greater design flexibility, replacing the design rules with six basic principles.

- (1) The main function of the woonerf shall be for residential purposes. Thus, roads within the “erf” area may only be geared to traffic terminating or originating from it. The intensity of traffic should not conflict with the character of the woonerf in practical terms: conditions should be optimal for walking, playing, shopping, etc. Motorists are guests. Within woonerven, traffic flows below 100 vehicles per hour should be maintained.
- (2) To slow traffic, the nature and condition of the roads and road segment must stress the need to drive slowly. Particular speed-reduction features are no longer mandated, so planners can utilize the most effective and appropriate facilities.
- (3) The entrances and exits of woonerven shall be recognizable as such from their construction. They may be located at an intersection with a major road (preferable) or at least 20 meters (60 feet) from such an intersection.
- (4) The impression shall not be created that the road is divided into a roadway and sidewalk. Therefore, there shall be no continuous height differences in the cross-section of a road within a woonerf. Provided this condition is met, a facility for pedestrians may be realized.

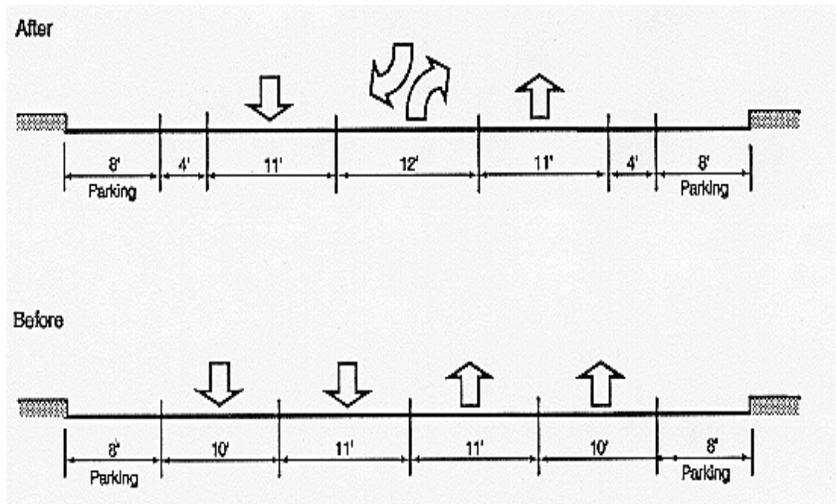
Thus, space can be designated for pedestrians and a measure of protection offered, for example, by use of bollards or trees.

- (5) The area of the road surface intended for parking one or more vehicles shall be marked at least at the corners. The marking and the letter “P” shall be clearly distinguishable from the rest of the road surface. In shopping street “erfs” (winkelerven), special loading spaces can be provided, as can short-term parking with time limits.
- (6) Informational signs may be placed under the international “erf” traffic sign to denote which type of “erf” is present.

2. Entry treatment across intersections.

Traffic-calming devices can be combined to provide an entry or “gateway” into a neighborhood or other district, reducing speed though both physical and psychological means. Surface alterations at intersections with local streets can include textured paving; pavement inserts; or concrete, brick, or stone materials. At the entry, the surface treatment can be raised as high as the level of the adjoining curb. Visual and tactile cues let people know that they are entering an area where motor vehicles are restricted.

Eugene, Oregon installs curb extensions at entrances to neighborhood areas, usually where a residential



The conversion of a 58-foot roadway. Elimination of one travel lane in each direction creates space for bicyclists.

street intersects an arterial. The curb extension is placed to prevent motor vehicle traffic from cutting through the neighborhood. The curb extension is signed as a neighborhood entrance or exit. Most of the street remains two-way, but one end becomes a one-way street. Compliance by motor vehicles is mostly good. Bikes are allowed to travel both ways at all curb extensions.

3. Bicycle boulevards.

The City of Palo Alto, California has moved beyond spot traffic-calming treatments and has created bicycle boulevards — streets on which bicycles have priority.

The purpose of a bicycle boulevard is to provide:

- Throughway where bicycle movements have precedence over automobiles.
- Direct route that reduces travel time for bicyclists.
- Safe travel route that reduces conflicts between bicyclists and motor vehicles.
- Facility that promotes and facilitates the use of bicycles as an alternative transportation mode for all purposes of travel.

The Palo Alto bicycle boulevard is a 2-mile stretch of Bryant Street — a residential street that runs parallel to a busy collector arterial. It was created in 1982 when barriers were fitted to restrict or prohibit through motor vehicle traffic, but to allow through bicycle traffic. In addition, a number of stop signs along the boulevard were removed. An evaluation after 6 months showed a reduction in the amount of motor vehicle traffic, a nearly twofold increase in bicycle traffic, and a slight reduction in bicycle traffic on nearby streets.

The City also found that anticipated problems failed to materialize and concluded that a predominately stop-free bikeway — on less traveled residential streets — can be an attractive and effective route for bicyclists. The bicycle boulevard bike traffic increased to amounts similar to those found on other established bike routes.

The bicycle boulevard continues to function as a normal local city street, providing access to residences, on-street parking, and unrestricted local

travel. The City received complaints about the visual appearance of the initial street closure barriers (since upgraded with landscaping), but is unaware of any other serious concerns of nearby residents.

Plans for the extension of the bicycle boulevard through downtown Palo Alto were approved by the City Council in the summer of 1992. Included in this extension was the installation of a traffic signal to help bicyclists cross a busy arterial.

4. Channelization changes.

The Seattle Engineering Department is changing some of its streets from four lanes to two lanes, with a center left-turn lane. These channelization changes can provide extra room for bicycle lanes or a wide lane for cars and bikes to share.

Numerous comments from users of some of those streets say motor vehicle speeds seem to have decreased. One street in particular, Dexter Avenue North, is a popular commuting route to downtown Seattle for bicyclists.

Traffic counts on the street show bicyclists make up about 10 to 15 percent of the traffic at certain times during the day. The rechannelization had little or no effect on capacity, reduced overtaking accidents, and made it easier for pedestrians to cross the street (by providing a refuge in the center of the road).

11.5 Exercise

Do one of the following exercises:

1. Choose a site-specific location (such as two to three blocks of a local street) where fast traffic or short cuts are a problem. Conduct a site analysis to determine problems. Prepare a detailed site solution that incorporates several traffic-calming devices. Illustrate with drawings and describe the anticipated changes in traffic speed.
2. Prepare a traffic-calming solution for an entire neighborhood or downtown area that illustrates an area-wide approach to slowing traffic. Conduct a site analysis to determine problem areas. Illustrate your solutions and describe the anticipated changes in traffic speed and flow.

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Abstract

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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 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

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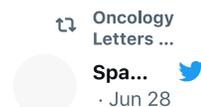
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The overall trend in Impact Factors for Spandidos Publications Journals featured in JCR over the course of the last 11 years is shown in the attached graphic.



Several Swiss citizens have brought to our attention that Associate Professor Martin Rösli 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 Rösli 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 Rösli, 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 Rösli, 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 Rösli 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 members

Factors for the Spandidos Publications journals that are indexed in JCR are shown in tabulated form in the attached graphic.



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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).

	<p>Table I.</p> <p>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).</p>
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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 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

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 Rössli (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/m² (Tables 5 and 7 in ref. no. 6) to 40 W/m² (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 electro-sensitive 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*

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, Emelie 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, Rööslö 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*

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.

'In 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.

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

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 Rösli *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 Rösli *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 Rösli 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 Rösli *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 Rösli *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 Rösli *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

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.

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 Rössli 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 foundations [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

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.

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Not applicable.

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Competing interests

The authors declare that they have no competing interests.

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COMMENT

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Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G

International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF)*

Abstract

In the late-1990s, the FCC and ICNIRP adopted radiofrequency radiation (RFR) exposure limits to protect the public and workers from adverse effects of RFR. These limits were based on results from behavioral studies conducted in the 1980s involving 40–60-minute exposures in 5 monkeys and 8 rats, and then applying arbitrary safety factors to an apparent threshold specific absorption rate (SAR) of 4W/kg. The limits were also based on two major assumptions: any biological effects were due to excessive tissue heating and no effects would occur below the putative threshold SAR, as well as twelve assumptions that were not specified by either the FCC or ICNIRP. In this paper, we show how the past 25 years of extensive research on RFR demonstrates that the assumptions underlying the FCC's and ICNIRP's exposure limits are invalid and continue to present a public health harm. Adverse effects observed at exposures below the assumed threshold SAR include non-thermal induction of reactive oxygen species, DNA damage, cardiomyopathy, carcinogenicity, sperm damage, and neurological effects, including electromagnetic hypersensitivity. Also, multiple human studies have found statistically significant associations between RFR exposure and increased brain and thyroid cancer risk. Yet, in 2020, and in light of the body of evidence reviewed in this article, the FCC and ICNIRP reaffirmed the same limits that were established in the 1990s. Consequently, these exposure limits, which are based on false suppositions, do not adequately protect workers, children, hypersensitive individuals, and the general population from short-term or long-term RFR exposures. Thus, urgently needed are health protective exposure limits for humans and the environment. These limits must be based on scientific evidence rather than on erroneous assumptions, especially given the increasing worldwide exposures of people and the environment to RFR, including novel forms of radiation from 5G telecommunications for which there are no adequate health effects studies.

Keywords: Federal Communications Commission (FCC), International commission on non-ionizing radiation protection (ICNIRP), Radiofrequency radiation (RFR), Exposure limits, Exposure assessment, Radiation health effects, Reactive oxygen species (ROS), DNA damage, 5G, Scientific integrity, Cell phone*, Mobile phone*

Introduction

In establishing exposure limits for toxic or carcinogenic agents, regulatory agencies generally set standards that take into account uncertainties of health risks for the general population [1] and for susceptible subgroups such as children [2]. That approach has not been applied in the same way to the setting of exposure limits for

*The terms cell phone and mobile phone are used interchangeably in this commentary; cell phone is the term used in the United States, while mobile phone is the term used in most of Europe.

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radiofrequency radiation (RFR) (frequency range: 3 kHz to 300 GHz). Moreover, assumptions underlying the current RFR exposure limits are flawed; hence, the limits that are currently applied do not adequately protect human and environmental health. This issue is discussed in greater detail under Assumption #9.

The Federal Communications Commission's (FCC) limits for maximum permissible exposure to RF electromagnetic fields (EMF) [3] were established in 1996 [4], and currently include many recommendations from the International Commission on Non-Ionizing Radiation Protection [5]. These exposure limits were expected to protect against adverse health effects in humans that might occur from short-term (i.e., acute) exposures to RFR and have been maintained by the FCC for the past 26 years. The exposure limits that were established by the FCC in 1996 relied on criteria recommended by the National Council on Radiation Protection & Measurements (NCRP) [6] and the Institute of Electrical and Electronics Engineers (ANSI/IEEE) [7, 8]. The limits were "based on a determination that potentially harmful biological effects can occur at a SAR (specific absorption rate) level of 4.0 W/kg as averaged over the whole-body." The SAR is a measure of the rate of RF energy absorbed per unit mass.

The threshold for a behavioral response and for acute thermal damage in sensitive tissues was considered to be an exposure that produced a whole-body SAR greater than 4 W/kg. In parallel with the development of the FCC's RFR exposure limits, ICNIRP's guidelines for limiting exposure to RF-EMF were also based on behavioral studies conducted in rats and monkeys in the 1980s [9].

The harmful effects that served as the basis for the exposure criteria were changes in behavior observed in small numbers of rats and monkeys when exposed to RFR for up to 60 minutes to power densities at which the whole-body SAR was approximately 4 W/kg or higher [10, 11]. Those studies were conducted in the early 1980s (1980 and 1984, respectively) by investigators of the US Navy Department. Consequently, 4 W/kg was identified as the threshold SAR for adverse health effects induced by RFR. In food-deprived monkeys that were exposed to three different frequencies (225 MHz, 1.3 GHz, and 5.8 GHz) during 60-min sessions, lever-pressing response rates for the delivery of food pellets were reduced compared to sham exposure sessions. The threshold SAR for this decreased response was reported to range from 3.2 to 8.4 W/kg [11]. Similarly, in food-deprived rats exposed to 40-min sessions at 1.28 or 5.62 GHz radiation, the threshold SAR for a decrease in response rate was reported to range from approximately 3.8 to 4.9 W/kg [10]. In experimental studies in which monkeys were exposed in an anechoic chamber for 4 hours to 1.29 GHz

radiation at various power densities, an increase in mean body temperature of 0.7°C was associated with a whole-body SAR of 4 W/kg [12]. Behavior disruption associated with an increase in body temperature of approximately 1.0°C was assumed to be the most sensitive measure of harmful effects from RF-EMF exposure.

After establishing 4 W/kg as the threshold dose for acute harmful effects, both the FCC [3, 4] and ICNIRP [5, 9] set exposure limits for controlled occupational exposures to 0.4 W/kg SAR averaged over the whole body (based on applying a 10-fold safety/uncertainty factor). For the general population, the FCC's and ICNIRP's exposure limits were set at 0.08 W/kg SAR averaged over the whole body (by applying an additional 5-fold safety/uncertainty factor) for frequencies between 3 MHz and 3 GHz. The exposure limits established by the FCC and ICNIRP do not account for any impact of differing signal characteristics, such as carrier wave modulations or pulsing of the signal. Whole-body exposures for the general population are based simply on power levels averaged over 30-minute periods [3, 5].

Based on SAR distributions from whole-body exposures in which local (i.e., partial body) SARs were estimated to be 10 to 20 times the average value, local exposure limits were set 20 times higher than the average whole-body exposure limit [4–7]. For occupational exposures, local peak exposure limits were permitted up to 8 W/kg averaged over any 1-g cube of tissue [4] or 10 W/kg averaged over any 10 g of contiguous tissue [9] by the FCC and ICNIRP, respectively. For the general population, local peak SARs for partial-body exposures were not to exceed 1.6 W/kg averaged over any 1 g of cube-shaped tissue [3], or not to exceed 2.0 W/kg averaged over any 10 g of cube-shaped tissue [5]. Higher limit values are permissible for extremities. Extremities include the hands, wrists, feet, ankles, and pinnae (the external part of the ear), despite the close proximity of the ear to the brain. These adjustments were made long before the widespread use of wireless communication devices in which the emitting antenna is typically held close to local body organs such as the brain. The NCRP document [6] acknowledges that exposures could be greater than the recommended safety limit values when people are in close proximity to emitters of RFR.

The setting of exposure limits for the prevention of excessive tissue heating was based on the following assumptions: 1) electromagnetic waves at frequencies used in wireless communications do not have sufficient energy to break chemical bonds or ionize molecules [13]; 2) RFR could not damage DNA; and 3) tissue heating was the only possible biological effect of nonionizing radiation [5, 9, 14–16]. For potential environmental and human health issues that are not addressed in the

A) Effects of RF radiation at exposures below the putative threshold SAR of 4 W/kg

Assumption 1) There is a threshold exposure for any adverse health effect caused by RF radiation; in the frequency range of 100 kHz to 6 GHz it is a whole-body exposure that exceeds an SAR of 4 W/kg. Any biological effect of RF radiation above the threshold exposure is due to tissue heating.

Assumption 2) RF radiation is incapable of causing DNA damage other than by heating; there is no mechanism for non-thermal DNA damage.

Assumption 3) Two to seven exposures to RF radiation for up to one hour duration are sufficient to exclude adverse effects for any duration of exposure including chronic exposures.

Assumption 4) No additional effects would occur from RF radiation with co-exposure to other environmental agents.

B) Factors affecting dosimetry

Assumption 5) Health effects are dependent only on the SAR value; carrier wave modulations, frequency, or pulsing do not matter except as they influence the SAR.

C) Human brain cancer risk

Assumption 6) The multiple human studies that find associations between exposure to cell phone RF radiation and increases in brain cancer risk are flawed because of biases in the published case-control studies, and because brain cancer rates have remained steady since the time that use of wireless communication devices became widespread.

D) Individual variations in exposure and sensitivity to RF-EMF

Assumption 7) There are no differences among individuals, including children, in the absorption of RF-EMF and susceptibility to this radiation.

Assumption 8) There are no differences among individuals in their sensitivity to RF radiation-induced health effects.

E) Applied safety factors for EMF-RF workers and the general population

Assumption 9) A 50-fold safety factor for whole body exposure to RF radiation is adequate for protecting the general population to any health risks from RF radiation.

Assumption 10) A 10-fold safety factor for whole body exposure to RF radiation is adequate for protecting workers to any health risks from RF radiation.

Assumption 11) Exposure of any gram of cube-shaped tissue up to 1.6 W/kg, or 10 grams of cube-shaped tissue up to 2 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in the general population.

Assumption 12) Exposure of any gram of cube-shaped tissue up to 8 W/kg, or 10 grams of cube-shaped tissue up to 10 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in workers.

F) Environmental exposure to RF radiation

Assumption 13) There is no concern for environmental effects of RF radiation or for effects on wildlife or household pets.

G) 5G (5th generation wireless)

Assumption 14) No health effects data are needed for exposures to 5G; safety is assumed because penetration is limited to the skin ("minimal body penetration").

Fig. 1 Assumptions Underlying the FCC/ICNIRP Exposure Limits for RF Radiation

setting of exposure limits (for example effects of chronic exposures, or effects of co-exposure of skin to RFR and other environmental agents, such as would occur with 5G exposure in combination with sunlight), the implicit assumption is that such effects do not matter, or that the arbitrarily selected safety/uncertainty factor is sufficient to deal with those concerns. In any case, it is expected that underlying assumptions applied to health risk assessments would be clearly described [1].

Exposure limits for RF radiation are based on numerous assumptions; however, research studies published over the past 25 years show that most of those assumptions are not supported by scientific evidence. In the NCRP report [6], the authors noted that when further understanding of biological effects of RF radiation becomes available, exposure guidelines will need to be evaluated and possibly revised. The ANSI/IEEE document [7] also notes that effects of chronic exposure or evidence of non-thermal interactions could result in revising exposure standards. Unfortunately, these recommendations were never implemented. Assumptions of

safety from exposures that could adversely affect human or environmental health should be tested and validated *before* widespread exposures occur, not afterwards, by agencies responsible for protecting public health.

In this paper, we highlight studies that demonstrate the fallacy of inherent assumptions in the FCC/ICNIRP guidelines for RF radiation exposure limits, and we find that the limits fail to protect human and environmental health. Fourteen assumptions that underlie the RFR exposure limits established in the 1990s and reaffirmed in 2020 by the FCC [4, 5] and ICNIRP [5, 9] are addressed in this paper and are shown in Fig. 1.

Assumptions underlying exposure limits for RF radiation and the scientific evidence demonstrating that these assumptions are not valid

A. Effects of RF radiation at exposures below the putative threshold SAR of 4W/kg

Assumption 1) *There is a threshold exposure for any adverse health effect caused by RF radiation; in the*

frequency range of 100 kHz to 6 GHz it is a whole-body exposure that exceeds an SAR of 4 W/kg. Any biological effect of RF radiation above the threshold exposure is due to tissue heating.

Cardiomyopathy and carcinogenicity

In response to a request from the Food and Drug Administration's (FDA) Center for Devices and Radiological Health [17], the National Toxicology Program (NTP) conducted toxicity and carcinogenicity studies of cell phone (CDMA- or GSM-modulated) radiation in rats and mice exposed to RFR at frequencies of 900 MHz and 1800 MHz, respectively [18, 19]. Exposures to RFR for up to 2 years occurred in reverberation chambers over 18 hours/day on a continuous cycle of 10 minutes on and 10 minutes off. In rats, the whole-body SAR levels during the 10-minute on cycles were 0, 1.5, 3, or 6 W/kg.

The major histopathological findings from the NTP study in male rats [18] included dose-related increases in cardiomyopathy, increased incidence of cancers and preneoplastic lesions in the heart (schwannoma and Schwann cell hyperplasia) and brain (glioma and glial cell hyperplasia), increases in prostate gland tumors and hyperplasias, significant increases in adrenal gland tumors, and significant increases in the overall incidence of benign or malignant neoplasms in all organs in the 3 W/kg groups. The incidence of cardiomyopathy was also increased in GSM-exposed female rats, and significant increases in DNA damage were found in rats and mice [18, 19]. Similarly, an earlier study by Chou et al. [20] found a significant (3.6-fold) increase in the incidence of primary malignant neoplasms in male rats exposed to 2450 MHz pulsed RFR for 25 months (21.5 hr./day) at an SAR that ranged from 0.15 to 0.4 W/kg.

A 3-day external peer-review of the NTP studies confirmed there was "clear evidence of carcinogenic activity" in male rats for heart schwannomas, and "some evidence of carcinogenic activity" for brain gliomas and adrenal gland tumors with exposure to either GSM- or CDMA-modulated RF radiation [21]. In addition, a lifetime study by the Ramazzini Institute reported a significant increase in heart schwannomas in male rats exposed 19 hour/day to 1800 MHz GSM-modulated RFR at a field strength of 50 V/m, equivalent to a whole-body SAR of 0.1 W/kg [22]. The incidence of heart Schwann cell hyperplasia was also increased in that exposure group. These findings are consistent with results from the NTP study and demonstrate that the proliferative effect of modulated RFR in heart Schwann cells is a reproducible finding that can occur at doses far below the assumed whole-body threshold SAR of 4 W/kg.

ICNIRP [23] dismissed the evidence of carcinogenicity for RFR that was provided in the studies by the NTP [18] and the Ramazzini Institute [22] based on their earlier critique of those studies [24]. However, that critique demonstrated an unfortunate lack of understanding together with a misrepresentation of the design, conduct, and interpretation of experimental carcinogenicity studies in animal models [25], as well as a lack of appreciation for the remarkable concordance between the tumor responses observed in experimental animals with those identified in cancer epidemiology studies of mobile phone users described under Assumption #6.

Neither heating effects nor thermal stress was likely causal of the adverse health effects observed in the NTP [18] study, since there was no tissue damage observed in a 28-day study at the same SARs, there was no significant effect on body weight during the 2-year study, and there were no exposure-related clinical observations that would indicate thermal or metabolic stress. Furthermore, a preliminary thermal pilot study demonstrated that body temperatures did not increase by more than 1°C at the exposure levels used in the chronic studies [26], and there is no evidence that a small change in body temperature associated with the RFR exposures in the NTP study can cause the types of carcinogenic effects that were observed. The similar findings of GSM-modulated RFR on Schwann cells by the Ramazzini Institute [22] at much lower whole-body SARs confirm these effects to be independent of tissue heating.

Neurological effects

Though the FCC and ICNIRP exposure limits are based on a putative threshold dose of 4 W/kg due to behavioral disruption observed at higher doses in rats and monkeys [10, 11] numerous studies have shown consistent and reproducible deficits in spatial learning and memory in laboratory animals exposed to RF radiation at SARs below 4 W/kg. Examples of study exposures that demonstrated these neurological effects included 900 MHz GSM at 0.41–0.98 W/kg, 2 hr./day for 4 days in mice [27]; 900 MHz GSM at 0.52–1.08 W/kg, 2 hr./day for 1 month in rats [28]; 900 MHz GSM at 1.15 W/kg, 1 hr./day for 28 days in rats [29]; 900 MHz pulsed RFR at 0.3–0.9 W/kg for 6 hr./day in rats from conception to birth and tested at 30 days of age [30]; 900 MHz GSM and 1966 MHz UMTS at 0.4 W/kg for 6 months in rats [31]; and 900 MHz continuous wave EMF at 0.016 W/kg 3 hr./day for 28 days in rats [32]. The studies cited above are not the only studies showing these effects, but they clearly demonstrate that exposure to RFR at an SAR of 4 W/kg is not a threshold dose for neurological effects in rodents. The effects of RF radiation on spatial learning and memory indicate

the hippocampus as a target site of these exposures. For a more complete listing of neurological effects of RFR reported between 2007 and 2017 see Lai [33].

In addition, many studies have reported changes in brain electrical activities in human subjects, measured by electroencephalography (EEG), including sleep disturbance from single exposures to cell phone RF radiation. This is not surprising since the nervous system transmits messages based on electrical signals generated by nerve cells. Decreased β -trace protein, which is a key enzyme in the synthesis of a sleep-promoting neurohormone, has been seen in young adults with high-cumulative amounts of hours of mobile phone use [34]. Another frequently reported effect of RF radiation is increased blood-brain barrier permeability in rats at SARs much lower than 4 W/kg, e.g. [32, 35–41]. Oxidative stress induced in the brain of animals exposed to RF-EMF has been associated with observed neurological effects [42]. Although many studies did not observe significant changes in neurological effects in humans and several studies did not observe increased permeability in the blood-brain barrier in animal models [33], differences in EMF frequency, modulation, duration of exposure, and direction of incident waves to the exposed subject, as well as difference in dielectric properties and the size and shape of the exposed subject likely account for differences in observed effects [43, 44].

Sperm damage

The effect of non-ionizing microwave radiation on the testis (testicular degeneration in mice) was first reported 60 years ago [45]. Since then, and with the rapid increase in use of RF-EMF emitting devices, numerous studies have investigated testicular effects of RFR and potential associations with male infertility [46–50]. Human and animal studies have shown that the testis is one of the most sensitive organs to RF-EMF exposures, and that keeping a mobile phone in trouser pockets in talk mode can affect fertility parameters e.g., sperm motility, sperm count, sperm morphology, and apoptosis [48, 51]. Meta-analyses of published epidemiologic studies on the impact of mobile phone radiation on sperm quality in adult men have found significant decreases in sperm motility, sperm viability and/or sperm concentrations that were associated with mobile phone usage [52–55]. Several physical factors associated with exposure conditions can affect the outcome of human studies, including depth of energy penetration, duration of call, type of transmission technology, distance of the device to the body or testis, and power density with defined SAR. For example, Zilberlicht et al. [56] observed higher rates of

abnormal sperm concentrations among men who held their phones less than 50 cm from their groin.

The effects of RFR on reproductive parameters in humans are consistent with results from experimental studies in animals and in vitro studies. For example, exposure of human semen to 850 MHz radiation from mobile phones for 1 hour at an SAR of 1.46 W/kg caused a significant decrease in sperm viability that was associated with an increase in reactive oxygen species (ROS) [50] or an increase in sperm DNA fragmentation [57]. Exposure of isolated human spermatozoa to 1.8 GHz RF-EMF significantly reduced sperm motility and induced ROS generation at an SAR of 1.0 W/kg, and significantly increased oxidative DNA damage and DNA fragmentation at an SAR of 2.8 W/kg [58].

Some examples of effects of RFR on male fertility factors in studies with experimental animals at SARs below 4 W/kg include: a decrease in sperm count and an increase in ROS in rats exposed to mobile phone frequencies 2 hr./day, for 35 days (SAR=0.9 W/kg) [59]; increases in oxidative stress, 8-hydroxy-deoxyguanosine (8-OHdG), and DNA strand breaks in the testes of rats exposed to 900 MHz (SAR=0.166 W/kg), 1800 MHz (0.166 W/kg), or 2100 MHz (0.174 W/kg) 2 hr./day for 6 months [60]; an increase in ROS, a decrease in sperm count, and altered sperm morphology in rats exposed to 900 MHz 3G mobile phone radiation (SAR=0.26 W/kg) 2 hr./day for 45 days [61]; decreased sperm quality in rats in which local exposure of the scrotum to 2575–2635 MHz 4G smartphone time division LTE radiation occurred for 1 min over 10 min intervals 6 hr./day for 150 days [62]; impaired testicular development at 35 days of age in male offspring of pregnant rats that were exposed to 2.45 GHz RFR (SAR=1.75 W/kg) 2 hr./day throughout pregnancy [63]; decreased sperm motility in mice exposed to 905 MHz RFR (SAR=2.2 W/kg) 12 hr./day for 5 weeks, and increased ROS formation and DNA fragmentation after 1 week of exposure [64]. Although negative studies have also been reported, it is important to remember that the outcome of experimental studies can be affected by differences in exposure conditions, including the frequency, modulation, polarization, stray electromagnetic fields, local SAR, duration of exposure, and analytical methods [43, 44].

Although the mechanism of testicular effects from exposure to non-thermal levels of RFR is not fully known, numerous studies in rats and mice, and in human sperm have found associations between negative effects on fertility parameters and increases in ROS and/or DNA damage [48, 51, 57, 58, 60, 61, 64–68]. Thus, the adverse effects of RFR on sperm quality are likely due in large part to induced generation of ROS.

Assumption 2) *RF radiation is incapable of causing DNA damage other than by heating; there is no mechanism for non-thermal DNA damage.*

In 2009, ICNIRP [16] claimed that “low energy photons of RF radiation are too weak to affect ionization or cause significant damage to biological molecules such as DNA, under ordinary circumstances.” However, DNA damage and other genotoxic effects have been observed in numerous studies of low intensity RFR in animal models and in humans. For example, the NTP study found statistically significant increases in DNA damage in brain cells of exposed rats and mice compared to sham controls [18, 19, 69], and Akdag et al. [70] found statistically significant increases in DNA damage in hair cells in the ear canal among 30 to 60 year-old men who used mobile phones for 10 years for 0–30 min/day, 30–60 min/day, or greater than 60 min/day compared to people who did not use mobile phones. In the latter study, the extent of DNA damage increased with increasing daily exposure duration. In a review of published studies on genetic effects of ELF- and RF-EMF, Lai [71] listed more than 150 studies in which non-thermal exposures to RFR produced increases in DNA damage, chromosome aberrations, or micronuclei formation.

In addition, it is well established that DNA damage can also be caused by indirect processes, such as by the generation of reactive oxygen species (ROS), and numerous studies have demonstrated DNA damage at exposures below the putative threshold SAR of 4 W/kg. More than 120 published studies have demonstrated oxidative effects associated with exposure to low intensity RFR (Additional file 1: Appendix 1). An analysis of experimental studies on molecular effects of low intensity RF radiation (RFR) in biological systems found that the majority (93 of 100 studies) demonstrated the induction of oxidative effects [72]. More recent studies (from 2017) revealed that all 30 relevant publications (100%) detected significant oxidative effects under low intensity RFR exposures, and most of these studies used modulated RFR from wireless communication devices.

Increased production of ROS in living cells may be caused by weak magnetic fields altering recombination rates of short-lived radical pairs generated by normal metabolic processes leading to changes in free radical concentrations [73], or by low intensity extremely low frequency (ELF) EMFs resulting in alterations in voltage-gated ion channels in cell membranes causing changes in cation flow across membranes [74]. These mechanisms apply to both ELF-EMFs and to RFR modulated by pulsed fields at extremely low frequencies. Other biophysical mechanisms by which non-thermal RF-EMF can

cause biological effects through interactions with normal cellular processes have been described [75].

Increasing NADH oxidase activity is another mechanism by which RFR can increase ROS production. NADH oxidases, which are membrane-associated enzymes that catalyze one-electron reduction of oxygen to superoxide radical using NADH as the electron donor, have been identified as primary mediators of RFR interactions in cellular systems [76]. A significant (3-fold) increase in the activity of NADH oxidase was measured in purified plasma membranes from HeLa cells exposed to 875 MHz for 5 or 10 min at a power density of 200 $\mu\text{W}/\text{cm}^2$. This exposure intensity is significantly lower than the ICNIRP [5] safety limit.

The major source of ROS in living cells is the mitochondrial electron transport chain, where leakage of electrons generates superoxide radicals due to the partial reduction of oxygen [77]. A dose-dependent effect of 1.8 GHz modulated RFR exposure (SAR=0.15 and 1.5 W/kg) on mitochondrial ROS production was detected in mouse spermatogonial germ cells [65]. Exposure of quail embryos to extremely low intensity modulated RFR (GSM 900 or 1800 MHz, 0.25 or 0.32 $\mu\text{W}/\text{cm}^2$) during the initial days of embryogenesis resulted in a robust overproduction of superoxide radical and nitrogen oxide in mitochondria of embryonic cells [78, 79]. Thus, multiple mechanisms for the increased production of ROS by low intensity RF radiation have been demonstrated.

Numerous studies have been published on mutagenic effects of low intensity RF-EMFs, especially studies that identified increases in levels of a specific marker of oxidative DNA damage and a risk factor for cancer, 8-hydroxy-2'-deoxyguanosine (8-OHdG) [58, 60, 78–84]. For example, the level of 8-OHdG in human spermatozoa was increased significantly after *in vitro* exposure for 16 hr. to 1.8 GHz at a power level of 2.8 W/kg and correlated with levels of ROS generation [58]. Likewise, exposure of quail embryos *in ovo* to GSM-modulated 900 MHz of 0.25 $\mu\text{W}/\text{cm}^2$ for 1.5, 5, or 10 days was sufficient to produce a significant, two-threefold, increase in 8-OHdG levels in embryonic cells [79]. Umbilical cord blood and placenta tissue samples obtained after delivery from women who used mobile phones during pregnancy had significantly higher levels of oxidative stress parameters, including 8-OHdG and malondialdehyde, compared to cord blood and placental tissue from women who did not use mobile phones during pregnancy [85]. In addition, DNA damage, analyzed by the comet assay, was increased significantly in cord blood lymphocytes obtained from women who used mobile phones during pregnancy compared to cord blood lymphocytes obtained from women who did not use mobile phones.

As low intensity RF radiation does not have sufficient energy to ionize DNA molecules, and as increased production of ROS in living cells due to RF-EMF exposures has been reliably documented, an indirect effect of this type of radiation is the formation of oxidative damage to DNA. The most aggressive form of ROS that can cause oxidative DNA damage is the hydroxyl radical; this reactive oxygen species can be generated from superoxide radical and hydrogen peroxide [86], which may be produced in living cells exposed to low intensity RF radiation. Ultraviolet radiation (UVR, encompassing UVA, UVB, and UVC), which is classified by IARC as “carcinogenic to humans”), can also cause indirect DNA damage by generating ROS [87]. Thus, both RFR and UVR, which can similarly induce oxidative DNA damage, can increase cancer risk by a similar mechanism.

Increased production of ROS and depletion of antioxidant capacity in living cells exposed to low intensity RF radiation can result in oxidative DNA damage. Induction of oxidative stress, which is a key characteristic of many human carcinogens [88], including UVR and asbestos, can also lead to genotoxicity and carcinogenicity of non-ionizing RF radiation without causing direct DNA damage.

Assumption 3) *Two to seven exposures to RF radiation for up to 1 hour duration are sufficient to exclude adverse effects for any duration of exposure including chronic exposures.*

The behavioral studies in 8 male rats and 5 male monkeys that served as the basis for the exposure limits to RF radiation adopted by the FCC and ICNIRP involved 2 to 7 exposure sessions of 40-minute duration for rats [10] and 3 exposure sessions of 60-minute duration for monkeys at each power density [11]. Additional support for the threshold SAR of 4 W/kg in the frequency range of 100 kHz to 6 GHz came from behavioral studies conducted in rats and monkeys by D’Andrea et al. [89, 90]. However, D’Andrea et al. [91, 92] also reported that exposure of rats to continuous wave 2450 MHz RFR for 14 or 16 weeks caused significant differences in behavioral activity between sham-exposed rats and RFR-exposed rats at mean SARs of 0.7 W/kg and at 1.23 W/kg, indicating that 4 W/kg is not a threshold SAR with extended exposure durations. Since that time many studies have shown that responses to non-thermal RFR depend on both exposure intensity and exposure duration [93]. Importantly, the same response was observed with lower exposure intensity but prolonged exposure duration as at higher exposure intensity and shorter duration [94].

Recognizing that the exposure limits do not address potential health effects after long-term exposures to

RF radiation emitted from wireless devices that people are experiencing, the FDA [17] nominated RF radiation to the NTP for chronic toxicology and carcinogenicity studies out of concern that “existing exposure guidelines are based on protection from acute injury from thermal effects of RFR exposure, and may not be protective against any non-thermal effects of chronic exposures.” Adverse health effects noted in Assumption #1, including cardiomyopathy, carcinogenicity, sperm damage, and neurological effects, as well as the human epidemiology studies to be described in Assumption #6, occurred with much longer exposures to RF radiation than the exposure durations used in the acute studies in rats [10] and monkeys [11]. Consequently, the acute behavioral exposure studies that served as the basis for exposure limits to RF radiation established by the FCC and ICNIRP are inadequate to identify and characterize adverse effects of RF radiation after longer exposure durations. Neither the exposure limits established in the 1990s by the FCC [4] or by ICNIRP [9], nor those reaffirmed more recently by these groups [3, 5] address health risks associated with long-term exposure to RF radiation.

Assumption 4) *No additional effects would occur from RF radiation with co-exposure to other environmental agents.*

The current FCC/ICNIRP exposure limits do not take into consideration interactive effects of RF radiation with other environmental agents even though such effects have been documented. Interactions of RF radiation with other agents may result in antagonistic or synergistic effects, i.e., effects that are greater than the sum of each agent alone.

In the International Agency for Research on Cancer (IARC) evaluation of the carcinogenicity of RF-EMF [44], the expert working group noted that 4 of 6 co-carcinogenesis studies available at that time showed increased responses with exposure to RF-EMF. One of those studies reported co-carcinogenic effects of UMTS-modulated RF radiation at 4.8 W/m² in the liver and lung of mice that had been treated with the carcinogen ethylnitrosourea (ENU) in utero [95]; the incidence of liver and lung cancers were increased in mice exposed to ENU plus RF radiation compared to cage controls, sham controls and ENU alone. After the IARC evaluation, Lerchl et al. [96] replicated the experimental design of Tillmann et al. [95] by exposing mice to RF-EMF at whole-body SAR levels of 0 (sham), 0.04, 0.4, and 2 W/kg. Significant increases in lung adenomas and/or liver carcinomas were observed at all exposure levels. Lerchl et al. [96] concluded that their “findings are a very clear indication that tumor-promoting effects

of life-long RF-EMF exposure may occur at levels supposedly too low to cause thermal effects.” Thus, the reproducibility of the tumor-promoting effects of RFR at non-thermal exposure levels has been demonstrated.

Other examples of reported synergistic effects include the following study results. Synergistic effects on damage to human lymphocytes were observed with co-exposure to RFR (1.8 GHz RFR, SAR 3 W/kg) and 2 different mutagens, namely, mitomycin C or 4-nitroquinoline-1-oxide [97], or with co-exposure to ultraviolet (UVC) light [98]. A synergistic effect was found on DNA damage in human blood cells exposed to 2450 MHz radiation (5 mW/cm²) and then exposed to mitomycin C [99]. A potentiation effect on DNA damage was observed in cultured mammalian cells exposed to CDMA-modulated 835 MHz RF-EMF (SAR = 4 W/kg) and the clastogens cyclophosphamide or 4-nitroquinoline-1-oxide [100]. Gene expression was altered in neuronal and glial cells of rats pre-treated with lipopolysaccharide, a neuroinflammatory agent, and then exposed to 1800 MHz GSM modulated radiation (SAR = 3.22 W/kg) for 2 hr. [101]. In rats pre-treated with picrotoxin, a chemical that induces seizures, exposure to pulse-modulated 900 MHz GSM-modulated RF radiation of mobile phones increased regional changes in brain activity and c-Fos expression [102, 103].

Exposure limits based on exposure to only RF radiation will result in an underestimation of the true risk and inadequate protection of human health under conditions in which co-exposures to other toxic agents lead to synergistic adverse effects [104].

B. Factors affecting dosimetry

Assumption 5) *Health effects are dependent only on the time-averaged SAR value; carrier wave modulations, frequency, or pulsing do not matter except as they influence the SAR.*

The FCC’s and ICNIRP’s exposure limits to RFR are based on SARs for frequencies up to 6 GHz and on power densities for frequencies between 6 GHz and 300 GHz averaged over 6-minute or 30-minute intervals for local areas and whole-body exposures [3, 5]. However, time-averaged dosimetry does not capture the unique characteristics of modulated or pulsed RFR. For example, GSM modulation may involve as many as 8 voice channels with a duration of 0.577 msec for each channel. Thus, the exposure from GSM modulation can be 8-times higher during each time slot pulse compared to exposure to a continuous wave at equivalent time-averaged SARs. Also, as noted under assumption #14, repetitive pulses of data in bursts with short exposures to 5G can cause localized

temperature spikes in the skin [105]. The impact of pulsed radiation on biological activities at the molecular or cellular levels is not taken into consideration with time-averaged dosimetry.

Another issue not addressed by time-averaged dosimetry is the importance of low frequency modulations on biological systems. As discussed under assumption #2, increased production of ROS in living cells and DNA damage have been demonstrated with exposure to low frequency modulations of radiofrequency carrier waves [106]. Exposure limits based on time-averaged SAR dosimetry or power density, without consideration of the impact of amplitude or frequency modulations, do not adequately address potential health effects of real-world exposures to RFR. There is ample evidence that various effects of RFR exposure depend on carrier wave modulations, frequency, or pulsing [43, 107, 108]. In contrast to ICNIRP/FCC, the IARC monograph on RFR carcinogenicity noted that RFR effects may be influenced by such exposure characteristics as duration of exposure, carrier frequency, type of modulation, polarization, exposure intermittence, and background electromagnetic fields [44].

C. Human brain tumor risk

Assumption 6) *The multiple human studies that find associations between exposure to cell phone RF radiation and increases in brain tumor risk are flawed because of biases in the published case-control studies, and because brain cancer rates have remained steady since the time that use of wireless communication devices became widespread.*

Although claims have been made that “current limits for cell phones are acceptable for protecting the public health” because “even with frequent daily use by the vast majority of adults, we have not seen an increase in events like brain tumors” [109], the SEER (Surveillance, Epidemiology, and End Results Program) database shows an annual decrease of 0.3% for all brain tumors, but an increase of 0.3% per year for glioblastoma in the US between 2000 and 2018 (<https://seer.cancer.gov/explorer/>). Most concerning was that the annual increase for glioblastoma was 2.7% per year for people under 20 years of age. In addition, Zada et al. [110] reported that the incidence of glioblastoma multiforme (GBM) in the frontal lobe, temporal lobe, and cerebellum increased in the US between 1992 and 2006, and Philips et al. [111] likewise reported a statistically significant increasing incidence of GBM in the frontal and temporal lobes of the brain in the UK during 1995–2015. In Sweden, rates of brain tumors in the Swedish National Inpatient Register and the Swedish Cancer Register increased from 1998 to

2015 [112]. In addition, it should be realized that cumulative exposure, side-of-head use, and latency for tumor formation from RFR are not fully captured in national cancer registries. Thus, the claim that trends in brain cancer incidence rates have not increased since mobile phones were introduced is both wrong and misleading. The specificity of effect needs to be factored into such trend analyses.

Case-control studies, using sound scientific methods, have consistently found increased risks with long-term, heavy mobile phone use for brain tumors of the glioma type and acoustic neuroma. This association was evaluated at IARC in 2011 by 30 expert participants who concluded that radiofrequency (RF) radiation is a “possible” human carcinogen [44]. In contrast, the much-cited Danish cohort study on ‘mobile phone users’ [113] was disregarded by IARC due to serious methodological shortcomings in the study design, including exposure misclassifications [44, 114].

Results of meta-analyses of glioma risk and acoustic neuroma from Swedish case-control studies conducted by Hardell and coworkers [115, 116], the 13-nation Interphone study [117], and the French study by Coureau et al. [118] are shown in Table 1 as odds ratios (OR) with 95% confidence intervals. For glioma on any location in the head, a statistically significant increase of nearly two-fold was found, while for ipsilateral mobile phone use (tumor and phone use on the same side of the head) the risk was increased by 2.5-fold. These ORs are based on the groups in each study with the highest category of cumulative call time, which were ≥ 1640 hr. in the Interphone study [117, 119] and the Swedish studies [115, 116], and ≥ 896 hr. in the study by Coureau et al. [118]. Decreased survival among glioma cases, especially astrocytoma grade IV, was associated with long-term and high cumulative use of wireless phones [120]. Increased risk for the mutant

type of *p53* gene expression in the peripheral zone of astrocytoma grade IV was associated with use of mobile phones for ≥ 3 hours a day. Increase in this mutation was significantly correlated with shorter overall survival time [121].

For acoustic neuroma, risk was significantly increased with cumulative exposure and ipsilateral use by 2.7-fold. A random effects model, which was based on a test for heterogeneity, was used for the meta-analyses of these published studies. Tumor volume of acoustic neuroma increased per 100 hr. of cumulative use of wireless phones in the Swedish study and years of latency, indicating tumor promotion [115].

Other case-control studies of mobile phone use also reported increased risk of acoustic neuroma [122–124]. Those studies were not included in the meta-analysis because data on cumulative mobile phone use with numbers of cases and controls were not given or there were other shortcomings. It is also noteworthy that tumor risks were increased in subsets of the Interphone study; for example, there was nearly a 2-fold increase in the risk of acoustic neuroma for ≥ 10 y and ipsilateral use among the North European countries that participated in the Interphone study [125].

Claims have been made that associations between increases in brain cancer risk and exposure to cell phone RF radiation in the published case-control studies may be attributable to recall and/or selection biases [5, 109]. However, a re-analysis of the Canadian data that was included in the Interphone study showed that there was no effect on the risk of glioma after adjustments were made for selection and recall biases [126]. Odds ratios (OR) for glioma were increased significantly and to a similar extent when comparing the highest quartile of use to those who were not regular users whether or not adjustments for biases were made. In addition, Hardell

Table 1 Odds ratios (OR) with 95% confidence interval (CI) for glioma and acoustic neuroma in case-control studies in the highest category for cumulative mobile phone use in hours^a

	Glioma				Acoustic neuroma			
	All		Ipsilateral		All		Ipsilateral	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Interphone [117, 119] Cumulative use ≥ 1640 hr	1.40	1.03–1.89	1.96	1.22–3.16	1.32	0.88–1.97	2.33	1.23–4.40
Coureau et al. [118] Cum use ≥ 896 hr	2.89	1.41–5.93	2.11	0.73–6.08				
Hardell et al. [115, 116] Cumulative use ≥ 1640 hr	2.13	1.61–2.82	3.11	2.18–4.44	2.40	1.39–4.16	3.18	1.65–6.12
Meta-analysis longest cumulative use	1.90	1.31–2.76	2.54	1.83–3.52	1.73	0.96–3.09	2.71	1.72–4.28

^a Note Hardell et al. [115, 116] also assessed use of cordless phones

and Carlberg [116] showed that the risk for glioma with mobile phone use was increased significantly even when compared to the risk for meningioma. Because risk of meningioma was not increased significantly, this tumor response could not be attributed to recall bias. Clearly, selection and recall biases do not explain the elevated brain tumor risk associated with the use of mobile phones. Thus, epidemiological evidence contradicts the opinions of the FCC and ICNIRP on brain tumor risk from RF radiation.

It should also be noted that the thyroid gland is a target organ for RFR from smartphones. A case-control study on mobile phone use suggested an increased risk for thyroid microcarcinoma associated with long-term cell phone use [127]. Peripheral lymphocyte DNA obtained from cases and controls was used to study genotype-environment interactions. The study showed that several genetic variants based on single nucleotide polymorphisms (SNPs) increased the risk of thyroid cancer with mobile phone use [128]. Increasing incidence of thyroid cancer in the Nordic countries, especially over the last two decades, has also been reported [129, 130]. In addition, a recent case-control study found significant increases in breast cancer risk among Taiwanese women based on their use of smartphones and distance between the breast and placement of their smartphone [131].

D. Individual variations in exposure and sensitivity to RF-EMF

Assumption 7) *There are no differences among individuals, including children, in the absorption of RF-EMF and susceptibility to this radiation.*

Differences between children and adults regarding the absorption of radiofrequency electromagnetic fields when mobile phones are operated close to the head have been demonstrated and widely documented [132–137]. The main factors accounting for these dissimilar absorption rates include differences in anatomy, tissue dielectric properties, and physiology. Through finite-difference time-domain (FDTD) simulations, employing detailed computational anthropomorphic models, it is possible to find differences relating to anatomy and to dimensions of the head.

Since EMF penetration into human tissues can be in the order of a few centimeters, depending on the wavelength, the inner tissues in the brain clearly will receive a significantly higher dose in the smaller heads of children compared to adults, despite the total absorption and the peak spatial SAR (psSAR) calculated across the whole head varying by smaller amounts [132, 133, 138]. Fernández et al. [136] estimated that the cell phone radiation psSAR in the hippocampus was 30-fold higher in

children compared to adults, while the psSAR in the eyes was 5-fold higher in children; these differences were due largely to closer proximity to the cell phone antennas. The thinner dimensions of children's skulls also contribute to this difference [135], resulting in a psSAR around 2-fold higher in children's brains [134–137, 139] compared to adults.

Additionally, tissues of young mammals have higher conductivity and electrical permittivity than those of mature animals [140]. This also contributes to greater EMF penetration and absorption, resulting in further increases in the psSAR. The psSAR in the skull bone marrow of children was estimated to increase by 10-fold due to higher conductivity in this tissue [137]. Distance between the mobile device and the body tissues is important in characterizing tissue dosimetry. The National Agency ANFR of France recently released cell phone SAR test data for 450 cell phones. Ten gram psSARs increased by 10–30% for each millimeter of proximal placement of the cell phone to the planar body phantom (<http://data.anfr.fr/explore/dataset/das-telephonie-mobile/?disjunctive.marque&disjunctive.modele&sort=marque>).

Finally, it is important to note that simulations of tissue dosimetry consider only the physical parameters of the tissues; they do not consider biological processes occurring in living tissues. While children are growing, developing organs and multi-organ systems are more susceptible to adverse effects of environmental agents; finite-difference time-domain (FDTD) simulations do not address differences in organ or system susceptibility for exposures occurring during child development.

Assumption 8) *There are no differences among individuals in their sensitivity to RF radiation-induced health effects.*

All life is “electrosensitive” to some degree as physiological processes are dependent on both subtle and substantial electromagnetic interactions at every level, from the molecular to the systemic. Responses to multiple types of electromagnetic exposure reveal that there is a far broader range of EMF sensitivity than previously assumed, and subgroups of extremely hypersensitive subjects exist [141–151]. Given the adverse health effects noted in Assumption #1, including cardiomyopathy, carcinogenicity and neurological effects, the acute, conscious symptoms manifesting in some individuals should not be unexpected. The term currently and most frequently used within the medical profession to describe those who are acutely, symptomatically sensitive to non-ionizing radiation exposures is Electromagnetic Hypersensitivity (EHS).

EHS is a multisystem, physical response characterized by awareness and/or symptoms triggered by EMF exposures. Common symptoms include (but are not limited to) headaches, dizziness, sleep disturbance, heart palpitations, tinnitus, skin rashes, visual disturbance, sensory disturbance, and mood disturbance [152, 153]. These symptoms are reported in response to even extremely low intensity (orders of magnitude below current safety levels) EMFs of multiple types (in terms of frequency, intensity and waveforms). Commonly noticed triggers of frequent and persistent EHS symptoms are pulse-modulated RF emissions, modulated at extremely low frequencies. Common triggering sources include mobile phones, DECT cordless landlines, Wi-Fi/Bluetooth-enabled computers, Wi-Fi routers, smart meters, base station antennas, and household electrical items. EMF avoidance/mitigation is found to be the most effective way to reduce symptoms [154].

Guidelines for EHS diagnosis and management have also been peer-reviewed and concur that the mainstay of medical management is avoidance of anthropogenic electromagnetic fields [152, 155, 156]. Case histories detailing clinical presentations, EMF measurements and mitigation are also published [157], and biomarkers including elevated markers of oxidative stress, inflammatory markers and changes in cerebral blood flow continue to be explored [152].

EHS has been proven to be a physical response under blinded conditions [145, 151, 158, 159] and, in addition to these studies, acute EMF-induced changes in cognition, behavior, and physiology reactions have been observed in studies involving animals [27, 30, 160–172]; plus further references under Assumption 13), which cannot be biased by media-cultivated fears. These studies provide further evidence which invalidates the nocebo response (physical symptoms induced by fear) as causal regarding symptoms.

It should not be expected that all provocation studies will reliably demonstrate adverse reactions; however, suggestions that the nocebo response may cause EHS symptoms were claimed from provocation studies which failed to show a relationship between the EMF exposure and the reported symptoms [173]. The failures of these studies are explainable given the very poor methodology in the majority of them. There were failures to account for a multitude of essential factors that must be tailored to the individual, such as variable symptom onset and offset, the necessity for adequate washout periods, specificity of trigger frequencies and intensities, requirement for complete EMF hygiene during sham exposures, requirement for life-like exposures (e.g., pulse-modulated information-carrying waves), etc. For example, it has been shown that various frequency channels from GSM/

UMTS mobile phones affect the same human cells differently [174–177]. Similarly, EHS has been shown to be frequency dependent [151]. As noted above, meaningful provocation studies need to take into consideration multiple physical parameters of exposure, including frequency, modulation, duration of exposure, and time after exposure [155]; however, most provocation studies that have failed to establish causative connection between RFR exposure and EHS symptoms [173] used only one or two conditions with short-term exposures.

There are many issues with the nocebo response as a cause of EHS, not least of which is also the absence of the required temporal link. For the nocebo response to be the cause of EHS, awareness and concern of negative health impacts from EMFs must precede symptoms. But, in the majority of EHS persons this is not the case [178]. As public risk communication improves, this will no longer be verifiable; however, this has been importantly observed at the only point in time when it could have been – prior to generalized awareness of health detriments from non-ionizing radiation (NIR).

While recognizing that some vulnerable groups may be more susceptible to effects of NIR exposure, ICNIRP [179] acknowledged that their guidelines may not safely accommodate these sensitive subgroups:

“Different groups in a population may have differences in their ability to tolerate a particular NIR [Non-Ionizing Radiation] exposure. For example, children, the elderly, and some chronically ill people might have a lower tolerance for one or more forms of NIR exposure than the rest of the population. Under such circumstances, it may be useful or necessary to develop separate guideline levels for different groups within the general population, but it may be more effective to adjust the guidelines for the general population to include such groups. Some guidelines may still not provide adequate protection for certain sensitive individuals nor for normal individuals exposed concomitantly to other agents, which may exacerbate the effect of the NIR exposure, an example being individuals with photosensitivity”.

In 2020, ICNIRP [23] also noted that biological effects are not easily discernible from adverse health effects, and that their guidelines:

“...are not intended to protect against biological effects as such (when compensatory mechanisms are overwhelmed or exhausted), unless there is also an associated adverse health effect. However, it is not always easy to draw a clear distinction between biological and adverse health effects, and indeed this can vary depending on individual susceptibility”.

to specific situations. An example is sensory effects from nonionizing radiation exposures under certain circumstances, such as a tingling sensation resulting from peripheral nerve stimulation by electric or magnetic fields; magnetophosphenes (light flickering sensations in the periphery of the visual field) resulting from stimulation of the retina by electric fields induced by exposure to low-frequency magnetic fields; and microwave hearing resulting from thermoelastic waves due to expansion of soft tissues in the head which travel via bone conduction to the inner ear. Such perceptions may sometimes lead to discomfort and annoyance. ICNIRP does not consider discomfort and annoyance to be adverse health effects by themselves, but, in some cases, annoyance may lead to adverse health effects by compromising well-being. The exposure circumstances under which discomfort and annoyance occur vary between individuals.”

Trivializing “discomfort” which is the pre-cursor to pain is not in keeping with WHO recommendations quoted by the same ICNIRP [23] document: “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.”

Discomfort is a sign that an organism is experiencing something which is compromising optimal health and although in some cases this can be trivial and reversible, in other cases it may not be reversed. There is an extremely broad range of both pain tolerance and also of pain perception among humans, and to achieve meaningful preventative health care, “discomfort” must be taken seriously and mitigated whenever possible. This is especially true in this case where symptoms such as headaches are being reported in response to mobile phone exposures at the same time as increased brain tumor risk is noted from those same exposures (see Assumption 6).

In reality, people with EHS are reporting far more serious health disruption than “discomfort” or “annoyance” and in some cases these symptoms are disabling [180, 181]. Increasingly, EHS is being recognized as a disability by national courts in France, Sweden, and Spain, which amplifies the requirement for safety guidelines that are deliberately accommodating to this more susceptible group [180].

E. Applied safety factors for RF-EMF-RF workers and the general population

Assumption 9) *A 50-fold safety factor for whole body exposure to RF radiation is adequate for protecting the general population to any health risks from RF radiation.*

Public health agencies in the US and worldwide apply multiple uncertainty factors to health effects data to establish exposure levels that are considered safe for the great majority of exposed populations [182–184]. Although guidelines for the use of uncertainty factors were developed for chemicals, they are also pertinent to other toxic agents, such as RFR. The uncertainty factors needed for toxic effects of RFR based on studies that demonstrate a no-observed-adverse-effect level (NOAEL) in experimental animals include:

- 1) Animal-to-human extrapolation. When data are based on studies in experimental animals, a factor of 3–10 is applied (for potential species differences in tissue dosimetry and response) unless there are convincing data demonstrating equivalent sensitivity in animals and humans. However, there is no evidence showing that humans are equally or less sensitive to RFR than animals that were used in studies from which exposure limits were established by the FCC and ICNIRP.
- 2) Adjustment for human variability. A second factor of 10 is used to account for interindividual variability in susceptibility (for instance, due to differences in age, sex, genetic variation, pre-existing diseases) to the toxic agent among the general population. It has been recognized that a factor of 10 for human variability is likely inadequate for sensitive subpopulations and may require an additional adjustment.
- 3) Extrapolation from short-term studies to lifetime exposure. An additional factor of 10 is applied for short-term studies, such as those used to establish exposure limits to RF radiation, to provide lifetime protection from chronic exposure. This is of particular importance considering the remarkably short periods over which RFR toxicity was originally assessed [10, 11].
- 4) Database insufficiencies. Finally, an uncertainty factor of 3-to-10 is applied for database inadequacy, i.e., for incomplete characterization of an agent’s toxicity. The behavioral studies [10, 11] that were used to establish the FCC and ICNIRP exposure limits to RFR do not provide a full characterization of the effects of this type of radiation nor did they identify the most sensitive adverse effect of RFR exposures.

Basing exposure limits to RFR on the behavioral studies in rats and monkeys [10, 11, 90, 91] would require the application of a composite uncertainty factor of about 900 to 10,000 to be consistent with approaches used by public health agencies to establish protective exposure limits for workers and the general population. Based on the size of the needed uncertainty/safety factor, the

data sets used by the FCC and ICNIRP are clearly inadequate to establish RF exposure limits with reasonable confidence. The arbitrarily selected safety factors of 10 for workers and 50 for the general population by the FCC and ICNIRP are woefully inadequate for protecting exposed populations.

When uncertainty/safety factors are applied to a misrepresented threshold exposure value for adverse effects, the resulting level does not provide assurance of health protection for the general population exposed to that agent. Studies cited above [18, 22, 91, 92, 96] show that the whole-body SAR of 4 W/kg is not a threshold level for adverse effects caused by RFR. In a recent quantitative analysis of various adverse health effects from the NTP study, Uche and Naidenko [185] showed that the permissible whole-body SAR of 0.08 W/kg (based on a 50-fold reduction of the assumed threshold SAR of 4 W/kg) was 20–40-fold higher than health protective SAR values derived by benchmark dose modelling of NTP data for cardiomyopathy (following application of 10-fold safety factors for interspecies and intraspecies variability). The approaches used by these authors are consistent with methodologies recommended by the US Environmental Protection Agency for quantifying health risks for toxic and carcinogenic environmental agents [1, 182]. Thus, a 50-fold reduction of the assumed threshold whole-body SAR of 4 W/kg is inadequate to protect the health of the general population from exposure to RF radiation.

Assumption 10) *A 10-fold safety factor for whole body exposure to RF radiation is adequate for protecting workers to any health risks from RF radiation.*

When RFR exposure limits were implemented in 1997, the rationale given for the difference in safety factors for the general population (50-fold) and for workers (10-fold) was “based on the exposure periods of the two populations, rounded to one digit (40 work hours per week/168 hours per week = ~0.2)” [6]. In addition to differences in exposure periods between workers and the general population, ICNIRP rationalizes the appropriateness of the lower safety factor for workers because “occupationally-exposed individuals can be considered a more homogeneous group than the general population,” they are, “in general, relatively healthy adults within a limited age range,” and “occupationally-exposed individuals should be operating under controlled conditions and be informed about the risks associated with non-ionizing radiation exposure for their specific situation and how to reduce these risks” [23]. In contrast, “the general public are, in most cases, unaware of their exposure to non-ionizing radiation and, without education, cannot

reasonably be expected to take precautions to minimize or avoid any adverse effects of exposure.”

The assumption that workers are trained in understanding health risks associated with exposure to RFR and in mitigating those risks to the greatest possible degree is not correct because neither the FCC nor the ICNIRP guidelines recognize any health effects from RFR at SARs below 4 W/kg, and the exposure limits authorized by the FCC and ICNIRP do not consider health effects from long-term exposures [3, 5]. The only health effect addressed by the FCC and ICNIRP is tissue damage due to excessive heating from acute exposures. Thus, the 10-fold reduction from the threshold whole-body SAR calculated from acute behavioral studies in rats and monkeys is inadequate for protecting the health of workers exposed long-term to RFR (see comments under assumption #9). There are no data demonstrating the adequacy of this arbitrarily chosen safety/uncertainty factor for occupationally-exposed workers, while on the contrary, excess cancer risks have been associated with exposure to RFR workers who operate radar and communication systems in military and occupational settings [186].

Assumption 11) *Exposure of any gram of cube-shaped tissue up to 1.6 W/kg, or 10g of cube-shaped tissue up to 2 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in the general population.*

Tissue dosimetry was analysed in the NTP study of cell phone RF radiation in rats and mice [187]. In rats, whole body exposures during the 10-minute on cycles were 1.5, 3.0, or 6.0 W/kg, and the brain and heart SARs varied from the whole-body SARs by about 7% to under 2-fold for the brain and heart, respectively. A quantitative risk assessment of the NTP tumor incidence data is needed to evaluate organ-specific cancer risk. The FDA [19] nomination to the NTP recognized the need for “large well-planned animal experiments to provide the basis to assess the risk to human health of wireless communications devices.” However, more than 3 years after an external peer-review of the NTP studies found “clear evidence of carcinogenic activity,” the FDA [109] has continued to downplay the importance of these findings and avoid conducting a quantitative risk assessment of the tumor data that they (the FDA) originally requested. In contrast to the FDA, Uche and Naidenko [185] analysed the NTP data on cardiomyopathy by a benchmark dose approach and found that the 10% extra risk level for this effect was in the range of a whole-body SAR of 0.2 to 0.4 W/kg. Thus, there is an increased risk (greater than 10%) of developing cardiomyopathy at local tissue SARs below 1.6 or 2.0 W/kg.

The peak spatial specific absorption rate (psSAR), as used by ICNIRP and the FCC, is an inadequate dosimetric of RF radiation at frequencies above 1 GHz. The psSAR is calculated by averaging fixed cubic volumes containing a given amount of mass, and assumes a homogeneous material with a given mass density. The ICNIRP recommendation is to average cubic volumes containing 10 g of tissue (10-g-psSAR), while the FCC recommendation is to average cubic volumes containing 1 g of tissue (1-g-psSAR). Current recommendations limit the use of psSAR to frequencies up to 6 GHz [3, 5].

An evaluation of the utility of using psSAR as a dosimetric parameter at different frequencies ranging from 100 MHz to 26 GHz and with cube sizes ranging from 10 mg to 10 g is shown in Additional file 2: Appendix 2. For the smaller cubes and lower frequencies, averaging in the cube does not underestimate the maximum value on the cube surface, but at higher frequencies the psSAR averaged on larger cubes can be several-fold lower than the psSAR averaged on smaller cubes. For example, at 2.45 GHz, averaging over a 10-g cube underestimates by 4 dB (approximately 2.5-fold) the psSAR averaged in smaller cubes, while for 5.8 GHz, averaging over a 10-g cube underestimates the psSAR by 12 dB (approximately 16-fold) compared with averaging in a 10-mg cube, and by 6 dB (approximately 4-fold) compared with averaging over a 1-g cube. When the frequency is increased, the underestimation of the psSAR averaged in larger cubes (e.g. 10 g or 1 g) compared to smaller cubes (e.g. 100 mg and 10 mg) becomes more pronounced. Considering the 10-g cube, the difference between the psSAR for 5.8 GHz EMF compared to 0.9 GHz EMF is around 7 dB (or approximately 5-fold underestimation). These large differences are due to reduced penetration of EMFs at higher frequencies. Therefore, the ICNIRP's 10-g-psSAR and FCC's 1-g-psSAR recommendations do not provide reliable dosimetric parameters to evaluate EMF absorption above 1 GHz.

The SAR averaging over a 10-g cube is also flawed for assessing carcinogenicity because it is too large a volume to focus on stem cells and their important role in carcinogenesis. Human stem cells were more sensitive to RFR exposures from GSM and UMTS mobile phones than lymphocytes and fibroblasts [175]. Instead of a random distribution of targets for carcinogenesis, localized distribution of SAR in smaller volumes is needed to more accurately characterize relationships between SAR and tumor induction. From the point of view of stem cell organization, the volume of SAR determinations may be especially important for setting safety limits for children, because most stem cells and their niches are spatially and temporally transient during brain development [188].

Assumption 12) *Exposure of any gram of cube-shaped tissue up to 8 W/kg, or 10 g of cube-shaped tissue up to 10 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in workers.*

Based on the analyses of tissue dosimetry in the NTP study [187], organ-specific toxic and carcinogenic effects were observed in rats at local tissue SARs that were much lower than 8 or 10 W/kg [18]. The tissue dosimetry in the NTP study and the inadequacy of the local SAR as specified by ICNIRP and the FCC is described in assumption #9.

F. Environmental exposure to RF radiation

Assumption 13) *There is no concern for environmental effects of RF radiation or for effects on wildlife or household pets.*

While background levels of RF-EMF are increasing in the environment, including rural remote areas [189], neither the FCC nor the ICNIRP take into consideration effects of this radiation on wildlife. The constant movement of most wildlife species in and out of varying artificial EMF can result in high exposures near communication structures, especially for flying species such as birds and insects. There is a substantial amount of scientific literature on the disrupting effects of RFR on wildlife (e.g., [190–206]).

Many nonhuman species use Earth's geomagnetic fields for activities such as orientation and seasonal migration, food finding, mating, nest and den building [190]. For example, migratory bird species [191, 192], honeybees [193], bats [194], fish [195–197], and numerous other species sense Earth's magnetic fields with specialized sensory receptors. Mechanisms likely involved in magneto-reception include magnetic induction of weak electric signals in specialized sensory receptors [198], magneto-mechanical interactions with the iron-based crystal magnetite [194], and/or free-radical interactions with cryptochrome photoreceptors [191, 192]. Each of these sensing processes shows extreme sensitivity to low intensity changes in electromagnetic fields. For a fuller description of the mechanisms by which non-human species use magneto-reception to perform essential life activities see Levitt et al. [190].

The following studies represent a few of the many examples of the disrupting effects of low-level exposures to RF-EMF on magneto-reception and the natural behavior of wildlife. Oscillating magnetic fields have been reported to disrupt the ability of migratory birds to orient and navigate in Earth's geomagnetic field [199–202].

Garden warblers became disoriented by exposure to a weak oscillating magnetic field of 1.403 MHz at an intensity as low as 2–3 nT [200]. The orientation of European robins that use Earth's magnetic field for compass orientation was completely disrupted by exposure to electromagnetic noise in the frequency range of 50 kHz to 5 MHz or a broadband noise-modulated ELF covering the range ~2 kHz to ~9 MHz [199, 201]. RFR in the low MHz range (7.0 MHz of 480 nT or 1.315 MHz of 15 nT) has been shown to disable the magneto-reception avian compass as long as the exposure was present [202].

In addition to effects on migratory birds, Landler et al. [203] found that exposure to a low-level magnetic field (1.43 MHz at an intensity of 30–52 nT) disrupted the natural orientation of juvenile turtles hatched on land. GSM-modulated 900 MHz RF radiation caused ants to lose their visual and olfactory memory for finding food [166]. Navigational abilities of trout were reduced when reared under conditions in which magnetic fields were spatially distorted [204].

Activities of honeybees are also disrupted by exposure to RF radiation. GSM-modulated cell phone radiation (900 MHz) caused a reduction in egg laying by queen bees and depletion of beehive pollen and honey counts [205]. GSM-modulated cell phone radiation (900 MHz) reduced hatching and altered pupal development of honey queen bee larvae [206].

The lack of consideration of chronic low-level RF radiation exposure on wildlife could result in dangerously disruptive effects on fragile ecosystems and on the behavior and survival of species that have long existed in Earth's natural environment.

G. 5G (5th generation wireless)

Assumption 14) *No health effects data are needed for exposures to 5G; safety is assumed because penetration is limited to the skin (“minimal body penetration”).*

Fifth generation (5G) wireless communication systems are being deployed worldwide to provide higher data transfer rates with shorter lag times between massive numbers of connected wireless devices. To provide faster transfer of large amounts of data (up to 20 gigabits per second peak data rates), the frequency range for 5G includes millimeter waves (30 to 300 GHz), in addition to carrier frequencies as low as 600 MHz. Extremely high frequency millimeter waves (MMW) that transmit large amounts of data to user devices are directed into narrow beams by line-of-sight transmission with beamforming antennas. Because millimeter waves do not penetrate solid structures such as building materials, hills, foliage, etc., and travel only short distances (a few hundred

meters), denser networks of base-stations with massive Multiple Input/Multiple Output (MIMO) transmitters and receivers in millions of small cell towers are being installed on structures such as utility poles. These features can lead to much closer proximity between humans and radiation-emitting antennas, and thereby change individual peak and average exposures to RFR.

For a 5G frequency of 26 GHz, EMF absorption is very superficial, which means that for typical human skin, more than 86% of the incident power is absorbed within the first millimeter. The skin penetration depth was computed as 1 mm based on the electrical conductivity of the skin and its electrical permittivity [5, 207]. This is expected to bring the SAR in this tissue well above the recommended limits ([208], and Additional file 2: Appendix 2). This is also expected to be harmful to very small species, such as birds and other small animals (e.g., insects) [209]. It is often claimed that because of its shallow penetration, exposure to high frequency 5G radiation is safe, and that the only effect is tissue heating [210]. However, this view ignores the deeper penetration of the ELF components of modulated RF signals, which are rated on the basis of heat alone, as well as the effects of short bursts of heat from pulsed signals [211, 212]. Within the first 1 mm of skin, cells divide to renew the stratum corneum (a consideration for skin cancer), and nerve endings in the dermis are situated within 0.6 mm (eyelids) to 3 mm (feet) of the surface (a consideration for neurological effects). Ultraviolet light, which exerts its action at a penetration depth of less than 0.1 mm [213, 214] is a recognized cause of skin cancer [87].

The higher the frequency of electromagnetic waves, the shorter the wavelength and the shallower the penetration of energy into exposed people or animals. For example, penetration depth in the human body is about 8 mm at 6 GHz and 0.92 mm at 30 GHz [5]. Because of the minimal depth of energy absorption at frequencies above 6 GHz, the FCC and ICNIRP have based exposure limits on power density instead of on SAR levels. The FCC [3] proposed a general localized power density exposure limit of 4 mW/cm² averaged over 1 cm² and not to exceed 30 minutes for 5G services up to 3000 GHz for the general population, claiming that this exposure is consistent with the peak spatial-average SAR of 1.6 W/kg averaged over any 1 g of tissue at 6 GHz. ICNIRP's [5] exposure limits for 5G are an absorbed power density of 200 W/m² (0.2 W/cm²) averaged over 4 cm² and a 6-minute interval for frequencies up to 30 GHz, and 400 W/m² (0.4 mW/cm²) averaged over 1 cm² and a 6-minute interval for frequencies of 30 GHz to 300 GHz.

Because of its minimal penetration, exposure to 5G radiation results in higher energy intensity on the skin and other directly-exposed body parts, such as the eye

cornea or lens. However, the skin, which is the largest organ in the human body, provides important functions such as acting as a protective physical and immunological barrier against mechanical injury, infection by pathogenic microorganisms, and entry of toxic substances. In addition, skin cancers, including basal cell carcinomas and squamous cell carcinomas, are the most prevalent human cancers, while melanomas are highly metastatic and increasing in prevalence. Although the high incidence of skin cancers are largely attributed to exposure to ultraviolet light, no studies have been reported on the effects of 5G radiation on (i) the skin's ability to provide protection from pathogenic microorganisms, (ii) the possible exacerbation of other skin diseases, (iii) promotion of sunlight-induced skin cancers, or (iv) initiation of skin cancer by itself. Information is also lacking on the effects of 5G radiation on nervous and immune systems which are also exposed even by the shallower penetration of MMW.

Another important factor is the maximum bandwidth with 5G radiation, which is up to 100 MHz in the frequency range of 450 MHz to 6 GHz, and up to 400 MHz in the ranges from 24 GHz to 52 GHz, compared to previous types of mobile communication where bandwidth is limited to 20 MHz. Because many studies indicated frequency-dependent, non-thermal RF effects from mobile communication RFR [43, 177] and for MMW effects [215, 216], the possibility of effective frequency windows for biological effects would increase with the increased bandwidth of 5G radiation.

Another consideration for effects of 5G exposures on human health is that radiation pulses created by extremely fast data transmission rates have the potential to generate bursts of energy that can travel much deeper than predicted by conventional models [217, 218]. Neufeld and Kuster [105] showed that repetitive pulses of data in bursts with short exposures to 5G can cause localized temperature spikes in the skin leading to permanent tissue damage even when the average power density values were within ICNIRP's acceptable safety limits. The authors urged the setting of new thermal safety standards to address the kind of health risks possible with 5G technology:

“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 millimetre-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...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...”

Areas of uncertainty and health concerns with 5G radiation include potential increase in skin cancer rates with (or possibly without) co-exposure to sunlight, exacerbation of skin diseases, greater susceptibility to pathogenic microorganisms, corneal damage or early development of cataracts, testicular effects, and possible resonant-enhanced absorption due to skin structures [219]. One of the complex technical challenges in relation to human exposure to 5G millimeter waves is that the unpredictable propagation patterns that could result in unacceptable levels of human exposure to electromagnetic radiation are not well understood [220]. Although MMW are almost completely absorbed within 1–2 mm in biologically-equivalent tissues, their effects may penetrate deeper in a live human body possibly by affecting signal transduction pathways. Thus, there are too many uncertainties with exposure to 5G to support an assumption of safety without adequate health effects data. There are no adequate studies on health effects from short-term or long-term exposures to 5G radiation in animal models or in humans.

Discussion

To develop health-based exposure limits for toxic and carcinogenic substances, regulatory agencies typically rely on available scientific evidence about the agent under review. In the mid- and late-1990s when the FCC [4] and the ICNIRP [9] initially established exposure limits for RFR, the prevailing assumptions were that any adverse effects from exposure to RFR were due to excessive heating because non-ionizing radiation did not have sufficient energy to break chemical bonds or damage DNA. However, non-thermal effects of RFR are demonstrated from studies that find different effects with exposure to continuous waves versus pulsed or modulated waves at the same frequency and the same SAR or power density, e.g., [221–226], and from studies that show adverse effects at very low exposure intensities, e.g., [78, 96].

Acute exposure studies conducted in rats and monkeys in the 1980s [10, 11] suggested that an SAR of 4 W/kg could be a threshold dose for behavioral effects. Because this SAR was associated with an approximate increase in body temperature of 1 °C, it was again assumed that no adverse health effects would occur if increases in core

body temperature were less than 1°C. From this putative threshold dose a “safety factor” of 10 was applied for occupational exposures and an additional factor of 5 (50x total) was applied for the general population, resulting in exposure limits in which the whole-body SAR was less than 0.4 W/kg for workers and 0.08 W/kg for the general population. However, realizing that local parts of the body could receive doses of RFR that were 10 to 20 times higher than the whole-body SARs, local peak exposure limits were set by the FCC at SARs 20-times higher than the whole-body SARs, i.e., 8 W/kg averaged over any 1-g of tissue for localized exposures for workers and 1.6 W/kg averaged over any 1-g for the general population [3, 4]. ICNIRP opted for partial body exposures that would not exceed 2.0 W/kg averaged over any 10g of cube-shaped tissue for the general population [5, 9]. To rationalize the smaller safety factor for workers (10-fold) versus the general population (50-fold), one claim made by ICNIRP [24] is that workers are informed about risks associated with non-ionizing radiation exposure and how to reduce these risks, whereas “the general public are, in most cases, unaware of their exposure to non-ionizing radiation and, without education, cannot reasonably be expected to take precautions to minimize or avoid any adverse effects of exposure.” From a public health perspective, the FCC and ICNIRP should make the public aware of their exposures to RFR and promote precautionary measures to minimize potential adverse effects, especially for children and pregnant women. Eight practical recommendations by the International EMF Scientist Appeal aimed at protecting and educating the public about potential adverse health effects from exposures to non-ionizing EMFs [227] are shown in Table 2.

The acute behavioral studies that provide the basis for the FCC’s and ICNIRP’s exposure limits lacked any information on potential effects of RF radiation that can occur after longer durations of exposure, and they did not address effects of carrier wave modulations used in wireless communications. Research on RFR conducted over

the past 25 years has produced thousands of scientific papers, with many demonstrating that acute behavioral studies are inadequate for developing health protective exposure limits for humans and wildlife, and that inherent assumptions underlying the FCC’s and ICNIRP’s exposure limits are not valid. First, 4 W/kg is not a threshold SAR for health effects caused by RFR exposures; experimental studies at lower doses and for longer durations of exposure demonstrated cardiomyopathy, carcinogenicity, DNA damage, neurological effects, increased permeability of the blood brain barrier, and sperm damage (see Assumptions 1–3). Multiple robust epidemiologic studies on cell phone radiation have found increased risks for brain tumors (Assumption 6), and these are supported by clear evidence of carcinogenicity of the same cell types (glial cell and Schwann cell) from animal studies. Even studies conducted by D’Andrea et al. [89, 90] before the limits were adopted found behavioral disruption in rats exposed to RFR for 14 or 16 weeks at mean SARs of 0.7 W/kg and at 1.23 W/kg. A combination of exposure duration and exposure intensity would be more appropriate for setting safety standards for exposure to RFR from mobile communication systems including mobile phones, base stations, and WiFi.

More than 120 studies have demonstrated oxidative effects associated with exposure to low intensity RFR (Additional file 1: Appendix 1). DNA damage that has been reported in studies of RFR was most likely caused by induction of oxidative stress, which is a key characteristic of human carcinogens [88], rather than by direct ionization (Assumption 2). The generation of reactive oxygen species has also been linked to DNA damage and the carcinogenicity of UVA radiation [87] and asbestos [228]. Despite the enormous amount of scientific evidence of low-dose effects of RFR, the IEEE [229] maintains that behavioral disruption is still the most sensitive and reproducible effect of RFR. It is this opinion that contributed to the FCC [3] and ICNIRP [5] reaffirming their previous exposure limits to RFR.

Table 2 Precautionary Measures Recommended by the International EMF Scientist Appeal

-
- 1) Priority should be given to protect children and pregnant women
 - 2) Guidelines and regulatory standards should be strengthened
 - 3) Manufacturers should be encouraged to develop safer technologies
 - 4) The public should be fully informed about the potential health risks from electromagnetic energy and taught harm reduction strategies
 - 5) Medical professionals need to be educated about the biological effects of electromagnetic energy and be provided training on treatment of patients with electromagnetic sensitivity
 - 6) Governments need to fund training and research on electromagnetic fields and health that is independent of industry
 - 7) The media should disclose experts’ financial relationships with industry when citing their opinions regarding health and safety aspects of EMF-emitting technologies
 - 8) Radiation-free areas need to be established, especially for individuals with EHS
-

Other concerns about the current exposure limits for RFR are that they do not consider potential synergistic effects due to co-exposure to other toxic or carcinogenic agents, the impact of pulsed radiation or frequency modulations, multiple frequencies, differences in levels of absorption or of susceptibility by children, or differences among individuals in their sensitivity to RFR (see Assumptions 4, 5, 7, 8). Currently, children's cumulative exposures are much higher than previous generations and they continue to increase [230]. ICNIRP [23, 179] acknowledged that their guidelines do not accommodate sensitive subgroups and admit to difficulties separating "biological effects" from "health effects." Neurological symptoms, some of which are acknowledged by ICNIRP and currently being experienced by persons with EHS, are most certainly non-thermal "health effects" that need to be mitigated by providing environments with reduced exposures to anthropogenic EMF for hypersensitive individuals.

The debilitating effects and restrictions suffered by adults and children with EHS constitutes a contravention of the 2010 Equalities Act, Human Rights Act and other ethical and legal frameworks. Failure to respond and appropriately safeguard this group is already causing preventable morbidity, mortality and economic deficit due to lost workdays, compensations for health damages and increased healthcare costs. Conversely, accommodating this group by, as suggested by ICNIRP [179], acting to 'adjust the guidelines for the general population to include such groups' would not only lessen the negative impacts for people with EHS, but would also improve public health more broadly, given the other NIR-related health concerns that are highlighted in this paper.

Basing local tissue exposure limits on 1-g [3] or 10-g [5] cubes substantially underestimates the peak spatial SAR compared to basing local tissue exposure limits on smaller cubes (e.g., 100 mg or 10 mg), and therefore are not reliable dosimetric parameters to evaluate EMF absorption at frequencies above 1 GHz (Assumptions 11, 12). The volumes specified by the FCC and ICNIRP for local tissue SAR limits are too large to focus on stem cells which are important targets for carcinogenesis. To reduce health risks from exposures to RFR, limits for localized distribution of the SAR should be based on 100 mg, or preferably 10 mg cubes.

Another important deficiency raised in this paper is that neither the FCC nor ICNIRP addresses concerns for environmental effects of RFR on wildlife, even though there is extensive literature demonstrating the disrupting effects of RFR on wildlife behavior (Assumption 13).

The arbitrarily selected uncertainty/safety factors applied to the putative threshold SAR for RFR are woefully inadequate for protecting public health

(Assumptions 9, 10). Based on the way the US Environmental Protection Agency, the International Council for Harmonization, and the National Institute for Occupational Safety and Health (US NIOSH) apply uncertainty/safety factors to a no-observed-adverse-effect level (NOAEL) in experimental animals [182–184], the safety factor for RFR would be at least 900 to 10,000, which is 18 to 200 times larger than the safety factor recommended by the FCC and ICNIRP for the general population. This large safety factor is based on adjustments for human variability, lifetime exposure from short-term studies, and database insufficiencies that include incomplete characterization of the toxicity of RFR. Clearly, the acute behavioral studies that served as the basis for the current exposure limits for RFR are not suitable for characterizing human health risks associated with long-term exposure to this type of radiation. The NCRP report from 1986 [6] and the ANSI/IEEE document from 1992 [7] recognized that when future studies on biological effects of RFR become available including effects of chronic exposures or evidence of non-thermal interactions there will be a need to evaluate and possibly revise exposure standards. When the FCC [3] and ICNIRP [5] reaffirmed their exposure limits from the 1990s, they dismissed the scientific evidence that invalidated the assumptions that underlie the basis for those exposure limits. An independent re-evaluation of RFR exposure limits based on the scientific knowledge gained over the past 25 years is needed and is long overdue. This evaluation should be performed by scientists and medical doctors who have no conflicting interests and who have expertise in RF-EMF exposure and dosimetry, toxicology, epidemiology, clinical assessment, and risk assessment. Special precautions should be taken to ensure that interpretations of health effects data and the setting of exposure limits for RFR are not influenced by the military or the telecommunications industry. In the meantime, manufacturers should be obliged to develop safer technologies [227].

Finally, we note our concern about the worldwide deployment of 5G communication networks for faster transfer of large amounts of data, but with no adequate health effects studies demonstrating the safety of high frequency millimeter waves. Because of limitations of the penetration and distance of travel of millimeter waves, dense networks of base stations are being mounted on structures such as utility poles in highly populated cities. Also, because the absorption of EMF at frequencies above 6 GHz is minimal, ICNIRP [5] has specified absorbed power density (S_{ab}) as the dosimetric parameter for "heating effects" at the higher frequencies. S_{ab} is a function of the incident power density (S_{inc}) and the input reflection coefficient (Γ). In near field scenarios, the S_{inc} does not have a singular value; this is largely due

to the heterogeneous nature of human body tissues and their relevant parameters (such as the permittivity, equivalent conductivity, mass density), which vary in different body regions and with frequency. Therefore, unless a powerful EMF simulation method together with realistic human models are used, the S_{inc} and the reflection coefficient values would be difficult to accurately estimate, making the resulting S_{ab} unreliable.

The assumption that 5G is safe at the power density limits recommended by ICNIRP (50 W/m² and 10 W/m² averaged over 6 min for occupational and 30 min for public exposures, respectively) because of its minimal penetration into the body does not justify the dismissal of the need for health effects studies prior to implementing 5G networks. The new communication networks will result in exposures to a form of radiation that has not been previously experienced by the public at large (Assumption 14). The implementation of 5G technology without adequate health effects information raises many questions, such as: Will exposure to 5G radiation: (i) compromise the skin's ability to provide protection from pathogenic microorganisms? (ii) will it exacerbate the development of skin diseases? (iii) will it increase the risk of sunlight-induced skin cancers? (iv) will it increase the risk of damage to the lens or cornea? (v) will it increase the risk of testicular damage? (vi) will it exert deeper tissue effects either indirectly following effects on superficial structures or more directly due to deeper penetration of the ELF components of modulated RF signals? (vii) will it adversely affect wildlife populations? Answers to these questions and others that are relevant to human and wildlife health should be provided *before* widespread exposures to 5G radiation occur, not afterwards. Based on lessons that should have been learned from studies on RFR at frequencies below 6 GHz, we should no longer rely on the untested assumption that current or future wireless technology, including 5G, is safe without adequate testing. To do otherwise is not in the best interest of either public or environmental health.

Abbreviations

ANSI: American National Standards Institute; CDMA: Code-division multiple access; dB: Decibel; DECT: Digital enhanced cordless technology; EHS: Electromagnetic hypersensitivity; ELF: Extremely low frequency; EMF: Electromagnetic field; FCC: Federal Communications Commission; FDA: Food and Drug Administration; GHz: Gigahertz; GBM: Glioblastoma multiforme brain cancer; GSM: Global system for mobile communication; IARC: International Agency for Research on Cancer; ICNIRP: International Commission on Non-Ionizing Radiation Protection; IEEE: Institute of Electrical and Electronics Engineers; LTE: Long Term Evolution (4G); MMW: Millimeter wave; NCRP: National Council on Radiation Protection and Measurements; NIR: Non-ionizing radiation; nT: Nanotesla; NTP: National Toxicology Program; 8-OHdG: 8-hydroxy-2'-deoxyguanosine; psSAR: Peak spatial specific absorption rate; RFR: Radiofrequency radiation; ROS: Reactive oxygen species; SAR: Specific absorption rate; UMTS: Universal mobile telecommunications service (3G); UVR: Ultraviolet radiation; 5G: 5th generation wireless.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12940-022-00900-9>.

Additional file 1: Appendix 1 Table 1. Studies demonstrating increased oxidative DNA damage and other indicators of oxidative stress at SAR < 4W/kg.

Additional file 2: Appendix 2. On the Inadequacy of the psSAR Dosimetric Parameter at Frequencies above 1 GHz. **Table 1.** Electric permittivity and electric conductivity of the gray matter. **Figure 1.** A block of gray matter radiated by different frequencies. The highlighted cubes are of 10g, 1g, 100mg and 10mg. **Fig. 2.** A block of gray matter radiated by different frequencies. The highlighted cubes are of 10g, 1g, 100mg and 10mg. **Fig. 3.** Electric field intensity averaged in each cube for different frequencies: in the left axis, the electric field is in dB and in the right axis the electric field is in V/m normalized to 100V/m.

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Authors' contributions

IB, AD, CF, LH, PH, KK, DM, EMB, RLM, and IY drafted the initial sections of this manuscript: by IB (factors affecting dosimetry), AD and CF (absorption in children versus adults, peak spatial specific absorption rate), LH (human brain cancer risk), KK (sperm damage), DM and DM (5G), EMB (electromagnetic hypersensitivity), RLM (cardiomyopathy, carcinogenicity, neurologic effects, safety factors), and IY (oxidative stress and DNA damage). IY prepared Appendix 1, and AD and CF prepared Appendix 2. The authors who drafted sections of the manuscript, as well as CB, KC, SD, EK, AM, JMM, and WS reviewed multiple manuscript drafts and made revisions. All authors reviewed and approved the final manuscript.

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Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G

International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF)*

Abstract

In the late-1990s, the FCC and ICNIRP adopted radiofrequency radiation (RFR) exposure limits to protect the public and workers from adverse effects of RFR. These limits were based on results from behavioral studies conducted in the 1980s involving 40–60-minute exposures in 5 monkeys and 8 rats, and then applying arbitrary safety factors to an apparent threshold specific absorption rate (SAR) of 4W/kg. The limits were also based on two major assumptions: any biological effects were due to excessive tissue heating and no effects would occur below the putative threshold SAR, as well as twelve assumptions that were not specified by either the FCC or ICNIRP. In this paper, we show how the past 25 years of extensive research on RFR demonstrates that the assumptions underlying the FCC's and ICNIRP's exposure limits are invalid and continue to present a public health harm. Adverse effects observed at exposures below the assumed threshold SAR include non-thermal induction of reactive oxygen species, DNA damage, cardiomyopathy, carcinogenicity, sperm damage, and neurological effects, including electromagnetic hypersensitivity. Also, multiple human studies have found statistically significant associations between RFR exposure and increased brain and thyroid cancer risk. Yet, in 2020, and in light of the body of evidence reviewed in this article, the FCC and ICNIRP reaffirmed the same limits that were established in the 1990s. Consequently, these exposure limits, which are based on false suppositions, do not adequately protect workers, children, hypersensitive individuals, and the general population from short-term or long-term RFR exposures. Thus, urgently needed are health protective exposure limits for humans and the environment. These limits must be based on scientific evidence rather than on erroneous assumptions, especially given the increasing worldwide exposures of people and the environment to RFR, including novel forms of radiation from 5G telecommunications for which there are no adequate health effects studies.

Keywords: Federal Communications Commission (FCC), International commission on non-ionizing radiation protection (ICNIRP), Radiofrequency radiation (RFR), Exposure limits, Exposure assessment, Radiation health effects, Reactive oxygen species (ROS), DNA damage, 5G, Scientific integrity, Cell phone*, Mobile phone*

Introduction

In establishing exposure limits for toxic or carcinogenic agents, regulatory agencies generally set standards that take into account uncertainties of health risks for the general population [1] and for susceptible subgroups such as children [2]. That approach has not been applied in the same way to the setting of exposure limits for

*The terms cell phone and mobile phone are used interchangeably in this commentary; cell phone is the term used in the United States, while mobile phone is the term used in most of Europe.

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radiofrequency radiation (RFR) (frequency range: 3 kHz to 300 GHz). Moreover, assumptions underlying the current RFR exposure limits are flawed; hence, the limits that are currently applied do not adequately protect human and environmental health. This issue is discussed in greater detail under Assumption #9.

The Federal Communications Commission's (FCC) limits for maximum permissible exposure to RF electromagnetic fields (EMF) [3] were established in 1996 [4], and currently include many recommendations from the International Commission on Non-Ionizing Radiation Protection [5]. These exposure limits were expected to protect against adverse health effects in humans that might occur from short-term (i.e., acute) exposures to RFR and have been maintained by the FCC for the past 26 years. The exposure limits that were established by the FCC in 1996 relied on criteria recommended by the National Council on Radiation Protection & Measurements (NCRP) [6] and the Institute of Electrical and Electronics Engineers (ANSI/IEEE) [7, 8]. The limits were "based on a determination that potentially harmful biological effects can occur at a SAR (specific absorption rate) level of 4.0 W/kg as averaged over the whole-body." The SAR is a measure of the rate of RF energy absorbed per unit mass.

The threshold for a behavioral response and for acute thermal damage in sensitive tissues was considered to be an exposure that produced a whole-body SAR greater than 4 W/kg. In parallel with the development of the FCC's RFR exposure limits, ICNIRP's guidelines for limiting exposure to RF-EMF were also based on behavioral studies conducted in rats and monkeys in the 1980s [9].

The harmful effects that served as the basis for the exposure criteria were changes in behavior observed in small numbers of rats and monkeys when exposed to RFR for up to 60 minutes to power densities at which the whole-body SAR was approximately 4 W/kg or higher [10, 11]. Those studies were conducted in the early 1980s (1980 and 1984, respectively) by investigators of the US Navy Department. Consequently, 4 W/kg was identified as the threshold SAR for adverse health effects induced by RFR. In food-deprived monkeys that were exposed to three different frequencies (225 MHz, 1.3 GHz, and 5.8 GHz) during 60-min sessions, lever-pressing response rates for the delivery of food pellets were reduced compared to sham exposure sessions. The threshold SAR for this decreased response was reported to range from 3.2 to 8.4 W/kg [11]. Similarly, in food-deprived rats exposed to 40-min sessions at 1.28 or 5.62 GHz radiation, the threshold SAR for a decrease in response rate was reported to range from approximately 3.8 to 4.9 W/kg [10]. In experimental studies in which monkeys were exposed in an anechoic chamber for 4 hours to 1.29 GHz

radiation at various power densities, an increase in mean body temperature of 0.7°C was associated with a whole-body SAR of 4 W/kg [12]. Behavior disruption associated with an increase in body temperature of approximately 1.0°C was assumed to be the most sensitive measure of harmful effects from RF-EMF exposure.

After establishing 4 W/kg as the threshold dose for acute harmful effects, both the FCC [3, 4] and ICNIRP [5, 9] set exposure limits for controlled occupational exposures to 0.4 W/kg SAR averaged over the whole body (based on applying a 10-fold safety/uncertainty factor). For the general population, the FCC's and ICNIRP's exposure limits were set at 0.08 W/kg SAR averaged over the whole body (by applying an additional 5-fold safety/uncertainty factor) for frequencies between 3 MHz and 3 GHz. The exposure limits established by the FCC and ICNIRP do not account for any impact of differing signal characteristics, such as carrier wave modulations or pulsing of the signal. Whole-body exposures for the general population are based simply on power levels averaged over 30-minute periods [3, 5].

Based on SAR distributions from whole-body exposures in which local (i.e., partial body) SARs were estimated to be 10 to 20 times the average value, local exposure limits were set 20 times higher than the average whole-body exposure limit [4–7]. For occupational exposures, local peak exposure limits were permitted up to 8 W/kg averaged over any 1-g cube of tissue [4] or 10 W/kg averaged over any 10 g of contiguous tissue [9] by the FCC and ICNIRP, respectively. For the general population, local peak SARs for partial-body exposures were not to exceed 1.6 W/kg averaged over any 1 g of cube-shaped tissue [3], or not to exceed 2.0 W/kg averaged over any 10 g of cube-shaped tissue [5]. Higher limit values are permissible for extremities. Extremities include the hands, wrists, feet, ankles, and pinnae (the external part of the ear), despite the close proximity of the ear to the brain. These adjustments were made long before the widespread use of wireless communication devices in which the emitting antenna is typically held close to local body organs such as the brain. The NCRP document [6] acknowledges that exposures could be greater than the recommended safety limit values when people are in close proximity to emitters of RFR.

The setting of exposure limits for the prevention of excessive tissue heating was based on the following assumptions: 1) electromagnetic waves at frequencies used in wireless communications do not have sufficient energy to break chemical bonds or ionize molecules [13]; 2) RFR could not damage DNA; and 3) tissue heating was the only possible biological effect of nonionizing radiation [5, 9, 14–16]. For potential environmental and human health issues that are not addressed in the

A) Effects of RF radiation at exposures below the putative threshold SAR of 4 W/kg

Assumption 1) There is a threshold exposure for any adverse health effect caused by RF radiation; in the frequency range of 100 kHz to 6 GHz it is a whole-body exposure that exceeds an SAR of 4 W/kg. Any biological effect of RF radiation above the threshold exposure is due to tissue heating.

Assumption 2) RF radiation is incapable of causing DNA damage other than by heating; there is no mechanism for non-thermal DNA damage.

Assumption 3) Two to seven exposures to RF radiation for up to one hour duration are sufficient to exclude adverse effects for any duration of exposure including chronic exposures.

Assumption 4) No additional effects would occur from RF radiation with co-exposure to other environmental agents.

B) Factors affecting dosimetry

Assumption 5) Health effects are dependent only on the SAR value; carrier wave modulations, frequency, or pulsing do not matter except as they influence the SAR.

C) Human brain cancer risk

Assumption 6) The multiple human studies that find associations between exposure to cell phone RF radiation and increases in brain cancer risk are flawed because of biases in the published case-control studies, and because brain cancer rates have remained steady since the time that use of wireless communication devices became widespread.

D) Individual variations in exposure and sensitivity to RF-EMF

Assumption 7) There are no differences among individuals, including children, in the absorption of RF-EMF and susceptibility to this radiation.

Assumption 8) There are no differences among individuals in their sensitivity to RF radiation-induced health effects.

E) Applied safety factors for EMF-RF workers and the general population

Assumption 9) A 50-fold safety factor for whole body exposure to RF radiation is adequate for protecting the general population to any health risks from RF radiation.

Assumption 10) A 10-fold safety factor for whole body exposure to RF radiation is adequate for protecting workers to any health risks from RF radiation.

Assumption 11) Exposure of any gram of cube-shaped tissue up to 1.6 W/kg, or 10 grams of cube-shaped tissue up to 2 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in the general population.

Assumption 12) Exposure of any gram of cube-shaped tissue up to 8 W/kg, or 10 grams of cube-shaped tissue up to 10 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in workers.

F) Environmental exposure to RF radiation

Assumption 13) There is no concern for environmental effects of RF radiation or for effects on wildlife or household pets.

G) 5G (5th generation wireless)

Assumption 14) No health effects data are needed for exposures to 5G; safety is assumed because penetration is limited to the skin (“minimal body penetration”).

Fig. 1 Assumptions Underlying the FCC/ICNIRP Exposure Limits for RF Radiation

setting of exposure limits (for example effects of chronic exposures, or effects of co-exposure of skin to RFR and other environmental agents, such as would occur with 5G exposure in combination with sunlight), the implicit assumption is that such effects do not matter, or that the arbitrarily selected safety/uncertainty factor is sufficient to deal with those concerns. In any case, it is expected that underlying assumptions applied to health risk assessments would be clearly described [1].

Exposure limits for RF radiation are based on numerous assumptions; however, research studies published over the past 25 years show that most of those assumptions are not supported by scientific evidence. In the NCRP report [6], the authors noted that when further understanding of biological effects of RF radiation becomes available, exposure guidelines will need to be evaluated and possibly revised. The ANSI/IEEE document [7] also notes that effects of chronic exposure or evidence of non-thermal interactions could result in revising exposure standards. Unfortunately, these recommendations were never implemented. Assumptions of

safety from exposures that could adversely affect human or environmental health should be tested and validated *before* widespread exposures occur, not afterwards, by agencies responsible for protecting public health.

In this paper, we highlight studies that demonstrate the fallacy of inherent assumptions in the FCC/ICNIRP guidelines for RF radiation exposure limits, and we find that the limits fail to protect human and environmental health. Fourteen assumptions that underlie the RFR exposure limits established in the 1990s and reaffirmed in 2020 by the FCC [4, 5] and ICNIRP [5, 9] are addressed in this paper and are shown in Fig. 1.

Assumptions underlying exposure limits for RF radiation and the scientific evidence demonstrating that these assumptions are not valid

A. Effects of RF radiation at exposures below the putative threshold SAR of 4W/kg

Assumption 1) *There is a threshold exposure for any adverse health effect caused by RF radiation; in the*

frequency range of 100 kHz to 6 GHz it is a whole-body exposure that exceeds an SAR of 4 W/kg. Any biological effect of RF radiation above the threshold exposure is due to tissue heating.

Cardiomyopathy and carcinogenicity

In response to a request from the Food and Drug Administration's (FDA) Center for Devices and Radiological Health [17], the National Toxicology Program (NTP) conducted toxicity and carcinogenicity studies of cell phone (CDMA- or GSM-modulated) radiation in rats and mice exposed to RFR at frequencies of 900 MHz and 1800 MHz, respectively [18, 19]. Exposures to RFR for up to 2 years occurred in reverberation chambers over 18 hours/day on a continuous cycle of 10 minutes on and 10 minutes off. In rats, the whole-body SAR levels during the 10-minute on cycles were 0, 1.5, 3, or 6 W/kg.

The major histopathological findings from the NTP study in male rats [18] included dose-related increases in cardiomyopathy, increased incidence of cancers and preneoplastic lesions in the heart (schwannoma and Schwann cell hyperplasia) and brain (glioma and glial cell hyperplasia), increases in prostate gland tumors and hyperplasias, significant increases in adrenal gland tumors, and significant increases in the overall incidence of benign or malignant neoplasms in all organs in the 3 W/kg groups. The incidence of cardiomyopathy was also increased in GSM-exposed female rats, and significant increases in DNA damage were found in rats and mice [18, 19]. Similarly, an earlier study by Chou et al. [20] found a significant (3.6-fold) increase in the incidence of primary malignant neoplasms in male rats exposed to 2450 MHz pulsed RFR for 25 months (21.5 hr./day) at an SAR that ranged from 0.15 to 0.4 W/kg.

A 3-day external peer-review of the NTP studies confirmed there was "clear evidence of carcinogenic activity" in male rats for heart schwannomas, and "some evidence of carcinogenic activity" for brain gliomas and adrenal gland tumors with exposure to either GSM- or CDMA-modulated RF radiation [21]. In addition, a lifetime study by the Ramazzini Institute reported a significant increase in heart schwannomas in male rats exposed 19 hour/day to 1800 MHz GSM-modulated RFR at a field strength of 50 V/m, equivalent to a whole-body SAR of 0.1 W/kg [22]. The incidence of heart Schwann cell hyperplasia was also increased in that exposure group. These findings are consistent with results from the NTP study and demonstrate that the proliferative effect of modulated RFR in heart Schwann cells is a reproducible finding that can occur at doses far below the assumed whole-body threshold SAR of 4 W/kg.

ICNIRP [23] dismissed the evidence of carcinogenicity for RFR that was provided in the studies by the NTP [18] and the Ramazzini Institute [22] based on their earlier critique of those studies [24]. However, that critique demonstrated an unfortunate lack of understanding together with a misrepresentation of the design, conduct, and interpretation of experimental carcinogenicity studies in animal models [25], as well as a lack of appreciation for the remarkable concordance between the tumor responses observed in experimental animals with those identified in cancer epidemiology studies of mobile phone users described under Assumption #6.

Neither heating effects nor thermal stress was likely causal of the adverse health effects observed in the NTP [18] study, since there was no tissue damage observed in a 28-day study at the same SARs, there was no significant effect on body weight during the 2-year study, and there were no exposure-related clinical observations that would indicate thermal or metabolic stress. Furthermore, a preliminary thermal pilot study demonstrated that body temperatures did not increase by more than 1°C at the exposure levels used in the chronic studies [26], and there is no evidence that a small change in body temperature associated with the RFR exposures in the NTP study can cause the types of carcinogenic effects that were observed. The similar findings of GSM-modulated RFR on Schwann cells by the Ramazzini Institute [22] at much lower whole-body SARs confirm these effects to be independent of tissue heating.

Neurological effects

Though the FCC and ICNIRP exposure limits are based on a putative threshold dose of 4 W/kg due to behavioral disruption observed at higher doses in rats and monkeys [10, 11] numerous studies have shown consistent and reproducible deficits in spatial learning and memory in laboratory animals exposed to RF radiation at SARs below 4 W/kg. Examples of study exposures that demonstrated these neurological effects included 900 MHz GSM at 0.41–0.98 W/kg, 2 hr./day for 4 days in mice [27]; 900 MHz GSM at 0.52–1.08 W/kg, 2 hr./day for 1 month in rats [28]; 900 MHz GSM at 1.15 W/kg, 1 hr./day for 28 days in rats [29]; 900 MHz pulsed RFR at 0.3–0.9 W/kg for 6 hr./day in rats from conception to birth and tested at 30 days of age [30]; 900 MHz GSM and 1966 MHz UMTS at 0.4 W/kg for 6 months in rats [31]; and 900 MHz continuous wave EMF at 0.016 W/kg 3 hr./day for 28 days in rats [32]. The studies cited above are not the only studies showing these effects, but they clearly demonstrate that exposure to RFR at an SAR of 4 W/kg is not a threshold dose for neurological effects in rodents. The effects of RF radiation on spatial learning and memory indicate

the hippocampus as a target site of these exposures. For a more complete listing of neurological effects of RFR reported between 2007 and 2017 see Lai [33].

In addition, many studies have reported changes in brain electrical activities in human subjects, measured by electroencephalography (EEG), including sleep disturbance from single exposures to cell phone RF radiation. This is not surprising since the nervous system transmits messages based on electrical signals generated by nerve cells. Decreased β -trace protein, which is a key enzyme in the synthesis of a sleep-promoting neurohormone, has been seen in young adults with high-cumulative amounts of hours of mobile phone use [34]. Another frequently reported effect of RF radiation is increased blood-brain barrier permeability in rats at SARs much lower than 4 W/kg, e.g. [32, 35–41]. Oxidative stress induced in the brain of animals exposed to RF-EMF has been associated with observed neurological effects [42]. Although many studies did not observe significant changes in neurological effects in humans and several studies did not observe increased permeability in the blood-brain barrier in animal models [33], differences in EMF frequency, modulation, duration of exposure, and direction of incident waves to the exposed subject, as well as difference in dielectric properties and the size and shape of the exposed subject likely account for differences in observed effects [43, 44].

Sperm damage

The effect of non-ionizing microwave radiation on the testis (testicular degeneration in mice) was first reported 60 years ago [45]. Since then, and with the rapid increase in use of RF-EMF emitting devices, numerous studies have investigated testicular effects of RFR and potential associations with male infertility [46–50]. Human and animal studies have shown that the testis is one of the most sensitive organs to RF-EMF exposures, and that keeping a mobile phone in trouser pockets in talk mode can affect fertility parameters e.g., sperm motility, sperm count, sperm morphology, and apoptosis [48, 51]. Meta-analyses of published epidemiologic studies on the impact of mobile phone radiation on sperm quality in adult men have found significant decreases in sperm motility, sperm viability and/or sperm concentrations that were associated with mobile phone usage [52–55]. Several physical factors associated with exposure conditions can affect the outcome of human studies, including depth of energy penetration, duration of call, type of transmission technology, distance of the device to the body or testis, and power density with defined SAR. For example, Zilberlicht et al. [56] observed higher rates of

abnormal sperm concentrations among men who held their phones less than 50 cm from their groin.

The effects of RFR on reproductive parameters in humans are consistent with results from experimental studies in animals and in vitro studies. For example, exposure of human semen to 850 MHz radiation from mobile phones for 1 hour at an SAR of 1.46 W/kg caused a significant decrease in sperm viability that was associated with an increase in reactive oxygen species (ROS) [50] or an increase in sperm DNA fragmentation [57]. Exposure of isolated human spermatozoa to 1.8 GHz RF-EMF significantly reduced sperm motility and induced ROS generation at an SAR of 1.0 W/kg, and significantly increased oxidative DNA damage and DNA fragmentation at an SAR of 2.8 W/kg [58].

Some examples of effects of RFR on male fertility factors in studies with experimental animals at SARs below 4 W/kg include: a decrease in sperm count and an increase in ROS in rats exposed to mobile phone frequencies 2 hr./day, for 35 days (SAR=0.9 W/kg) [59]; increases in oxidative stress, 8-hydroxy-deoxyguanosine (8-OHdG), and DNA strand breaks in the testes of rats exposed to 900 MHz (SAR=0.166 W/kg), 1800 MHz (0.166 W/kg), or 2100 MHz (0.174 W/kg) 2 hr./day for 6 months [60]; an increase in ROS, a decrease in sperm count, and altered sperm morphology in rats exposed to 900 MHz 3G mobile phone radiation (SAR=0.26 W/kg) 2 hr./day for 45 days [61]; decreased sperm quality in rats in which local exposure of the scrotum to 2575–2635 MHz 4G smartphone time division LTE radiation occurred for 1 min over 10 min intervals 6 hr./day for 150 days [62]; impaired testicular development at 35 days of age in male offspring of pregnant rats that were exposed to 2.45 GHz RFR (SAR=1.75 W/kg) 2 hr./day throughout pregnancy [63]; decreased sperm motility in mice exposed to 905 MHz RFR (SAR=2.2 W/kg) 12 hr./day for 5 weeks, and increased ROS formation and DNA fragmentation after 1 week of exposure [64]. Although negative studies have also been reported, it is important to remember that the outcome of experimental studies can be affected by differences in exposure conditions, including the frequency, modulation, polarization, stray electromagnetic fields, local SAR, duration of exposure, and analytical methods [43, 44].

Although the mechanism of testicular effects from exposure to non-thermal levels of RFR is not fully known, numerous studies in rats and mice, and in human sperm have found associations between negative effects on fertility parameters and increases in ROS and/or DNA damage [48, 51, 57, 58, 60, 61, 64–68]. Thus, the adverse effects of RFR on sperm quality are likely due in large part to induced generation of ROS.

Assumption 2) *RF radiation is incapable of causing DNA damage other than by heating; there is no mechanism for non-thermal DNA damage.*

In 2009, ICNIRP [16] claimed that “low energy photons of RF radiation are too weak to affect ionization or cause significant damage to biological molecules such as DNA, under ordinary circumstances.” However, DNA damage and other genotoxic effects have been observed in numerous studies of low intensity RFR in animal models and in humans. For example, the NTP study found statistically significant increases in DNA damage in brain cells of exposed rats and mice compared to sham controls [18, 19, 69], and Akdag et al. [70] found statistically significant increases in DNA damage in hair cells in the ear canal among 30 to 60 year-old men who used mobile phones for 10 years for 0–30 min/day, 30–60 min/day, or greater than 60 min/day compared to people who did not use mobile phones. In the latter study, the extent of DNA damage increased with increasing daily exposure duration. In a review of published studies on genetic effects of ELF- and RF-EMF, Lai [71] listed more than 150 studies in which non-thermal exposures to RFR produced increases in DNA damage, chromosome aberrations, or micronuclei formation.

In addition, it is well established that DNA damage can also be caused by indirect processes, such as by the generation of reactive oxygen species (ROS), and numerous studies have demonstrated DNA damage at exposures below the putative threshold SAR of 4 W/kg. More than 120 published studies have demonstrated oxidative effects associated with exposure to low intensity RFR (Additional file 1: Appendix 1). An analysis of experimental studies on molecular effects of low intensity RF radiation (RFR) in biological systems found that the majority (93 of 100 studies) demonstrated the induction of oxidative effects [72]. More recent studies (from 2017) revealed that all 30 relevant publications (100%) detected significant oxidative effects under low intensity RFR exposures, and most of these studies used modulated RFR from wireless communication devices.

Increased production of ROS in living cells may be caused by weak magnetic fields altering recombination rates of short-lived radical pairs generated by normal metabolic processes leading to changes in free radical concentrations [73], or by low intensity extremely low frequency (ELF) EMFs resulting in alterations in voltage-gated ion channels in cell membranes causing changes in cation flow across membranes [74]. These mechanisms apply to both ELF-EMFs and to RFR modulated by pulsed fields at extremely low frequencies. Other biophysical mechanisms by which non-thermal RF-EMF can

cause biological effects through interactions with normal cellular processes have been described [75].

Increasing NADH oxidase activity is another mechanism by which RFR can increase ROS production. NADH oxidases, which are membrane-associated enzymes that catalyze one-electron reduction of oxygen to superoxide radical using NADH as the electron donor, have been identified as primary mediators of RFR interactions in cellular systems [76]. A significant (3-fold) increase in the activity of NADH oxidase was measured in purified plasma membranes from HeLa cells exposed to 875 MHz for 5 or 10 min at a power density of 200 $\mu\text{W}/\text{cm}^2$. This exposure intensity is significantly lower than the ICNIRP [5] safety limit.

The major source of ROS in living cells is the mitochondrial electron transport chain, where leakage of electrons generates superoxide radicals due to the partial reduction of oxygen [77]. A dose-dependent effect of 1.8 GHz modulated RFR exposure (SAR=0.15 and 1.5 W/kg) on mitochondrial ROS production was detected in mouse spermatogonial germ cells [65]. Exposure of quail embryos to extremely low intensity modulated RFR (GSM 900 or 1800 MHz, 0.25 or 0.32 $\mu\text{W}/\text{cm}^2$) during the initial days of embryogenesis resulted in a robust overproduction of superoxide radical and nitrogen oxide in mitochondria of embryonic cells [78, 79]. Thus, multiple mechanisms for the increased production of ROS by low intensity RF radiation have been demonstrated.

Numerous studies have been published on mutagenic effects of low intensity RF-EMFs, especially studies that identified increases in levels of a specific marker of oxidative DNA damage and a risk factor for cancer, 8-hydroxy-2'-deoxyguanosine (8-OHdG) [58, 60, 78–84]. For example, the level of 8-OHdG in human spermatozoa was increased significantly after *in vitro* exposure for 16 hr. to 1.8 GHz at a power level of 2.8 W/kg and correlated with levels of ROS generation [58]. Likewise, exposure of quail embryos *in ovo* to GSM-modulated 900 MHz of 0.25 $\mu\text{W}/\text{cm}^2$ for 1.5, 5, or 10 days was sufficient to produce a significant, two-threefold, increase in 8-OHdG levels in embryonic cells [79]. Umbilical cord blood and placenta tissue samples obtained after delivery from women who used mobile phones during pregnancy had significantly higher levels of oxidative stress parameters, including 8-OHdG and malondialdehyde, compared to cord blood and placental tissue from women who did not use mobile phones during pregnancy [85]. In addition, DNA damage, analyzed by the comet assay, was increased significantly in cord blood lymphocytes obtained from women who used mobile phones during pregnancy compared to cord blood lymphocytes obtained from women who did not use mobile phones.

As low intensity RF radiation does not have sufficient energy to ionize DNA molecules, and as increased production of ROS in living cells due to RF-EMF exposures has been reliably documented, an indirect effect of this type of radiation is the formation of oxidative damage to DNA. The most aggressive form of ROS that can cause oxidative DNA damage is the hydroxyl radical; this reactive oxygen species can be generated from superoxide radical and hydrogen peroxide [86], which may be produced in living cells exposed to low intensity RF radiation. Ultraviolet radiation (UVR, encompassing UVA, UVB, and UVC), which is classified by IARC as “carcinogenic to humans”), can also cause indirect DNA damage by generating ROS [87]. Thus, both RFR and UVR, which can similarly induce oxidative DNA damage, can increase cancer risk by a similar mechanism.

Increased production of ROS and depletion of antioxidant capacity in living cells exposed to low intensity RF radiation can result in oxidative DNA damage. Induction of oxidative stress, which is a key characteristic of many human carcinogens [88], including UVR and asbestos, can also lead to genotoxicity and carcinogenicity of non-ionizing RF radiation without causing direct DNA damage.

Assumption 3) *Two to seven exposures to RF radiation for up to 1 hour duration are sufficient to exclude adverse effects for any duration of exposure including chronic exposures.*

The behavioral studies in 8 male rats and 5 male monkeys that served as the basis for the exposure limits to RF radiation adopted by the FCC and ICNIRP involved 2 to 7 exposure sessions of 40-minute duration for rats [10] and 3 exposure sessions of 60-minute duration for monkeys at each power density [11]. Additional support for the threshold SAR of 4 W/kg in the frequency range of 100 kHz to 6 GHz came from behavioral studies conducted in rats and monkeys by D’Andrea et al. [89, 90]. However, D’Andrea et al. [91, 92] also reported that exposure of rats to continuous wave 2450 MHz RFR for 14 or 16 weeks caused significant differences in behavioral activity between sham-exposed rats and RFR-exposed rats at mean SARs of 0.7 W/kg and at 1.23 W/kg, indicating that 4 W/kg is not a threshold SAR with extended exposure durations. Since that time many studies have shown that responses to non-thermal RFR depend on both exposure intensity and exposure duration [93]. Importantly, the same response was observed with lower exposure intensity but prolonged exposure duration as at higher exposure intensity and shorter duration [94].

Recognizing that the exposure limits do not address potential health effects after long-term exposures to

RF radiation emitted from wireless devices that people are experiencing, the FDA [17] nominated RF radiation to the NTP for chronic toxicology and carcinogenicity studies out of concern that “existing exposure guidelines are based on protection from acute injury from thermal effects of RFR exposure, and may not be protective against any non-thermal effects of chronic exposures.” Adverse health effects noted in Assumption #1, including cardiomyopathy, carcinogenicity, sperm damage, and neurological effects, as well as the human epidemiology studies to be described in Assumption #6, occurred with much longer exposures to RF radiation than the exposure durations used in the acute studies in rats [10] and monkeys [11]. Consequently, the acute behavioral exposure studies that served as the basis for exposure limits to RF radiation established by the FCC and ICNIRP are inadequate to identify and characterize adverse effects of RF radiation after longer exposure durations. Neither the exposure limits established in the 1990s by the FCC [4] or by ICNIRP [9], nor those reaffirmed more recently by these groups [3, 5] address health risks associated with long-term exposure to RF radiation.

Assumption 4) *No additional effects would occur from RF radiation with co-exposure to other environmental agents.*

The current FCC/ICNIRP exposure limits do not take into consideration interactive effects of RF radiation with other environmental agents even though such effects have been documented. Interactions of RF radiation with other agents may result in antagonistic or synergistic effects, i.e., effects that are greater than the sum of each agent alone.

In the International Agency for Research on Cancer (IARC) evaluation of the carcinogenicity of RF-EMF [44], the expert working group noted that 4 of 6 co-carcinogenesis studies available at that time showed increased responses with exposure to RF-EMF. One of those studies reported co-carcinogenic effects of UMTS-modulated RF radiation at 4.8 W/m² in the liver and lung of mice that had been treated with the carcinogen ethylnitrosourea (ENU) in utero [95]; the incidence of liver and lung cancers were increased in mice exposed to ENU plus RF radiation compared to cage controls, sham controls and ENU alone. After the IARC evaluation, Lerchl et al. [96] replicated the experimental design of Tillmann et al. [95] by exposing mice to RF-EMF at whole-body SAR levels of 0 (sham), 0.04, 0.4, and 2 W/kg. Significant increases in lung adenomas and/or liver carcinomas were observed at all exposure levels. Lerchl et al. [96] concluded that their “findings are a very clear indication that tumor-promoting effects

of life-long RF-EMF exposure may occur at levels supposedly too low to cause thermal effects.” Thus, the reproducibility of the tumor-promoting effects of RFR at non-thermal exposure levels has been demonstrated.

Other examples of reported synergistic effects include the following study results. Synergistic effects on damage to human lymphocytes were observed with co-exposure to RFR (1.8 GHz RFR, SAR 3 W/kg) and 2 different mutagens, namely, mitomycin C or 4-nitroquinoline-1-oxide [97], or with co-exposure to ultraviolet (UVC) light [98]. A synergistic effect was found on DNA damage in human blood cells exposed to 2450 MHz radiation (5 mW/cm²) and then exposed to mitomycin C [99]. A potentiation effect on DNA damage was observed in cultured mammalian cells exposed to CDMA-modulated 835 MHz RF-EMF (SAR = 4 W/kg) and the clastogens cyclophosphamide or 4-nitroquinoline-1-oxide [100]. Gene expression was altered in neuronal and glial cells of rats pre-treated with lipopolysaccharide, a neuroinflammatory agent, and then exposed to 1800 MHz GSM modulated radiation (SAR = 3.22 W/kg) for 2 hr. [101]. In rats pre-treated with picrotoxin, a chemical that induces seizures, exposure to pulse-modulated 900 MHz GSM-modulated RF radiation of mobile phones increased regional changes in brain activity and c-Fos expression [102, 103].

Exposure limits based on exposure to only RF radiation will result in an underestimation of the true risk and inadequate protection of human health under conditions in which co-exposures to other toxic agents lead to synergistic adverse effects [104].

B. Factors affecting dosimetry

Assumption 5) *Health effects are dependent only on the time-averaged SAR value; carrier wave modulations, frequency, or pulsing do not matter except as they influence the SAR.*

The FCC’s and ICNIRP’s exposure limits to RFR are based on SARs for frequencies up to 6 GHz and on power densities for frequencies between 6 GHz and 300 GHz averaged over 6-minute or 30-minute intervals for local areas and whole-body exposures [3, 5]. However, time-averaged dosimetry does not capture the unique characteristics of modulated or pulsed RFR. For example, GSM modulation may involve as many as 8 voice channels with a duration of 0.577 msec for each channel. Thus, the exposure from GSM modulation can be 8-times higher during each time slot pulse compared to exposure to a continuous wave at equivalent time-averaged SARs. Also, as noted under assumption #14, repetitive pulses of data in bursts with short exposures to 5G can cause localized

temperature spikes in the skin [105]. The impact of pulsed radiation on biological activities at the molecular or cellular levels is not taken into consideration with time-averaged dosimetry.

Another issue not addressed by time-averaged dosimetry is the importance of low frequency modulations on biological systems. As discussed under assumption #2, increased production of ROS in living cells and DNA damage have been demonstrated with exposure to low frequency modulations of radiofrequency carrier waves [106]. Exposure limits based on time-averaged SAR dosimetry or power density, without consideration of the impact of amplitude or frequency modulations, do not adequately address potential health effects of real-world exposures to RFR. There is ample evidence that various effects of RFR exposure depend on carrier wave modulations, frequency, or pulsing [43, 107, 108]. In contrast to ICNIRP/FCC, the IARC monograph on RFR carcinogenicity noted that RFR effects may be influenced by such exposure characteristics as duration of exposure, carrier frequency, type of modulation, polarization, exposure intermittence, and background electromagnetic fields [44].

C. Human brain tumor risk

Assumption 6) *The multiple human studies that find associations between exposure to cell phone RF radiation and increases in brain tumor risk are flawed because of biases in the published case-control studies, and because brain cancer rates have remained steady since the time that use of wireless communication devices became widespread.*

Although claims have been made that “current limits for cell phones are acceptable for protecting the public health” because “even with frequent daily use by the vast majority of adults, we have not seen an increase in events like brain tumors” [109], the SEER (Surveillance, Epidemiology, and End Results Program) database shows an annual decrease of 0.3% for all brain tumors, but an increase of 0.3% per year for glioblastoma in the US between 2000 and 2018 (<https://seer.cancer.gov/explore/>). Most concerning was that the annual increase for glioblastoma was 2.7% per year for people under 20 years of age. In addition, Zada et al. [110] reported that the incidence of glioblastoma multiforme (GBM) in the frontal lobe, temporal lobe, and cerebellum increased in the US between 1992 and 2006, and Philips et al. [111] likewise reported a statistically significant increasing incidence of GBM in the frontal and temporal lobes of the brain in the UK during 1995–2015. In Sweden, rates of brain tumors in the Swedish National Inpatient Register and the Swedish Cancer Register increased from 1998 to

2015 [112]. In addition, it should be realized that cumulative exposure, side-of-head use, and latency for tumor formation from RFR are not fully captured in national cancer registries. Thus, the claim that trends in brain cancer incidence rates have not increased since mobile phones were introduced is both wrong and misleading. The specificity of effect needs to be factored into such trend analyses.

Case-control studies, using sound scientific methods, have consistently found increased risks with long-term, heavy mobile phone use for brain tumors of the glioma type and acoustic neuroma. This association was evaluated at IARC in 2011 by 30 expert participants who concluded that radiofrequency (RF) radiation is a “possible” human carcinogen [44]. In contrast, the much-cited Danish cohort study on ‘mobile phone users’ [113] was disregarded by IARC due to serious methodological shortcomings in the study design, including exposure misclassifications [44, 114].

Results of meta-analyses of glioma risk and acoustic neuroma from Swedish case-control studies conducted by Hardell and coworkers [115, 116], the 13-nation Interphone study [117], and the French study by Coureau et al. [118] are shown in Table 1 as odds ratios (OR) with 95% confidence intervals. For glioma on any location in the head, a statistically significant increase of nearly two-fold was found, while for ipsilateral mobile phone use (tumor and phone use on the same side of the head) the risk was increased by 2.5-fold. These ORs are based on the groups in each study with the highest category of cumulative call time, which were ≥ 1640 hr. in the Interphone study [117, 119] and the Swedish studies [115, 116], and ≥ 896 hr. in the study by Coureau et al. [118]. Decreased survival among glioma cases, especially astrocytoma grade IV, was associated with long-term and high cumulative use of wireless phones [120]. Increased risk for the mutant

type of *p53* gene expression in the peripheral zone of astrocytoma grade IV was associated with use of mobile phones for ≥ 3 hours a day. Increase in this mutation was significantly correlated with shorter overall survival time [121].

For acoustic neuroma, risk was significantly increased with cumulative exposure and ipsilateral use by 2.7-fold. A random effects model, which was based on a test for heterogeneity, was used for the meta-analyses of these published studies. Tumor volume of acoustic neuroma increased per 100 hr. of cumulative use of wireless phones in the Swedish study and years of latency, indicating tumor promotion [115].

Other case-control studies of mobile phone use also reported increased risk of acoustic neuroma [122–124]. Those studies were not included in the meta-analysis because data on cumulative mobile phone use with numbers of cases and controls were not given or there were other shortcomings. It is also noteworthy that tumor risks were increased in subsets of the Interphone study; for example, there was nearly a 2-fold increase in the risk of acoustic neuroma for ≥ 10 y and ipsilateral use among the North European countries that participated in the Interphone study [125].

Claims have been made that associations between increases in brain cancer risk and exposure to cell phone RF radiation in the published case-control studies may be attributable to recall and/or selection biases [5, 109]. However, a re-analysis of the Canadian data that was included in the Interphone study showed that there was no effect on the risk of glioma after adjustments were made for selection and recall biases [126]. Odds ratios (OR) for glioma were increased significantly and to a similar extent when comparing the highest quartile of use to those who were not regular users whether or not adjustments for biases were made. In addition, Hardell

Table 1 Odds ratios (OR) with 95% confidence interval (CI) for glioma and acoustic neuroma in case-control studies in the highest category for cumulative mobile phone use in hours^a

	Glioma				Acoustic neuroma			
	All		Ipsilateral		All		Ipsilateral	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Interphone [117, 119] Cumulative use ≥ 1640 hr	1.40	1.03–1.89	1.96	1.22–3.16	1.32	0.88–1.97	2.33	1.23–4.40
Coureau et al. [118] Cum use ≥ 896 hr	2.89	1.41–5.93	2.11	0.73–6.08				
Hardell et al. [115, 116] Cumulative use ≥ 1640 hr	2.13	1.61–2.82	3.11	2.18–4.44	2.40	1.39–4.16	3.18	1.65–6.12
Meta-analysis longest cumulative use	1.90	1.31–2.76	2.54	1.83–3.52	1.73	0.96–3.09	2.71	1.72–4.28

^a Note Hardell et al. [115, 116] also assessed use of cordless phones

and Carlberg [116] showed that the risk for glioma with mobile phone use was increased significantly even when compared to the risk for meningioma. Because risk of meningioma was not increased significantly, this tumor response could not be attributed to recall bias. Clearly, selection and recall biases do not explain the elevated brain tumor risk associated with the use of mobile phones. Thus, epidemiological evidence contradicts the opinions of the FCC and ICNIRP on brain tumor risk from RF radiation.

It should also be noted that the thyroid gland is a target organ for RFR from smartphones. A case-control study on mobile phone use suggested an increased risk for thyroid microcarcinoma associated with long-term cell phone use [127]. Peripheral lymphocyte DNA obtained from cases and controls was used to study genotype-environment interactions. The study showed that several genetic variants based on single nucleotide polymorphisms (SNPs) increased the risk of thyroid cancer with mobile phone use [128]. Increasing incidence of thyroid cancer in the Nordic countries, especially over the last two decades, has also been reported [129, 130]. In addition, a recent case-control study found significant increases in breast cancer risk among Taiwanese women based on their use of smartphones and distance between the breast and placement of their smartphone [131].

D. Individual variations in exposure and sensitivity to RF-EMF

Assumption 7) *There are no differences among individuals, including children, in the absorption of RF-EMF and susceptibility to this radiation.*

Differences between children and adults regarding the absorption of radiofrequency electromagnetic fields when mobile phones are operated close to the head have been demonstrated and widely documented [132–137]. The main factors accounting for these dissimilar absorption rates include differences in anatomy, tissue dielectric properties, and physiology. Through finite-difference time-domain (FDTD) simulations, employing detailed computational anthropomorphic models, it is possible to find differences relating to anatomy and to dimensions of the head.

Since EMF penetration into human tissues can be in the order of a few centimeters, depending on the wavelength, the inner tissues in the brain clearly will receive a significantly higher dose in the smaller heads of children compared to adults, despite the total absorption and the peak spatial SAR (psSAR) calculated across the whole head varying by smaller amounts [132, 133, 138]. Fernández et al. [136] estimated that the cell phone radiation psSAR in the hippocampus was 30-fold higher in

children compared to adults, while the psSAR in the eyes was 5-fold higher in children; these differences were due largely to closer proximity to the cell phone antennas. The thinner dimensions of children's skulls also contribute to this difference [135], resulting in a psSAR around 2-fold higher in children's brains [134–137, 139] compared to adults.

Additionally, tissues of young mammals have higher conductivity and electrical permittivity than those of mature animals [140]. This also contributes to greater EMF penetration and absorption, resulting in further increases in the psSAR. The psSAR in the skull bone marrow of children was estimated to increase by 10-fold due to higher conductivity in this tissue [137]. Distance between the mobile device and the body tissues is important in characterizing tissue dosimetry. The National Agency ANFR of France recently released cell phone SAR test data for 450 cell phones. Ten gram psSARs increased by 10–30% for each millimeter of proximal placement of the cell phone to the planar body phantom (<http://data.anfr.fr/explore/dataset/das-telephonie-mobile/?disjunctive.marque&disjunctive.modele&sort=marque>).

Finally, it is important to note that simulations of tissue dosimetry consider only the physical parameters of the tissues; they do not consider biological processes occurring in living tissues. While children are growing, developing organs and multi-organ systems are more susceptible to adverse effects of environmental agents; finite-difference time-domain (FDTD) simulations do not address differences in organ or system susceptibility for exposures occurring during child development.

Assumption 8) *There are no differences among individuals in their sensitivity to RF radiation-induced health effects.*

All life is “electrosensitive” to some degree as physiological processes are dependent on both subtle and substantial electromagnetic interactions at every level, from the molecular to the systemic. Responses to multiple types of electromagnetic exposure reveal that there is a far broader range of EMF sensitivity than previously assumed, and subgroups of extremely hypersensitive subjects exist [141–151]. Given the adverse health effects noted in Assumption #1, including cardiomyopathy, carcinogenicity and neurological effects, the acute, conscious symptoms manifesting in some individuals should not be unexpected. The term currently and most frequently used within the medical profession to describe those who are acutely, symptomatically sensitive to non-ionizing radiation exposures is Electromagnetic Hypersensitivity (EHS).

EHS is a multisystem, physical response characterized by awareness and/or symptoms triggered by EMF exposures. Common symptoms include (but are not limited to) headaches, dizziness, sleep disturbance, heart palpitations, tinnitus, skin rashes, visual disturbance, sensory disturbance, and mood disturbance [152, 153]. These symptoms are reported in response to even extremely low intensity (orders of magnitude below current safety levels) EMFs of multiple types (in terms of frequency, intensity and waveforms). Commonly noticed triggers of frequent and persistent EHS symptoms are pulse-modulated RF emissions, modulated at extremely low frequencies. Common triggering sources include mobile phones, DECT cordless landlines, Wi-Fi/Bluetooth-enabled computers, Wi-Fi routers, smart meters, base station antennas, and household electrical items. EMF avoidance/mitigation is found to be the most effective way to reduce symptoms [154].

Guidelines for EHS diagnosis and management have also been peer-reviewed and concur that the mainstay of medical management is avoidance of anthropogenic electromagnetic fields [152, 155, 156]. Case histories detailing clinical presentations, EMF measurements and mitigation are also published [157], and biomarkers including elevated markers of oxidative stress, inflammatory markers and changes in cerebral blood flow continue to be explored [152].

EHS has been proven to be a physical response under blinded conditions [145, 151, 158, 159] and, in addition to these studies, acute EMF-induced changes in cognition, behavior, and physiology reactions have been observed in studies involving animals [27, 30, 160–172]; plus further references under Assumption 13), which cannot be biased by media-cultivated fears. These studies provide further evidence which invalidates the nocebo response (physical symptoms induced by fear) as causal regarding symptoms.

It should not be expected that all provocation studies will reliably demonstrate adverse reactions; however, suggestions that the nocebo response may cause EHS symptoms were claimed from provocation studies which failed to show a relationship between the EMF exposure and the reported symptoms [173]. The failures of these studies are explainable given the very poor methodology in the majority of them. There were failures to account for a multitude of essential factors that must be tailored to the individual, such as variable symptom onset and offset, the necessity for adequate washout periods, specificity of trigger frequencies and intensities, requirement for complete EMF hygiene during sham exposures, requirement for life-like exposures (e.g., pulse-modulated information-carrying waves), etc. For example, it has been shown that various frequency channels from GSM/

UMTS mobile phones affect the same human cells differently [174–177]. Similarly, EHS has been shown to be frequency dependent [151]. As noted above, meaningful provocation studies need to take into consideration multiple physical parameters of exposure, including frequency, modulation, duration of exposure, and time after exposure [155]; however, most provocation studies that have failed to establish causative connection between RFR exposure and EHS symptoms [173] used only one or two conditions with short-term exposures.

There are many issues with the nocebo response as a cause of EHS, not least of which is also the absence of the required temporal link. For the nocebo response to be the cause of EHS, awareness and concern of negative health impacts from EMFs must precede symptoms. But, in the majority of EHS persons this is not the case [178]. As public risk communication improves, this will no longer be verifiable; however, this has been importantly observed at the only point in time when it could have been – prior to generalized awareness of health detriments from non-ionizing radiation (NIR).

While recognizing that some vulnerable groups may be more susceptible to effects of NIR exposure, ICNIRP [179] acknowledged that their guidelines may not safely accommodate these sensitive subgroups:

“Different groups in a population may have differences in their ability to tolerate a particular NIR [Non-Ionizing Radiation] exposure. For example, children, the elderly, and some chronically ill people might have a lower tolerance for one or more forms of NIR exposure than the rest of the population. Under such circumstances, it may be useful or necessary to develop separate guideline levels for different groups within the general population, but it may be more effective to adjust the guidelines for the general population to include such groups. Some guidelines may still not provide adequate protection for certain sensitive individuals nor for normal individuals exposed concomitantly to other agents, which may exacerbate the effect of the NIR exposure, an example being individuals with photosensitivity”.

In 2020, ICNIRP [23] also noted that biological effects are not easily discernible from adverse health effects, and that their guidelines:

“...are not intended to protect against biological effects as such (when compensatory mechanisms are overwhelmed or exhausted), unless there is also an associated adverse health effect. However, it is not always easy to draw a clear distinction between biological and adverse health effects, and indeed this can vary depending on individual susceptibility”.

to specific situations. An example is sensory effects from nonionizing radiation exposures under certain circumstances, such as a tingling sensation resulting from peripheral nerve stimulation by electric or magnetic fields; magnetophosphenes (light flickering sensations in the periphery of the visual field) resulting from stimulation of the retina by electric fields induced by exposure to low-frequency magnetic fields; and microwave hearing resulting from thermoelastic waves due to expansion of soft tissues in the head which travel via bone conduction to the inner ear. Such perceptions may sometimes lead to discomfort and annoyance. ICNIRP does not consider discomfort and annoyance to be adverse health effects by themselves, but, in some cases, annoyance may lead to adverse health effects by compromising well-being. The exposure circumstances under which discomfort and annoyance occur vary between individuals.”

Trivializing “discomfort” which is the pre-cursor to pain is not in keeping with WHO recommendations quoted by the same ICNIRP [23] document: “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.”

Discomfort is a sign that an organism is experiencing something which is compromising optimal health and although in some cases this can be trivial and reversible, in other cases it may not be reversed. There is an extremely broad range of both pain tolerance and also of pain perception among humans, and to achieve meaningful preventative health care, “discomfort” must be taken seriously and mitigated whenever possible. This is especially true in this case where symptoms such as headaches are being reported in response to mobile phone exposures at the same time as increased brain tumor risk is noted from those same exposures (see Assumption 6).

In reality, people with EHS are reporting far more serious health disruption than “discomfort” or “annoyance” and in some cases these symptoms are disabling [180, 181]. Increasingly, EHS is being recognized as a disability by national courts in France, Sweden, and Spain, which amplifies the requirement for safety guidelines that are deliberately accommodating to this more susceptible group [180].

E. Applied safety factors for RF-EMF-RF workers and the general population

Assumption 9) *A 50-fold safety factor for whole body exposure to RF radiation is adequate for protecting the general population to any health risks from RF radiation.*

Public health agencies in the US and worldwide apply multiple uncertainty factors to health effects data to establish exposure levels that are considered safe for the great majority of exposed populations [182–184]. Although guidelines for the use of uncertainty factors were developed for chemicals, they are also pertinent to other toxic agents, such as RFR. The uncertainty factors needed for toxic effects of RFR based on studies that demonstrate a no-observed-adverse-effect level (NOAEL) in experimental animals include:

- 1) Animal-to-human extrapolation. When data are based on studies in experimental animals, a factor of 3–10 is applied (for potential species differences in tissue dosimetry and response) unless there are convincing data demonstrating equivalent sensitivity in animals and humans. However, there is no evidence showing that humans are equally or less sensitive to RFR than animals that were used in studies from which exposure limits were established by the FCC and ICNIRP.
- 2) Adjustment for human variability. A second factor of 10 is used to account for interindividual variability in susceptibility (for instance, due to differences in age, sex, genetic variation, pre-existing diseases) to the toxic agent among the general population. It has been recognized that a factor of 10 for human variability is likely inadequate for sensitive subpopulations and may require an additional adjustment.
- 3) Extrapolation from short-term studies to lifetime exposure. An additional factor of 10 is applied for short-term studies, such as those used to establish exposure limits to RF radiation, to provide lifetime protection from chronic exposure. This is of particular importance considering the remarkably short periods over which RFR toxicity was originally assessed [10, 11].
- 4) Database insufficiencies. Finally, an uncertainty factor of 3-to-10 is applied for database inadequacy, i.e., for incomplete characterization of an agent’s toxicity. The behavioral studies [10, 11] that were used to establish the FCC and ICNIRP exposure limits to RFR do not provide a full characterization of the effects of this type of radiation nor did they identify the most sensitive adverse effect of RFR exposures.

Basing exposure limits to RFR on the behavioral studies in rats and monkeys [10, 11, 90, 91] would require the application of a composite uncertainty factor of about 900 to 10,000 to be consistent with approaches used by public health agencies to establish protective exposure limits for workers and the general population. Based on the size of the needed uncertainty/safety factor, the

data sets used by the FCC and ICNIRP are clearly inadequate to establish RF exposure limits with reasonable confidence. The arbitrarily selected safety factors of 10 for workers and 50 for the general population by the FCC and ICNIRP are woefully inadequate for protecting exposed populations.

When uncertainty/safety factors are applied to a misrepresented threshold exposure value for adverse effects, the resulting level does not provide assurance of health protection for the general population exposed to that agent. Studies cited above [18, 22, 91, 92, 96] show that the whole-body SAR of 4 W/kg is not a threshold level for adverse effects caused by RFR. In a recent quantitative analysis of various adverse health effects from the NTP study, Uche and Naidenko [185] showed that the permissible whole-body SAR of 0.08 W/kg (based on a 50-fold reduction of the assumed threshold SAR of 4 W/kg) was 20–40-fold higher than health protective SAR values derived by benchmark dose modelling of NTP data for cardiomyopathy (following application of 10-fold safety factors for interspecies and intraspecies variability). The approaches used by these authors are consistent with methodologies recommended by the US Environmental Protection Agency for quantifying health risks for toxic and carcinogenic environmental agents [1, 182]. Thus, a 50-fold reduction of the assumed threshold whole-body SAR of 4 W/kg is inadequate to protect the health of the general population from exposure to RF radiation.

Assumption 10) *A 10-fold safety factor for whole body exposure to RF radiation is adequate for protecting workers to any health risks from RF radiation.*

When RFR exposure limits were implemented in 1997, the rationale given for the difference in safety factors for the general population (50-fold) and for workers (10-fold) was “based on the exposure periods of the two populations, rounded to one digit (40 work hours per week/168 hours per week = ~0.2)” [6]. In addition to differences in exposure periods between workers and the general population, ICNIRP rationalizes the appropriateness of the lower safety factor for workers because “occupationally-exposed individuals can be considered a more homogeneous group than the general population,” they are, “in general, relatively healthy adults within a limited age range,” and “occupationally-exposed individuals should be operating under controlled conditions and be informed about the risks associated with non-ionizing radiation exposure for their specific situation and how to reduce these risks” [23]. In contrast, “the general public are, in most cases, unaware of their exposure to non-ionizing radiation and, without education, cannot

reasonably be expected to take precautions to minimize or avoid any adverse effects of exposure.”

The assumption that workers are trained in understanding health risks associated with exposure to RFR and in mitigating those risks to the greatest possible degree is not correct because neither the FCC nor the ICNIRP guidelines recognize any health effects from RFR at SARs below 4 W/kg, and the exposure limits authorized by the FCC and ICNIRP do not consider health effects from long-term exposures [3, 5]. The only health effect addressed by the FCC and ICNIRP is tissue damage due to excessive heating from acute exposures. Thus, the 10-fold reduction from the threshold whole-body SAR calculated from acute behavioral studies in rats and monkeys is inadequate for protecting the health of workers exposed long-term to RFR (see comments under assumption #9). There are no data demonstrating the adequacy of this arbitrarily chosen safety/uncertainty factor for occupationally-exposed workers, while on the contrary, excess cancer risks have been associated with exposure to RFR workers who operate radar and communication systems in military and occupational settings [186].

Assumption 11) *Exposure of any gram of cube-shaped tissue up to 1.6 W/kg, or 10g of cube-shaped tissue up to 2 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in the general population.*

Tissue dosimetry was analysed in the NTP study of cell phone RF radiation in rats and mice [187]. In rats, whole body exposures during the 10-minute on cycles were 1.5, 3.0, or 6.0 W/kg, and the brain and heart SARs varied from the whole-body SARs by about 7% to under 2-fold for the brain and heart, respectively. A quantitative risk assessment of the NTP tumor incidence data is needed to evaluate organ-specific cancer risk. The FDA [19] nomination to the NTP recognized the need for “large well-planned animal experiments to provide the basis to assess the risk to human health of wireless communications devices.” However, more than 3 years after an external peer-review of the NTP studies found “clear evidence of carcinogenic activity,” the FDA [109] has continued to downplay the importance of these findings and avoid conducting a quantitative risk assessment of the tumor data that they (the FDA) originally requested. In contrast to the FDA, Uche and Naidenko [185] analysed the NTP data on cardiomyopathy by a benchmark dose approach and found that the 10% extra risk level for this effect was in the range of a whole-body SAR of 0.2 to 0.4 W/kg. Thus, there is an increased risk (greater than 10%) of developing cardiomyopathy at local tissue SARs below 1.6 or 2.0 W/kg.

The peak spatial specific absorption rate (psSAR), as used by ICNIRP and the FCC, is an inadequate dosimetric of RF radiation at frequencies above 1 GHz. The psSAR is calculated by averaging fixed cubic volumes containing a given amount of mass, and assumes a homogeneous material with a given mass density. The ICNIRP recommendation is to average cubic volumes containing 10 g of tissue (10-g-psSAR), while the FCC recommendation is to average cubic volumes containing 1 g of tissue (1-g-psSAR). Current recommendations limit the use of psSAR to frequencies up to 6 GHz [3, 5].

An evaluation of the utility of using psSAR as a dosimetric parameter at different frequencies ranging from 100 MHz to 26 GHz and with cube sizes ranging from 10 mg to 10 g is shown in Additional file 2: Appendix 2. For the smaller cubes and lower frequencies, averaging in the cube does not underestimate the maximum value on the cube surface, but at higher frequencies the psSAR averaged on larger cubes can be several-fold lower than the psSAR averaged on smaller cubes. For example, at 2.45 GHz, averaging over a 10-g cube underestimates by 4 dB (approximately 2.5-fold) the psSAR averaged in smaller cubes, while for 5.8 GHz, averaging over a 10-g cube underestimates the psSAR by 12 dB (approximately 16-fold) compared with averaging in a 10-mg cube, and by 6 dB (approximately 4-fold) compared with averaging over a 1-g cube. When the frequency is increased, the underestimation of the psSAR averaged in larger cubes (e.g. 10 g or 1 g) compared to smaller cubes (e.g. 100 mg and 10 mg) becomes more pronounced. Considering the 10-g cube, the difference between the psSAR for 5.8 GHz EMF compared to 0.9 GHz EMF is around 7 dB (or approximately 5-fold underestimation). These large differences are due to reduced penetration of EMFs at higher frequencies. Therefore, the ICNIRP's 10-g-psSAR and FCC's 1-g-psSAR recommendations do not provide reliable dosimetric parameters to evaluate EMF absorption above 1 GHz.

The SAR averaging over a 10-g cube is also flawed for assessing carcinogenicity because it is too large a volume to focus on stem cells and their important role in carcinogenesis. Human stem cells were more sensitive to RFR exposures from GSM and UMTS mobile phones than lymphocytes and fibroblasts [175]. Instead of a random distribution of targets for carcinogenesis, localized distribution of SAR in smaller volumes is needed to more accurately characterize relationships between SAR and tumor induction. From the point of view of stem cell organization, the volume of SAR determinations may be especially important for setting safety limits for children, because most stem cells and their niches are spatially and temporally transient during brain development [188].

Assumption 12) *Exposure of any gram of cube-shaped tissue up to 8 W/kg, or 10 g of cube-shaped tissue up to 10 W/kg, (duration not specified) will not increase the risk of that tissue to any toxic or carcinogenic effects in workers.*

Based on the analyses of tissue dosimetry in the NTP study [187], organ-specific toxic and carcinogenic effects were observed in rats at local tissue SARs that were much lower than 8 or 10 W/kg [18]. The tissue dosimetry in the NTP study and the inadequacy of the local SAR as specified by ICNIRP and the FCC is described in assumption #9.

F. Environmental exposure to RF radiation

Assumption 13) *There is no concern for environmental effects of RF radiation or for effects on wildlife or household pets.*

While background levels of RF-EMF are increasing in the environment, including rural remote areas [189], neither the FCC nor the ICNIRP take into consideration effects of this radiation on wildlife. The constant movement of most wildlife species in and out of varying artificial EMF can result in high exposures near communication structures, especially for flying species such as birds and insects. There is a substantial amount of scientific literature on the disrupting effects of RFR on wildlife (e.g., [190–206]).

Many nonhuman species use Earth's geomagnetic fields for activities such as orientation and seasonal migration, food finding, mating, nest and den building [190]. For example, migratory bird species [191, 192], honeybees [193], bats [194], fish [195–197], and numerous other species sense Earth's magnetic fields with specialized sensory receptors. Mechanisms likely involved in magneto-reception include magnetic induction of weak electric signals in specialized sensory receptors [198], magneto-mechanical interactions with the iron-based crystal magnetite [194], and/or free-radical interactions with cryptochrome photoreceptors [191, 192]. Each of these sensing processes shows extreme sensitivity to low intensity changes in electromagnetic fields. For a fuller description of the mechanisms by which non-human species use magneto-reception to perform essential life activities see Levitt et al. [190].

The following studies represent a few of the many examples of the disrupting effects of low-level exposures to RF-EMF on magneto-reception and the natural behavior of wildlife. Oscillating magnetic fields have been reported to disrupt the ability of migratory birds to orient and navigate in Earth's geomagnetic field [199–202].

Garden warblers became disoriented by exposure to a weak oscillating magnetic field of 1.403 MHz at an intensity as low as 2–3 nT [200]. The orientation of European robins that use Earth's magnetic field for compass orientation was completely disrupted by exposure to electromagnetic noise in the frequency range of 50 kHz to 5 MHz or a broadband noise-modulated ELF covering the range ~2 kHz to ~9 MHz [199, 201]. RFR in the low MHz range (7.0 MHz of 480 nT or 1.315 MHz of 15 nT) has been shown to disable the magneto-reception avian compass as long as the exposure was present [202].

In addition to effects on migratory birds, Landler et al. [203] found that exposure to a low-level magnetic field (1.43 MHz at an intensity of 30–52 nT) disrupted the natural orientation of juvenile turtles hatched on land. GSM-modulated 900 MHz RF radiation caused ants to lose their visual and olfactory memory for finding food [166]. Navigational abilities of trout were reduced when reared under conditions in which magnetic fields were spatially distorted [204].

Activities of honeybees are also disrupted by exposure to RF radiation. GSM-modulated cell phone radiation (900 MHz) caused a reduction in egg laying by queen bees and depletion of beehive pollen and honey counts [205]. GSM-modulated cell phone radiation (900 MHz) reduced hatching and altered pupal development of honey queen bee larvae [206].

The lack of consideration of chronic low-level RF radiation exposure on wildlife could result in dangerously disruptive effects on fragile ecosystems and on the behavior and survival of species that have long existed in Earth's natural environment.

G. 5G (5th generation wireless)

Assumption 14) *No health effects data are needed for exposures to 5G; safety is assumed because penetration is limited to the skin (“minimal body penetration”).*

Fifth generation (5G) wireless communication systems are being deployed worldwide to provide higher data transfer rates with shorter lag times between massive numbers of connected wireless devices. To provide faster transfer of large amounts of data (up to 20 gigabits per second peak data rates), the frequency range for 5G includes millimeter waves (30 to 300 GHz), in addition to carrier frequencies as low as 600 MHz. Extremely high frequency millimeter waves (MMW) that transmit large amounts of data to user devices are directed into narrow beams by line-of-sight transmission with beamforming antennas. Because millimeter waves do not penetrate solid structures such as building materials, hills, foliage, etc., and travel only short distances (a few hundred

meters), denser networks of base-stations with massive Multiple Input/Multiple Output (MIMO) transmitters and receivers in millions of small cell towers are being installed on structures such as utility poles. These features can lead to much closer proximity between humans and radiation-emitting antennas, and thereby change individual peak and average exposures to RFR.

For a 5G frequency of 26 GHz, EMF absorption is very superficial, which means that for typical human skin, more than 86% of the incident power is absorbed within the first millimeter. The skin penetration depth was computed as 1 mm based on the electrical conductivity of the skin and its electrical permittivity [5, 207]. This is expected to bring the SAR in this tissue well above the recommended limits ([208], and Additional file 2: Appendix 2). This is also expected to be harmful to very small species, such as birds and other small animals (e.g., insects) [209]. It is often claimed that because of its shallow penetration, exposure to high frequency 5G radiation is safe, and that the only effect is tissue heating [210]. However, this view ignores the deeper penetration of the ELF components of modulated RF signals, which are rated on the basis of heat alone, as well as the effects of short bursts of heat from pulsed signals [211, 212]. Within the first 1 mm of skin, cells divide to renew the stratum corneum (a consideration for skin cancer), and nerve endings in the dermis are situated within 0.6 mm (eyelids) to 3 mm (feet) of the surface (a consideration for neurological effects). Ultraviolet light, which exerts its action at a penetration depth of less than 0.1 mm [213, 214] is a recognized cause of skin cancer [87].

The higher the frequency of electromagnetic waves, the shorter the wavelength and the shallower the penetration of energy into exposed people or animals. For example, penetration depth in the human body is about 8 mm at 6 GHz and 0.92 mm at 30 GHz [5]. Because of the minimal depth of energy absorption at frequencies above 6 GHz, the FCC and ICNIRP have based exposure limits on power density instead of on SAR levels. The FCC [3] proposed a general localized power density exposure limit of 4 mW/cm² averaged over 1 cm² and not to exceed 30 minutes for 5G services up to 3000 GHz for the general population, claiming that this exposure is consistent with the peak spatial-average SAR of 1.6 W/kg averaged over any 1 g of tissue at 6 GHz. ICNIRP's [5] exposure limits for 5G are an absorbed power density of 200 W/m² (0.2 W/cm²) averaged over 4 cm² and a 6-minute interval for frequencies up to 30 GHz, and 400 W/m² (0.4 mW/cm²) averaged over 1 cm² and a 6-minute interval for frequencies of 30 GHz to 300 GHz.

Because of its minimal penetration, exposure to 5G radiation results in higher energy intensity on the skin and other directly-exposed body parts, such as the eye

cornea or lens. However, the skin, which is the largest organ in the human body, provides important functions such as acting as a protective physical and immunological barrier against mechanical injury, infection by pathogenic microorganisms, and entry of toxic substances. In addition, skin cancers, including basal cell carcinomas and squamous cell carcinomas, are the most prevalent human cancers, while melanomas are highly metastatic and increasing in prevalence. Although the high incidence of skin cancers are largely attributed to exposure to ultraviolet light, no studies have been reported on the effects of 5G radiation on (i) the skin's ability to provide protection from pathogenic microorganisms, (ii) the possible exacerbation of other skin diseases, (iii) promotion of sunlight-induced skin cancers, or (iv) initiation of skin cancer by itself. Information is also lacking on the effects of 5G radiation on nervous and immune systems which are also exposed even by the shallower penetration of MMW.

Another important factor is the maximum bandwidth with 5G radiation, which is up to 100 MHz in the frequency range of 450 MHz to 6 GHz, and up to 400 MHz in the ranges from 24 GHz to 52 GHz, compared to previous types of mobile communication where bandwidth is limited to 20 MHz. Because many studies indicated frequency-dependent, non-thermal RF effects from mobile communication RFR [43, 177] and for MMW effects [215, 216], the possibility of effective frequency windows for biological effects would increase with the increased bandwidth of 5G radiation.

Another consideration for effects of 5G exposures on human health is that radiation pulses created by extremely fast data transmission rates have the potential to generate bursts of energy that can travel much deeper than predicted by conventional models [217, 218]. Neufeld and Kuster [105] showed that repetitive pulses of data in bursts with short exposures to 5G can cause localized temperature spikes in the skin leading to permanent tissue damage even when the average power density values were within ICNIRP's acceptable safety limits. The authors urged the setting of new thermal safety standards to address the kind of health risks possible with 5G technology:

“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 millimetre-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...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...”

Areas of uncertainty and health concerns with 5G radiation include potential increase in skin cancer rates with (or possibly without) co-exposure to sunlight, exacerbation of skin diseases, greater susceptibility to pathogenic microorganisms, corneal damage or early development of cataracts, testicular effects, and possible resonant-enhanced absorption due to skin structures [219]. One of the complex technical challenges in relation to human exposure to 5G millimeter waves is that the unpredictable propagation patterns that could result in unacceptable levels of human exposure to electromagnetic radiation are not well understood [220]. Although MMW are almost completely absorbed within 1–2 mm in biologically-equivalent tissues, their effects may penetrate deeper in a live human body possibly by affecting signal transduction pathways. Thus, there are too many uncertainties with exposure to 5G to support an assumption of safety without adequate health effects data. There are no adequate studies on health effects from short-term or long-term exposures to 5G radiation in animal models or in humans.

Discussion

To develop health-based exposure limits for toxic and carcinogenic substances, regulatory agencies typically rely on available scientific evidence about the agent under review. In the mid- and late-1990s when the FCC [4] and the ICNIRP [9] initially established exposure limits for RFR, the prevailing assumptions were that any adverse effects from exposure to RFR were due to excessive heating because non-ionizing radiation did not have sufficient energy to break chemical bonds or damage DNA. However, non-thermal effects of RFR are demonstrated from studies that find different effects with exposure to continuous waves versus pulsed or modulated waves at the same frequency and the same SAR or power density, e.g., [221–226], and from studies that show adverse effects at very low exposure intensities, e.g., [78, 96].

Acute exposure studies conducted in rats and monkeys in the 1980s [10, 11] suggested that an SAR of 4 W/kg could be a threshold dose for behavioral effects. Because this SAR was associated with an approximate increase in body temperature of 1 °C, it was again assumed that no adverse health effects would occur if increases in core

body temperature were less than 1°C. From this putative threshold dose a “safety factor” of 10 was applied for occupational exposures and an additional factor of 5 (50x total) was applied for the general population, resulting in exposure limits in which the whole-body SAR was less than 0.4 W/kg for workers and 0.08 W/kg for the general population. However, realizing that local parts of the body could receive doses of RFR that were 10 to 20 times higher than the whole-body SARs, local peak exposure limits were set by the FCC at SARs 20-times higher than the whole-body SARs, i.e., 8 W/kg averaged over any 1-g of tissue for localized exposures for workers and 1.6 W/kg averaged over any 1-g for the general population [3, 4]. ICNIRP opted for partial body exposures that would not exceed 2.0 W/kg averaged over any 10g of cube-shaped tissue for the general population [5, 9]. To rationalize the smaller safety factor for workers (10-fold) versus the general population (50-fold), one claim made by ICNIRP [24] is that workers are informed about risks associated with non-ionizing radiation exposure and how to reduce these risks, whereas “the general public are, in most cases, unaware of their exposure to non-ionizing radiation and, without education, cannot reasonably be expected to take precautions to minimize or avoid any adverse effects of exposure.” From a public health perspective, the FCC and ICNIRP should make the public aware of their exposures to RFR and promote precautionary measures to minimize potential adverse effects, especially for children and pregnant women. Eight practical recommendations by the International EMF Scientist Appeal aimed at protecting and educating the public about potential adverse health effects from exposures to non-ionizing EMFs [227] are shown in Table 2.

The acute behavioral studies that provide the basis for the FCC’s and ICNIRP’s exposure limits lacked any information on potential effects of RF radiation that can occur after longer durations of exposure, and they did not address effects of carrier wave modulations used in wireless communications. Research on RFR conducted over

the past 25 years has produced thousands of scientific papers, with many demonstrating that acute behavioral studies are inadequate for developing health protective exposure limits for humans and wildlife, and that inherent assumptions underlying the FCC’s and ICNIRP’s exposure limits are not valid. First, 4 W/kg is not a threshold SAR for health effects caused by RFR exposures; experimental studies at lower doses and for longer durations of exposure demonstrated cardiomyopathy, carcinogenicity, DNA damage, neurological effects, increased permeability of the blood brain barrier, and sperm damage (see Assumptions 1–3). Multiple robust epidemiologic studies on cell phone radiation have found increased risks for brain tumors (Assumption 6), and these are supported by clear evidence of carcinogenicity of the same cell types (glial cell and Schwann cell) from animal studies. Even studies conducted by D’Andrea et al. [89, 90] before the limits were adopted found behavioral disruption in rats exposed to RFR for 14 or 16 weeks at mean SARs of 0.7 W/kg and at 1.23 W/kg. A combination of exposure duration and exposure intensity would be more appropriate for setting safety standards for exposure to RFR from mobile communication systems including mobile phones, base stations, and WiFi.

More than 120 studies have demonstrated oxidative effects associated with exposure to low intensity RFR (Additional file 1: Appendix 1). DNA damage that has been reported in studies of RFR was most likely caused by induction of oxidative stress, which is a key characteristic of human carcinogens [88], rather than by direct ionization (Assumption 2). The generation of reactive oxygen species has also been linked to DNA damage and the carcinogenicity of UVA radiation [87] and asbestos [228]. Despite the enormous amount of scientific evidence of low-dose effects of RFR, the IEEE [229] maintains that behavioral disruption is still the most sensitive and reproducible effect of RFR. It is this opinion that contributed to the FCC [3] and ICNIRP [5] reaffirming their previous exposure limits to RFR.

Table 2 Precautionary Measures Recommended by the International EMF Scientist Appeal

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- 1) Priority should be given to protect children and pregnant women
 - 2) Guidelines and regulatory standards should be strengthened
 - 3) Manufacturers should be encouraged to develop safer technologies
 - 4) The public should be fully informed about the potential health risks from electromagnetic energy and taught harm reduction strategies
 - 5) Medical professionals need to be educated about the biological effects of electromagnetic energy and be provided training on treatment of patients with electromagnetic sensitivity
 - 6) Governments need to fund training and research on electromagnetic fields and health that is independent of industry
 - 7) The media should disclose experts’ financial relationships with industry when citing their opinions regarding health and safety aspects of EMF-emitting technologies
 - 8) Radiation-free areas need to be established, especially for individuals with EHS
-

Other concerns about the current exposure limits for RFR are that they do not consider potential synergistic effects due to co-exposure to other toxic or carcinogenic agents, the impact of pulsed radiation or frequency modulations, multiple frequencies, differences in levels of absorption or of susceptibility by children, or differences among individuals in their sensitivity to RFR (see Assumptions 4, 5, 7, 8). Currently, children's cumulative exposures are much higher than previous generations and they continue to increase [230]. ICNIRP [23, 179] acknowledged that their guidelines do not accommodate sensitive subgroups and admit to difficulties separating "biological effects" from "health effects." Neurological symptoms, some of which are acknowledged by ICNIRP and currently being experienced by persons with EHS, are most certainly non-thermal "health effects" that need to be mitigated by providing environments with reduced exposures to anthropogenic EMF for hypersensitive individuals.

The debilitating effects and restrictions suffered by adults and children with EHS constitutes a contravention of the 2010 Equalities Act, Human Rights Act and other ethical and legal frameworks. Failure to respond and appropriately safeguard this group is already causing preventable morbidity, mortality and economic deficit due to lost workdays, compensations for health damages and increased healthcare costs. Conversely, accommodating this group by, as suggested by ICNIRP [179], acting to 'adjust the guidelines for the general population to include such groups' would not only lessen the negative impacts for people with EHS, but would also improve public health more broadly, given the other NIR-related health concerns that are highlighted in this paper.

Basing local tissue exposure limits on 1-g [3] or 10-g [5] cubes substantially underestimates the peak spatial SAR compared to basing local tissue exposure limits on smaller cubes (e.g., 100 mg or 10 mg), and therefore are not reliable dosimetric parameters to evaluate EMF absorption at frequencies above 1 GHz (Assumptions 11, 12). The volumes specified by the FCC and ICNIRP for local tissue SAR limits are too large to focus on stem cells which are important targets for carcinogenesis. To reduce health risks from exposures to RFR, limits for localized distribution of the SAR should be based on 100 mg, or preferably 10 mg cubes.

Another important deficiency raised in this paper is that neither the FCC nor ICNIRP addresses concerns for environmental effects of RFR on wildlife, even though there is extensive literature demonstrating the disrupting effects of RFR on wildlife behavior (Assumption 13).

The arbitrarily selected uncertainty/safety factors applied to the putative threshold SAR for RFR are woefully inadequate for protecting public health

(Assumptions 9, 10). Based on the way the US Environmental Protection Agency, the International Council for Harmonization, and the National Institute for Occupational Safety and Health (US NIOSH) apply uncertainty/safety factors to a no-observed-adverse-effect level (NOAEL) in experimental animals [182–184], the safety factor for RFR would be at least 900 to 10,000, which is 18 to 200 times larger than the safety factor recommended by the FCC and ICNIRP for the general population. This large safety factor is based on adjustments for human variability, lifetime exposure from short-term studies, and database insufficiencies that include incomplete characterization of the toxicity of RFR. Clearly, the acute behavioral studies that served as the basis for the current exposure limits for RFR are not suitable for characterizing human health risks associated with long-term exposure to this type of radiation. The NCRP report from 1986 [6] and the ANSI/IEEE document from 1992 [7] recognized that when future studies on biological effects of RFR become available including effects of chronic exposures or evidence of non-thermal interactions there will be a need to evaluate and possibly revise exposure standards. When the FCC [3] and ICNIRP [5] reaffirmed their exposure limits from the 1990s, they dismissed the scientific evidence that invalidated the assumptions that underlie the basis for those exposure limits. An independent re-evaluation of RFR exposure limits based on the scientific knowledge gained over the past 25 years is needed and is long overdue. This evaluation should be performed by scientists and medical doctors who have no conflicting interests and who have expertise in RF-EMF exposure and dosimetry, toxicology, epidemiology, clinical assessment, and risk assessment. Special precautions should be taken to ensure that interpretations of health effects data and the setting of exposure limits for RFR are not influenced by the military or the telecommunications industry. In the meantime, manufacturers should be obliged to develop safer technologies [227].

Finally, we note our concern about the worldwide deployment of 5G communication networks for faster transfer of large amounts of data, but with no adequate health effects studies demonstrating the safety of high frequency millimeter waves. Because of limitations of the penetration and distance of travel of millimeter waves, dense networks of base stations are being mounted on structures such as utility poles in highly populated cities. Also, because the absorption of EMF at frequencies above 6 GHz is minimal, ICNIRP [5] has specified absorbed power density (S_{ab}) as the dosimetric parameter for "heating effects" at the higher frequencies. S_{ab} is a function of the incident power density (S_{inc}) and the input reflection coefficient (Γ). In near field scenarios, the S_{inc} does not have a singular value; this is largely due

to the heterogeneous nature of human body tissues and their relevant parameters (such as the permittivity, equivalent conductivity, mass density), which vary in different body regions and with frequency. Therefore, unless a powerful EMF simulation method together with realistic human models are used, the S_{inc} and the reflection coefficient values would be difficult to accurately estimate, making the resulting S_{ab} unreliable.

The assumption that 5G is safe at the power density limits recommended by ICNIRP (50 W/m² and 10 W/m² averaged over 6 min for occupational and 30 min for public exposures, respectively) because of its minimal penetration into the body does not justify the dismissal of the need for health effects studies prior to implementing 5G networks. The new communication networks will result in exposures to a form of radiation that has not been previously experienced by the public at large (Assumption 14). The implementation of 5G technology without adequate health effects information raises many questions, such as: Will exposure to 5G radiation: (i) compromise the skin's ability to provide protection from pathogenic microorganisms? (ii) will it exacerbate the development of skin diseases? (iii) will it increase the risk of sunlight-induced skin cancers? (iv) will it increase the risk of damage to the lens or cornea? (v) will it increase the risk of testicular damage? (vi) will it exert deeper tissue effects either indirectly following effects on superficial structures or more directly due to deeper penetration of the ELF components of modulated RF signals? (vii) will it adversely affect wildlife populations? Answers to these questions and others that are relevant to human and wildlife health should be provided *before* widespread exposures to 5G radiation occur, not afterwards. Based on lessons that should have been learned from studies on RFR at frequencies below 6 GHz, we should no longer rely on the untested assumption that current or future wireless technology, including 5G, is safe without adequate testing. To do otherwise is not in the best interest of either public or environmental health.

Abbreviations

ANSI: American National Standards Institute; CDMA: Code-division multiple access; dB: Decibel; DECT: Digital enhanced cordless technology; EHS: Electromagnetic hypersensitivity; ELF: Extremely low frequency; EMF: Electromagnetic field; FCC: Federal Communications Commission; FDA: Food and Drug Administration; GHz: Gigahertz; GBM: Glioblastoma multiforme brain cancer; GSM: Global system for mobile communication; IARC: International Agency for Research on Cancer; ICNIRP: International Commission on Non-Ionizing Radiation Protection; IEEE: Institute of Electrical and Electronics Engineers; LTE: Long Term Evolution (4G); MMW: Millimeter wave; NCRP: National Council on Radiation Protection and Measurements; NIR: Non-ionizing radiation; nT: Nanotesla; NTP: National Toxicology Program; 8-OHdG: 8-hydroxy-2'-deoxyguanosine; psSAR: Peak spatial specific absorption rate; RFR: Radiofrequency radiation; ROS: Reactive oxygen species; SAR: Specific absorption rate; UMTS: Universal mobile telecommunications service (3G); UVR: Ultraviolet radiation; 5G: 5th generation wireless.

Supplementary Information

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Additional file 1: Appendix 1 Table 1. Studies demonstrating increased oxidative DNA damage and other indicators of oxidative stress at SAR < 4W/kg.

Additional file 2: Appendix 2. On the Inadequacy of the psSAR Dosimetric Parameter at Frequencies above 1 GHz. **Table 1.** Electric permittivity and electric conductivity of the gray matter. **Figure 1.** A block of gray matter radiated by different frequencies. The highlighted cubes are of 10g, 1g, 100mg and 10mg. **Fig. 2.** A block of gray matter radiated by different frequencies. The highlighted cubes are of 10g, 1g, 100mg and 10mg. **Fig. 3.** Electric field intensity averaged in each cube for different frequencies: in the left axis, the electric field is in dB and in the right axis the electric field is in V/m normalized to 100V/m.

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Authors' contributions

IB, AD, CF, LH, PH, KK, DM, EMB, RLM, and IY drafted the initial sections of this manuscript: by IB (factors affecting dosimetry), AD and CF (absorption in children versus adults, peak spatial specific absorption rate), LH (human brain cancer risk), KK (sperm damage), DM and DM (5G), EMB (electromagnetic hypersensitivity), RLM (cardiomyopathy, carcinogenicity, neurologic effects, safety factors), and IY (oxidative stress and DNA damage). IY prepared Appendix 1, and AD and CF prepared Appendix 2. The authors who drafted sections of the manuscript, as well as CB, KC, SD, EK, AM, JMM, and WS reviewed multiple manuscript drafts and made revisions. All authors reviewed and approved the final manuscript.

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Re: Links for Info on 5G (for you to have available if needed)

Kayla DiCristina <kayla@landofsky.org>

Thu 11/10/2022 9:09 AM

To: L J <lhjohnson62@gmail.com>

Hi Liz,

Thanks for sending these along. I will transmit these to the Board digitally but will not be able to print them before our meeting. As stated in my previous email, the scope of the text amendment to the Wireless Communications Ordinance is to bring the Ordinance into compliance with the new State Regs. Changes to content should be addressed at a later date.

Best,

Kayla DiCristina, AICP

(*For inquiries regarding the Town of Montreat, please see below)

Regional Planner | Economic and Community Development

Land of Sky Regional Council

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From: L J <lhjohnson62@gmail.com>**Sent:** Thursday, November 10, 2022 8:33 AM**To:** Kayla DiCristina <kayla@landofsky.org>**Subject:** Links for Info on 5G (for you to have available if needed)**Be Advised: This email originated from outside Land of Sky**

Hi Kayla-

I am just gathering some info incase there is discussion opportunity about 5G today. I wanted you to have the links incase we need to pass them on to the rest of the P and Z. I will bring this up if it is appropriate during our meeting. I might be sending a couple more in other emails. See you later this morning.

Thanks so much,

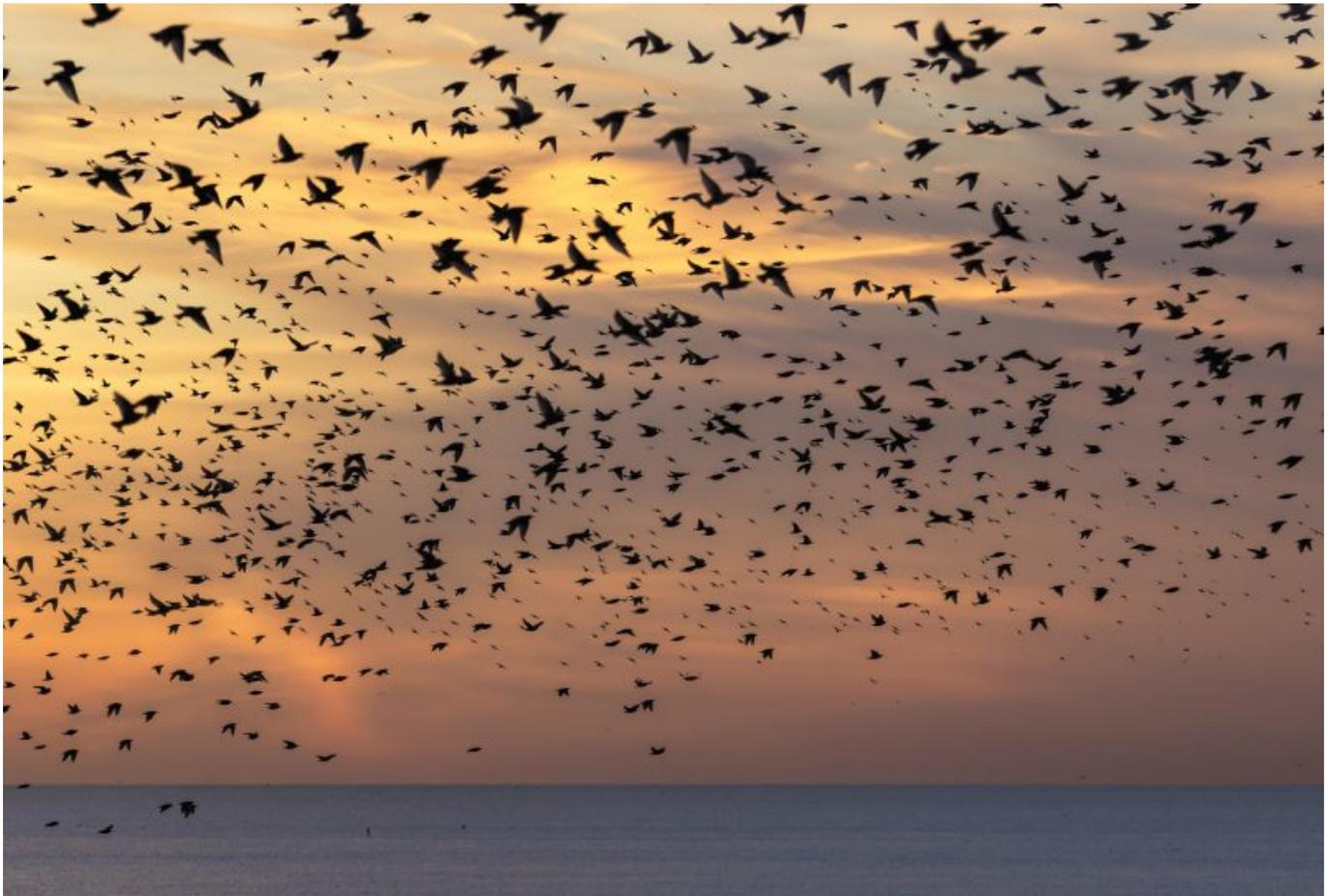
Liz

Thanks,

Liz

<https://jsis.washington.edu/news/what-will-5g-mean-for-the-environment/>

What Will 5G Mean for the Environment?



Beginning 2020, the fifth generation of wireless technology is expected to be widely implemented throughout the world. The new network, called 5G, promises to give faster speeds and a higher capacity for the use of more devices. However, while companies from countries such as the United States and China are competing to be the first to deliver 5G to the consumer, the environmental impacts of the new network are being overlooked. In a time when the environment is at its most delicate, overlooking these impacts is extremely risky for future generations.

The main environmental issues associated with the implementation of the 5G network come with the manufacturing of the many component parts of the 5G infrastructure. In addition, the proliferation of new devices that will use the 5G network that is tied to the acceleration of demand from consumers for new 5G-dependent devices will have serious environmental consequences.

The 5G network will inevitably cause a large increase in energy usage among consumers, which is already one of the main contributors to climate change. Additionally, the manufacturing and maintenance of the new technologies associated with 5G creates waste and uses important resources that have detrimental consequences for the environment. 5G networks use technology that has harmful effects on birds, which in turn has cascading effects through entire ecosystems. And, while 5G developers are seeking to create a network that has fewer environmental impacts than past networks, there is still room for improvement and the consequences of 5G should be considered before it is widely rolled out.

What is 5G?

5G stands for the fifth generation of wireless technology. It is the wave of wireless technology surpassing the 4G network that is used now. Previous generations brought the first cell phones (1G), text messaging (2G), online capabilities (3G), and faster speed (4G).[1] The fifth generation aims to increase the speed of data movement, be more responsive, and allow for greater connectivity of devices simultaneously.[2] This means that 5G will allow for nearly instantaneous downloading of data that, with the current network, would take hours. For example, downloading a movie using 5G would take mere seconds. These new improvements will allow for self-driving cars, massive expansion of Internet of Things (IoT) device use, and acceleration of new technological advancements used in everyday activities by a much wider range of people.

While 5G is not fully developed, it is expected to consist of at least five new technologies that allow it to perform much more complicated tasks at faster speeds. The new technologies 5G will use are hardware that works with much higher frequencies (millimeter wavelengths), small cells, massive MIMO (multiple input multiple output), beamforming, and full duplex.[3] Working together, these new technologies will expand the potential of many of the devices used today and devices being developed for the future.

Millimeter waves are a higher frequency wavelength than the radio wavelength generally used in wireless transmission today.[4] The use of this portion of the spectrum corresponds to higher frequency and shorter wavelengths, in this case in the millimeter range (vs the lower radio frequencies where the wavelengths can be in the meters to hundreds of kilometers). Higher frequency waves allow for more devices to be connected to the same network at the same time, because there is more space available compared to the radio waves that are used today. The use of this portion of the spectrum has much longer wavelengths than of that anticipated for a portion of the 5G implementation. The waves in use now can measure up to tens of centimeters, while the new 5G waves would be no greater than ten millimeters.[5] The millimeter waves will create more transmission space for the ever-expanding number of people and devices crowding the current networks. The millimeter waves will create more space for devices to be used by consumers, which will increase energy usage, subsequently leading to increased global warming.

Millimeter waves are very weak in their ability to connect two devices, which is why 5G needs something called "small cells" to give full, uninterrupted coverage. Small cells are essentially miniature cell towers that would be placed 250 meters apart throughout cities and other areas needing coverage.[6] The small cells are necessary as emissions [or signals] at this higher frequency/shorter wavelength have more difficulty passing through solid objects and are even easily intercepted by rain.[7] The small cells could be placed on anything from trees to street lights to the sides of businesses and homes to maximize connection and limit "dead zones" (areas where connections are lost).[8]

The next new piece of technology necessary for 5G is massive MIMO, which stands for multiple input multiple output. The MIMO describes the capacity of 5G's base stations, because those base stations would be able to handle a much higher amount of data at any one moment of time. Currently, 4G base stations have around eight transmitters and four receivers which direct the flow of data between devices.[9] 5G will exceed this capacity with the use of massive MIMO that can handle 22 times more ports.[10] Figure 1 shows how a massive MIMO tower would be able to direct a higher number of connections at once. However, massive MIMO causes signals to be crossed more easily.

Crossed signals cause an interruption in the transmission of data from one device to the next due to a clashing of the wavelengths as they travel to their respective destinations. To overcome the cross signals problem, beamforming is needed.

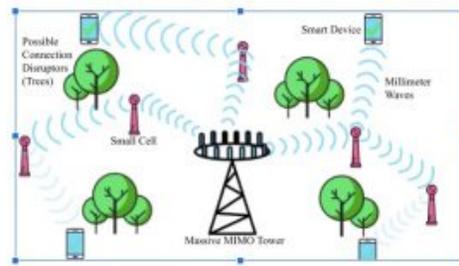


Figure 1. 5G Network of Base Towers, Small Cells, and Stylized Disruptions[11]

To maximize the efficiency of sending data another new technology called beamforming will be used in 5G. For data to be sent to the correct user, a way of directing the wavelengths without interference is necessary. This is done through a technique called beamforming. Beamforming directs where exactly data are being sent by using a variety of antennas to organize signals based on certain characteristics, such as the magnitude of the signal.[12] By directly sending signals to where they need to go, beamforming decreases the chances that a signal is dropped due to the interference of a physical object.

One way that 5G will follow through on its promise of faster data transmission is through sending and receiving data simultaneously.[13] The method that allows for simultaneous input and output of data is called full duplexing. While full duplex capabilities allow for faster transmission of data, there is an issue of signal interference, because of echoes.[14] Full duplexing will cut transmission times in half, because it allows for a response to occur as soon as an input is delivered, eliminating the turnaround time that is seen in transmission today.

Because these technologies are new and untested, it is hard to say how they will impact our environment. This raises another issue: there are impacts that can be anticipated and predicted, but there are also unanticipated impacts because much of the new technologies are untested. Nevertheless, it is possible to anticipate some of detrimental environmental consequences of the new technologies and the 5G network, because we know these technologies will increase exposure to harmful radiation, increase mining of rare minerals, increase waste, and increase energy usage. The main 5G environmental concerns have to do with two of the five new components: the millimeter waves and the small cells.

Increased Energy Usage of the 5G Network

The whole aim of the new 5G network is to allow for more devices to be used by the consumer at faster rates than ever before, because of this goal there will certainly be an increase in energy usage globally. Energy usage is one of the main contributors to climate change today and an increase in energy usage would cause climate change to increase drastically as well. 5G will operate on a higher frequency portion of the spectrum to open new space for more devices. The smaller size of the millimeter waves compared to radio frequency waves allows for more data to be shared more quickly and creates a wide bandwidth that can support much larger tasks.[15] While the idea of more space for devices to be used is great for consumers, this will lead to a spike in energy usage for

two reasons – the technology itself is energy demanding and will increase demand for more electronic devices. The ability for more devices to be used on the same network creates more incentive for consumers to buy electronics and use them more often. This will have a harmful impact on the environment through increased energy use.

Climate change has several underlying contributors; however, energy usage is gaining attention in its severity with regards to perpetuating climate change. Before 5G has even been released, about 2% of the world's greenhouse gas emissions can be attributed to the ICT industry.[16] While 2% may not seem like a very large portion, it translates to around 860 million tons of greenhouse gas emissions. [17] Greenhouse gas emissions are the main contributors to natural disasters, such as flooding and drought, which are increasing severity and occurrence every year. Currently, roughly 85% of the energy used in the United States can be attributed to fossil fuel consumption.[18] The dwindling availability of fossil fuels and the environmental burden of releasing these fossil fuels into our atmosphere signal an immediate need to shift to other energy sources. Without a shift to other forms of energy production and the addition of technology allowed by the implementation of 5G, the strain on our environment will rise and the damage may never be repaired. With an increase in energy usage through technology and the implementation of 5G, it can be expected that the climate change issues faced today will only increase.

The overall contribution of carbon dioxide emissions from the ICT industry has a huge impact on climate change and will continue to have even larger impacts without proper actions. In a European Union report, researchers estimated that in order to keep the increase in global temperature below 2° Celsius a decrease in carbon emissions of around 15-30% is necessary by 2020.[19]

Engineers claim that the small cells used to provide the 5G connection will be energy efficient and powered in a sustainable way; however the maintenance and production of these cells is more of an issue. Supporters of the 5G network advocate that the small cells will use solar or wind energy to stay sustainable and green.[20] These devices, labeled "fuel-cell energy servers" will work as clean energy-based generators for the small cells.[21] While implementing base stations that use sustainable energy to function would be a step in the right direction in environmental conservation, it is not the solution to the main issue caused by 5G, which is the impact that the massive amount of new devices in the hands of consumers will have on the amount of energy required to power these devices.

Consumption Increases and 5G Technologies

The wasteful nature of manufacturing and maintenance of both individual devices and the devices used to deliver 5G connection could become a major contributor of climate change. The promise of 5G technology is to expand the number of devices functioning might be the most troubling aspect of the new technology. Cell phones, computers, and other everyday devices are manufactured in a way that puts stress on the environment. A report by the EPA estimated that in 2010, 25% of the world's greenhouse gas emissions comes from electricity and heat production making it the largest single source of emissions.[22] The main gas emitted by this sector is carbon dioxide, due to the burning of natural gas, such as coal, to fuel electricity sources.[23] Carbon dioxide is one of the most

common greenhouse gases seen in our atmosphere, it traps heat in earth's atmosphere trying to escape into space, which causes the atmosphere to warm generating climate change.[24]

Increased consumption of devices is taking a toll on the environment.[25] As consumers gain access to more technologies the cycle of consumption only expands. As new devices are developed, the older devices are thrown out even if they are still functional. Often, big companies will purposefully change their products in ways that make certain partner devices (such as chargers or earphones) unusable—creating demand for new products. Economic incentives mean that companies will continue these practices in spite of the environmental impacts.

One of the main issues with the 5G network and the resulting increase in consumption of technological devices is that the production required for these devices is not sustainable. In the case of making new devices, whether they be new smart-phones or the small cells needed for 5G, the use of nonrenewable metals is required. It is extremely difficult to use metals for manufacturing sustainably, because metals are not a renewable resource.[26] Metals used in the manufacturing of the smart devices frequently used today often cannot be recycled in the same way many household items can be recycled. Because these technologies cannot be recycled, they create tons of waste when they are created and tons of waste when they are thrown away.

There are around six billion mobile devices in use today, with this number expected to increase drastically as the global population increases and new devices enter the market.[27] One estimate of the life-time carbon emissions of a single device—not including related accessories and network connection—is that a device produces a total of 45kg of carbon dioxide at a medium level of usage over three years. This amount of emission is comparable to that of driving the average European car for 300km.[28]

But, the most environmentally taxing stage of a mobile device life cycle is during the production stage, where around 68% of total carbon emissions is produced, equating to 30kg of carbon dioxide. [29] To put this into perspective, an iPhone X weighs approximately 0.174kg, so in order to produce the actual device, 172 iPhone X's worth of carbon dioxide is also created. These emissions vary from person to person and between different devices, but it's possible to estimate the impact one device has on the environment. 5G grants the capacity for more devices to be used, significantly increase the existing carbon footprint of smart devices today.

Energy usage for the ever-growing number of devices on the market and in homes is another environmental threat that would be greatly increased by the new capabilities brought by the 5G network. Often, energy forecasts overlook the amount of energy that will be consumed by new technologies, which leads to a skewed understanding of the actual amount of energy expected to be used.[30] One example of this is with IoT devices.[31] IoT is one of the main aspects of 5G people in the technology field are most excited about. 5G will allow for a larger expansion of IoT into the everyday household.[32] While some IoT devices promise lower energy usage abilities, the 50 billion new IoT devices expected to be produced and used by consumers will surpass the energy used by today's electronics.

The small cells required for the 5G network to properly function causes another issue of waste with the new network. Because of the weak nature of the millimeter waves used in the 5G technology, small cells will need to be placed around 250 meters apart to insure continuous connection.[33] The main issue with these small cells is that the manufacturing and maintenance of these cells will create

a lot of waste. The manufacturing of technology takes a large toll on the environment, due to the consumption of non-renewable resources to produce devices, and technology ending up in landfills. Implementing these small cells into large cities where they must be placed at such a high density will have a drastic impact on technology waste.

Technology is constantly changing and improving, which is one of the huge reasons it has such high economic value. But, when a technological advancement in small cells happens, the current small cells would have to be replaced. The short lifespan of devices created today makes waste predictable and inevitable. In New York City, where there would have to be at least 3,135,200 small cells, the waste created in just one city when a new advancement in small cells is implemented would have overwhelming consequences on the environment. 5G is just one of many examples of how important it is to look at the consequences of new advancements before their implementation. While it is exciting to see new technology that promises to improve everyday life, the consequences of additional waste and energy usage must be considered to preserve a sustainable environment in the future.

The Impact of 5G on Ecosystems

There is some evidence that the new devices and technologies associated with 5G will be harmful to delicate ecosystems. The main component of the 5G network that will affect the earth's ecosystems is the millimeter waves. The millimeter waves that are being used in developing the 5G network have never been used at such scale before. This makes it especially difficult to know how they will impact the environment and certain ecosystems. However, studies have found that there are some harms caused by these new technologies.

The millimeter waves, specifically, have been linked to many disturbances in the ecosystems of birds. In a study by the Centre for Environment and Vocational Studies of Punjab University, researchers observed that after exposure to radiation from a cell tower for just 5-30 minutes, the eggs of sparrows were disfigured.[34] The disfiguration of birds exposed for such a short amount of time to these frequencies is significant considering that the new 5G network will have a much higher density of base stations (small cells) throughout areas needing connection. The potential dangers of having so many small cells all over areas where birds live could cause whole populations of birds to have mutations that threaten their population's survival. Additionally, a study done in Spain showed breeding, nesting, and roosting was negatively affected by microwave radiation emitted by a cell tower.[35] Again, the issue of the increase in the amount of connection conductors in the form of small cells to provide connection with the 5G network is seen to be harmful to species that live around humans.

Additionally, Warnke found that cellular devices had a detrimental impact on bees.[36] In this study, beehives exposed for just ten minutes to 900MHz waves fell victim to colony collapse disorder.[37] Colony collapse disorder is when many of the bees living in the hive abandon the hive leaving the queen, the eggs, and a few worker bees. The worker bees exposed to this radiation also had worsened navigational skills, causing them to stop returning to their original hive after about ten days.[38] Bees are an incredibly important part of the earth's ecosystem. Around one-third of the food produced today is dependent on bees for pollination, making bees are a vital part of the

agricultural system.[39] Bees not only provide pollination for the plant-based food we eat, but they are also important to maintaining the food livestock eats. Without bees, a vast majority of the food eaten today would be lost or at the very least highly limited. Climate change has already caused a large decline in the world's bee population.

The impact that the cell towers have on birds and bees is important to understand, because all ecosystems of the earth are interconnected. If one component of an ecosystem is disrupted the whole system will be affected. The disturbances of birds with the cell towers of today would only increase, because with 5G a larger number of small cell radio-tower-like devices would be necessary to ensure high quality connection for users. Having a larger number of high concentrations of these millimeter waves in the form of small cells would cause a wider exposure to bees and birds, and possibly other species that are equally important to our environment.

The Importance of a Proactive Approach

As innovation continues, it is important that big mobile companies around the world consider the impact 5G will have on the environment before pushing to have it widely implemented. The companies pushing for the expansion of 5G may stand to make short term economic gains. While the new network will undoubtedly benefit consumers greatly, looking at 5G's long-term environmental impacts is also very important so that the risks are clearly understood and articulated. The technology needed to power the new 5G network will inevitably change how mobile devices are used as well as their capabilities. This technological advancement will also change the way technology and the environment interact. The change from using radio waves to using millimeter waves and the new use of small cells in 5G will allow more devices to be used and manufactured, more energy to be used, and have detrimental consequences for important ecosystems.

While it is unrealistic to call for 5G to not become the new network norm, companies, governments, and consumers should be proactive and understand the impact that this new technology will have on the environment. 5G developers should carry out Environmental Impact Assessments that fully estimate the impact that the new technology will have on the environment before rushing to widely implement it. Environmental Impact Assessments are intended to assess the impact new technologies have on the environment, while also maximizing potential benefits to the environment. [40] This process mitigates, prevents, and identifies environmental harm, which is imperative to ensuring that the environment is sustainable and sound in the future.

Additionally, the method of Life Cycle Assessments (LCA) of devices would also be extremely beneficial for understanding the impact that 5G will inevitably have on the environment. An LCA can be used to assess the impact that devices have on carbon emissions throughout their life span, from the manufacturing of the device to the energy required to power the device and ultimately the waste created when the device is discarded into a landfill or other disposal system.[41] By having full awareness of the impact new technology will have on the environment ways to combat the negative impacts can be developed and implemented effectively.

Endnotes

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5G Wireless Communication and Health Effects—A Pragmatic Review Based on Available Studies Regarding 6 to 100 GHz - PMC

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5G Wireless Communication and Health Effects —A Pragmatic Review Based on Available Studies Regarding 6 to 100 GHz

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Abstract

The introduction of the fifth generation (5G) of wireless communication will increase the number of high-frequency-powered base stations and other devices. The question is if such higher frequencies (in this review, 6–100 GHz, millimeter waves, MMW) can have a health impact. This review analyzed 94 relevant publications performing in vivo or in vitro investigations. Each study was characterized for: study type (in vivo, in vitro), biological material (species, cell type, etc.), biological endpoint, exposure (frequency, exposure duration, power density), results, and certain quality criteria. Eighty percent of the in vivo studies showed responses to exposure, while 58% of the in vitro studies demonstrated effects. The responses affected all biological endpoints studied. There was no consistent relationship between power density, exposure duration, or frequency, and exposure effects. 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. Our quality analysis shows that for future studies to be useful for safety assessment, design and implementation need to be significantly improved.

Keywords: radiofrequency electromagnetic fields, MMW, in vivo, in vitro

1. Introduction

Recent decades have experienced an unparalleled development of technologies that are categorized as information and communication technologies (ICT), which include wireless communication used for mobile telephony (MP) and e.g., Wi-Fi by using electromagnetic fields (EMF). The first generation of handheld mobile phones were available for individual, private, customers in a few countries in the late 1980's. Subsequently, the second (2G), third (3G), and fourth (4G, LTE) generations increased their penetration rates in the society in a dramatic way, so that today there are more devices than inhabitants of the Earth. In addition, Wi-Fi and other forms of wireless data transfer have become ubiquitous, and are globally available. At present we are starting to introduce the next generation, 5G, of mobile networks. Importantly, 5G is not a new technology, but an evolution of already existing G1 to G4 technologies.

With the upcoming deployment of 5G mobile networks, significantly faster mobile broadband speeds and increasingly extensive mobile data usage will be ensured. This is made possible by the use of additional higher frequency bands. 5G is intended to be the intersection of communications, from virtual reality to autonomous vehicles to the industrial Internet and smart cities. In addition, 5G is considered the base technology for the Internet of Things (IoT), where machines communicate with machines (M2M communication). At the same time, a change in the exposure to electromagnetic fields (EMF) of humans and the environment is expected (see, for example [1,2]).

The 5G networks will work with within several different frequency bands ([/pmc/articles/PMC6765906/table/ijerph-16-03406-t001/]Table 1), of which the lower frequencies are being proposed for the first phase of the 5G networks. Several of these frequencies (principally below 1 GHz; Ultra-high frequencies, UHF) have actually been or are presently used for earlier mobile communication generations. Furthermore, much higher radio frequencies (RF) are also planned to be used at later stages of technology evolutions. The new bands are well above the UHF ranges, having wavelengths in the centimeter (3–30 GHz) or the millimeter ranges (30–300 GHz; millimeter waves, MMW). These latter bands have traditionally been used for radars and microwave links.

Table 1

Subdivision of the 5G frequency spectrum.

Frequency Range	Use	Comments
<1 GHz	Net coverage, IoT	Already partly used for earlier MP generations, longer range coverage, less costly infrastructure
1–6 GHz	Net coverage, IoT, capacity for data transfer	More spectrum available, shorter range and reduced performance compared to higher frequencies
>6 GHz	Capacity for very high data transfer	Short range, allows high speed data transfer and short latency times

The introduction of wireless communication devices that operate in the high frequency parts of the electromagnetic spectrum has attracted considerable amounts of studies that focus on health concerns. These studies encompass studies on humans (epidemiology as well as experimental studies), on animals, and on in vitro systems. Summaries and conclusions from such studies are regularly published by both national and international committees containing relevant experts (see e.g., [3,4,5]). The conclusions from these agencies and committees are that low level RF exposure does not cause symptoms ("Idiopathic Environmental Intolerance attributed to Electromagnetic Fields", IEI-EMF), but that a "nocebo" effect (expectation of a negative outcome) can be at hand. Some studies suggest that RF exposure can cause cancer, and thus the International Agency for Research on Cancer classified RF EMF as a "possibly carcinogenic to humans" (Group 2B) [3]. In a recent recommendation of a periodically working Advisory Group for IARC "to ensure that the Monographs evaluations reflect the current state of scientific evidence relevant to carcinogenicity" the group recommended radiofrequency exposure (among others) for re-evaluation "with high priority" [6]. There is further no scientific support for that effects on other health parameters occur at exposure levels that are below exposure guideline levels, even though some research groups have published non-carcinogen related findings after RF exposure at such levels (see [4,5]). Environmental aspects of this technological development are much less investigated.

Frequencies in the MMW range are used in applications such as radar, and for some medical uses. Occupational exposure to radars have been investigated in some epidemiological studies, and the overall conclusion is that this exposure does not constitute a health hazard for the exposed personnel [7]. This is due to that exposures for all practical purposes are below the guideline levels and thus not causing tissue heating. However, further studies are considered necessary concerning the possible cancer risk in exposed workers. Medical use of MMW has been recently reviewed [8,9] suggesting a possibility for certain therapeutic applications, although the action mechanisms are unclear.

The 5G networks and the associated IoT will greatly increase the number of wireless devices compared to the present situation, necessitating a high density of infrastructure. Thus, a much higher mobile data volume per geographic area is to be created. Consequently, it is necessary to build a higher network density because the higher frequencies have shorter ranges. The question that arises, is whether using the higher frequencies can cause health effects?

Exposure limits for both the general public and occupational exposure are available and recommended by the WHO in most countries, based on recommendations from ICNIRP [10] or IEEE [11] guidelines. These limits, which have considerable safety factors included, are set so that exposure will not cause thermal damage to the biological material (thermal effects). Thus, for 10 GHz to 300 GHz, 10 W/m^2 is recommended as the basic restriction (no thermal effects), with reference values for 400 MHz to 2 GHz ($2\text{--}10 \text{ W/m}^2$) and $>2 \text{ GHz}$ (10 W/m^2). It should be pointed out that the present ICNIRP guidelines [10] are currently being revised, and new versions are to be expected in the near future. In addition, ICNIRP proposes two categories of recommendations: (1) the basic restriction values based on proven biological effects from the exposure and (2) the reference levels given for the purpose of comparison with physical value measurements. ICNIRP guidelines present

no reference values above 10 GHz, only considering the basic restriction values. This is due to that only surface heating occurs since the penetration depth is so small at these frequencies. Therefore any calculations of the Specific Absorption Rate (SAR) values, that take larger volumes into consideration, are not reasonable to perform.

The SAR is the measure of the absorption of electromagnetic fields in a material and is expressed as power per mass/volume (W/kg), where the penetration depth of the electromagnetic fields depends on the wavelength of the radiation and the type of matter. The penetration depth of MMW is very shallow, hence the exposed surface area and not the volume is considered. The appropriate exposure metric for MMW is therefore the power density, power per area (W/m²).

It is of course too early to forecast the actual exposures to 5G networks. However, the antennas planned for 5G will have narrow antenna beams with direct alignment [12] to the receiving device. This could possibly significantly reduce environmental exposure compared to the present exposure situation. However, it is also argued that the addition of a very high number of 5G network components will increase the total EMF exposure in the environment, and that higher exposures to the higher frequencies can lead to adverse health effects.

Therefore, the question arises, what do we know so far about the effects on biological structures and on health due to exposure to the higher frequency bands (in this review we consider 6–100 GHz, since lower frequencies have been extensively investigated due to their use in already existing wireless communication networks)? Do so-called “non-thermal” effects (effects that occur below the thermal effect threshold) occur, that can lead to health effects? Is there relevant health-oriented research using the 5G technology relevant frequencies? Is there relevant research that can make a significant contribution to improving the risk assessment of exposure to the general population? Answers to these questions are necessary for a rapid and safe implementation of a technology with great potential.

2. Materials and Methods

This review takes into account scientific studies that used frequencies from 6 GHz to 100 GHz as the source of exposure. The review is based on available data in the field of public literature, papers written in English until the end of 2018 (PubMed database: www.ncbi.nlm.nih.gov/pubmed), EMF-Portal (www.emf-portal.org), and other relevant literature such as documents from ICNIRP, SCENIHR, WHO, IARC, IEEE, etc.). In addition, more refined research was conducted when necessary from sources that were not included in the above-mentioned databases (relevant abstracts from conferences, abstract books, and archives of journals). The resulting studies were examined for technical and scientific data and presented in the supplementary Table S1.

As a pragmatic approach, we interpreted the results as a “response” when the authors themselves reported the result as an “effect/response” based on a statistical analysis and the *p*-value < 0.05.

Next we defined necessary criteria for study quality, both from a biomedical and physical point of view (see [13]). The results of the studies were (if possible) analysed for correlations with study

quality according to the correlation approach done by Simkó et al. [14]. The studies were analysed with reference to a minimum of criteria in terms of experimental design and implementation. The following criteria were considered: were the experiments performed in the presence of an appropriate sham/exposure control, temperature control, positive control, were the samples blinded, and was a comprehensive dosimetry presented.

The study is divided into a descriptive part, which covers the description of all selected studies, their exposure conditions, frequency ranges (6 GHz to 100 GHz), dose levels, etc., as well as the biological results, presented in a Master-Table (Table S1). Review articles were not considered. The outcomes of the studies were furthermore analyzed and discussed according to frequency domains, and power density and exposure duration. If appropriate, we include an evidence-based interpretative part regarding risk from exposures according to the criteria of SCHEER [15].

3. Results

In the following, health-related published scientific papers dealing with frequencies from 6 GHz to 100 GHz (using the term MMW for all the frequencies) are described in detail. It should be noted that there are no epidemiological studies dealing with wireless communication for this frequency range, thus, this review will cover studies performed in vivo and in vitro.

Thermal biological effects of radiofrequency electromagnetic fields occur when the SAR values exceed a certain limit, namely 4 W/kg (general population exposure limit: SAR 0.08 W/kg), which causes a tissue heating of 1 °C. However, in the literature, biological effects below 4 W/kg SAR values have been described. Since such effects are considered to be not due to warming, they are termed non-thermal effects. In the present review, in some individual studies, the authors interpreted thermal effects as "no effect". Those ones and studies without response/effect of MMW exposure were considered as "no response/effect" in our present analysis.

3.1. Grouping of Selected Parameters

For analysis, 94 publications were identified and selected from the accessible databases (in vivo and in vitro)

[16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79,80,81,82,83,84,85,86,87,88,89,90,91,92,93,94,95,96,97,98,99,100,101,102,103,104,105,106,107,108,109]. It should be noted that the total number of individual examinations is larger than the number of publications, since some authors investigated several physical and/or biological conditions in the same publication.

Various biological endpoints have been identified, which are referred to as "response" or effects when appropriate. Since the list of these endpoints is relatively long, we have not mentioned them in detail, but summarized them in groups: Physiological, neurological, histological changes, or in in

vitro studies gene or protein expression, cytotoxic effects, genotoxic changes, and also temperature-related reactions.

For a detailed analysis, a "Master-table" (Table S1) was prepared in which all parameters considered in the studies were included. The table contains the following information: frequency, in vivo or in vitro study (the latter distinguishes between primary cells and cell lines), power density, exposure duration, biological endpoints, and response. Some studies lack information on individual parameters. For example, a publication had to be excluded completely because there was no information about the frequency. In nine studies the power density data were absent and in seven studies the calculated SAR values were provided instead of the power density. In ten studies, the exposure time was not given.

The 45 in vivo studies were mainly conducted on mammals (mouse, rat, rabbit) and a few on humans. In some studies, bacteria, fungi, and other living material were also used for the experiments. 80% of all in vivo studies showed exposure-related reactions.

Primary cells (n = 24) or cell lines (n = 29) were used in the 53 in vitro studies, with approximately 70% of the primary cell studies and 40% of the cell line investigations showing exposure-related responses ([/pmc/articles/PMC6765906/table/ijerph-16-03406-t002/]Table 2).

Table 2

Overview of the total number of publications examinations.

All Publications (94)	No Response	Response	All
In vivo	10	35	45
In vitro	22	31	
Primary cells	6	18	53
Cell lines	16	13	

All identified studies were analyzed as a function of frequency. For this purpose, frequency domains (groups) have been created ([/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/]Figure 1) to analyze and illustrate the results. The frequency groups from 30 to 60 GHz were grouped in ten-GHz increments (up to 30, 30.1–40, 40.1–50, 50.1–60 GHz). The frequency range 60–65 GHz was extra analyzed as in this group a larger number of publications was identified (in comparison to the other groups). Due to the low number of publications above 65.0 GHz, data was merged into the groups of "65.1–90" and "above 90 GHz". As shown in [/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/]Figure 1, the majority of studies show a frequency-independent response after MMW exposure.

[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/]

[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/]Figure 1

The number of publications as a function of frequency domains. The black line represents the total number of publications, and bars represent the *in vivo* (dark blue) and *in vitro* (light blue) studies with biological responses.

3.1.1. Frequency Ranges

All data regarding the individual papers are found in Table S1.

Up to 30 GHz

The first group “up to 30 GHz” was introduced since some of the 5G frequencies fall within this frequency range. Unfortunately, there are only two publications in this group, both showing responses to the MMW exposure. A study that was conducted on bacteria and fungi showed an increase in cell growth [58]. The other *in vitro* study was performed on fibroblasts (25 GHz, 0.80 mW/cm², 20 min), with genotoxic effects observed at high SAR levels (20 W/kg) [24]. A graphical presentation of the outcomes is presented in [\[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/\]](#)Figure 1 for this and all other frequency domains.

Frequency Group 30.1–40 GHz

As shown in [\[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/\]](#)Figure 1, responses were detected in approximately 95% of the 19 studies. In all *in vivo* studies responses were described after exposure [25,27,36,37,55,56,78,79,87,91,103,104]. Endpoints ranged from recorded footpad edema, which is a frequent endpoint for the measurement of inflammatory responses, to morphological changes, changes in skin temperature, blood pressure, heart rate, body temperature, neuronal electrical activity, and EEG analyses. Protein expression studies, oxidative stress marker measurements, histological investigations, and induction of cell death (apoptosis) were performed. Only one study used lower power densities (0.01 mW/cm², 0.1 mW/cm²; SAR: 0.15, 1.5 W/kg; 20 min, 40 min) to study inflammatory responses [27]. The authors determined the frequency-dependent anti-inflammatory effect as a function of power density and exposure duration and did not rule out temperature-related effects. The power densities of the other *in vivo* studies were extremely high (10, 75, 500–5000 mW/cm²), so the induced effects were likely temperature dependent.

Eight *in vitro* studies were performed [18,20,47,91,97,99,101,102] of which seven reported responses. In one study [99], human blood cells (*ex vivo*) were exposed to MMW for 5, 15 and 30 min (32.9–39.6 GHz, 10 mW/cm²). The activation of the cells was examined in the presence or absence of bacteria. It was shown that in the presence of bacterial activation and after 15 min of exposure, the cells were activated to release free radicals. These results were similar to the heated samples (positive controls), so a temperature effect is plausible. The induction of differentiation of bone marrow cells in to neuronal phenotype cells was also demonstrated (36.11 GHz, 10 mW/cm², 3 × 10 min every 2 h for 24 h) [97]. In two studies, temperature-related reactions were described at the protein level [18,91]. When the cell cultures were cooled during exposure to prevent the induced temperature increase, no responses were detected.

In three publications, a research group described cell cycle changes, induction of cell death and activation of differentiation processes in primary cells (rat bone cells and mesenchymal stem cells) after exposure to 30–40 GHz (4 mW/cm², different exposure durations) [47,101,102]. Unfortunately, the minimum quality criteria were not fulfilled in any of the three studies, mainly because there were no temperature controls.

Frequency Group 40.1–50 GHz

In the 40.1–50 GHz frequency group, 26 studies were identified, 13 in vivo [16,17,26,48,49,51,53,65,69,74,80,84,98] and 13 in vitro [29,30,31,62,64,86,89,92,93,100,105,107] with nine studies showing responses. A large number of studies have tested cell biology endpoints such as cell proliferation, gene or protein expression, and changes in oxidative stress. In addition, immunological, neurological, morphological and genotoxic effects were investigated. The power densities used vary enormously, from 0.02 to 450 mW/cm², and one publication gave no information.

In healthy volunteers, a double-blind study was performed to investigate the effects of MMW on experimentally induced cold pain (42.25 GHz, <17.2 mW/cm², 30 min) [74]. The authors found no difference from the placebo effect. This study was a repeat of a previous study with volunteers and the results of the older study could not be confirmed. The other four in vivo studies with no detectable effects were investigating genotoxic effects or oxidative stress [17,48,49,98].

Five in vivo publications addressed the effects of MMW on the immune system of mice or rats, finding activation of the immune system at both the cellular and molecular levels (41.95 or 42.2 GHz, 19.5 μW/cm², 0, 1, 31.5 mW/cm², 20 min or intermittently over 3 days) [26,48,51,53,84].

MMW exposure of frog isolated nerve cells, (41.34 GHz, 0.02, 0.1, 0.5, 2.6 mW/cm², 10–23 min) lead to a reduction of the action potential frequency. Interestingly, the effects at higher power density (2.6 mW/cm²) were similar to conventional heating [49].

One study detected an increase in the motility of human spermatozoa after 15 min of exposure (42.25 GHz, 0.03 mW/cm²) [100]. Additional in vitro tests have identified the formation of free radicals, the activation of calcium-dependent potassium ion channels (around 42 GHz, 100, 150, 240 μW/cm², 20–40 min) as well as changes at the cell membrane in exposed cells [29,30,100].

No responses on cell biological endpoints (cell cycle changes, cell death, heat shock proteins) were detected in four additional in vitro studies.

Frequency Group 50.1–60 GHz

We identified 16 studies in the frequency group 50.1–60 GHz (six in vivo, ten in vitro) and 60% of the studies showed responses to MMW exposures [21,23,35,38,43,46,59,61,72,77,81,83,85,94,109].

In five of the in vivo studies very different responses were shown. In a study on healthy volunteers, the authors wanted to find out whether the human skin at a so-called acupuncture point has

different dielectric properties during exposure to MMW. They found that these properties change during exposure to 50–61 GHz from the surrounding skin [23].

A pilot study on mice (60 GHz, 0.5 mW/cm², lifelong exposure for 30 min/5 days a week) showed that MMW exposure affects cancer-induced cells and increases in motor activity of healthy mice [61].

In rats, the influence of 54 GHz, 150 mW/cm², on an area of approximately 2 cm² on the head was examined [81]. This transcranial electromagnetic brain stimulation induced pain prevention and prevented the conditioned avoidance response to a pain stimulus in 50% of the animals. However, no changes were detected when serotonin inhibitors were previously administered. Therefore, the authors concluded that transcranial electromagnetic brain stimulation promotes the synthesis of serotonin, a transmitter that changes the animals' pain threshold.

The effects of MMW were also tested (60 GHz, 475 mW/cm², 1.898 mW/cm², 6, 30 min) on rabbit eyes, describing acute thermal injuries of various types [38]. The authors also pointed out that the higher temperature just below the eye surface could induce injury.

Neurological investigations were performed on leeches (60 GHz, 1 min, 1, 2, 4 mW/cm²) [77] and electrophysiological studies were performed on frog oocytes (60 GHz, up to 5 min) [85]. In both experimental systems effects were described, which were induced by the temperature rise.

Cell biological and morphological changes after exposure to 0.7–1.0 µW/cm² (intermittent) were reported in three in vitro studies [72,83,94], with two publications providing no information regarding power density or exposure duration. At the level of protein analysis and total genome analysis no changes were identified in four in vitro studies [35,46,59,109].

Frequency Group 60.1–65 GHz

The number of studies in the 60.1–65 GHz frequency group is 27. Of these, twelve reported effects from exposure to MMW, and no responses were found in 15 studies.

The in vivo studies investigated different topics [23,27,44,52,67,68,70,71,73,75,76]. Thus, two studies examined the effects on tumor development in mice injected with tumor cells [52,70]. In one of the studies it was reported that exposure to 61.22 GHz, 13.3 mW/cm², inhibited the growth of melanoma cells (exposure 15 days after tumor cell injection, 15 min/day) [70].

Other publications from one research group investigated the potential of MMW for pain relief and the associated biological mechanisms of action [67,71,73,75,76]. Several of the studies were performed on mice skin exposed to 61.22 GHz for 15 min. The most commonly used power density was 15 mW/cm². Another study addressed the dose issue with no effect below 1.5 mW/cm². The authors' conclusion is that MMW can lower the hypoalgesia threshold, which is likely mediated by the release of opioids.

The effects of 61.22 GHz exposure of mice were examined also with respect to the immune system [52]. The animals were exposed on three consecutive days for 30 min per day. The exposure caused

peak SAR values of 885 W/kg on the nose of the animals where the exposure took place. The power density was 31 mW/cm² and the measured temperature rise reached 1 °C. It was found that MMW modulates the effects of the cancer drug cyclophosphamide. In particular, the T-cell system of the immune system was activated and various other immune system relevant parameters affected.

The similar exposure condition was used in a study on gastrointestinal function, however no effects were identified [68].

A single exposure for eight hours (61 GHz, 10 mW/cm²), or five times four hours, did not cause eye damage to rabbits and rhesus monkeys [44]. It should be emphasized that several of the mentioned studies come from the same laboratory, and all criteria for the study quality are met. However, the authors were able to replicate their own findings on pain relief whereas other laboratories have not replicated this work. In the in vitro studies, various biological endpoints were examined [28,32,33,34,42,45,50,59,60,66,83,88,94,95,108].

In one study, neurons of snails (*Lymnea*) were exposed at 60.22–62.22 GHz and no non-thermal responses on the ion currents were identified [28].

In a series of investigations with nerve cell-relevant cell lines, the dopamine transmission properties, stress, pain and membrane protein expression were investigated (60.4 GHz, 10 mW/cm², 24 h) and no responses were detected [32,33,34,59,60,108].

The same exposure setup has also been used in studies examining different stress response related genes (0.14–20 mW/cm²) [59]. No effects were found at the gene expression level. Interestingly, the overall genome impact was influenced when the exposure (60.4 GHz, 20 mW/cm², 3 h) of the primary human keratinocytes was combined with 2-deoxyglucose, a glucose-6-phosphatase inhibitor. This co-exposure caused a change in the amount of six different transcription factors, the effect differing from the effect of 2-deoxyglucose alone and 60.4 GHz alone (both factors alone induced no changes).

Other studies also examined human keratinocytes and astrocytoma glial cells after exposure to 60 GHz (0.54, 1 and 5.4 mW/cm²) [60,108]. Various parameters such as cell survival, intracellular protein homeostasis, and stress-sensitive gene expression were investigated. Also, in these studies, no effects were observed. In contrast, in one publication, the elevation of an inflammatory marker (IL1-β) was observed in human keratinocytes after exposure (61.2 GHz, 29 mW/cm², 15, 30 min), while other inflammatory markers (chemotaxis, adhesion and proliferation) have remained unchanged [95].

Another type of study was performed on rat brain cortical slices [66]. The brain slices were exposed to a field of 60.125 GHz (1 μW/cm²) for 1 min, and then specific electrophysiological parameters were measured. In many slices, transient responses on membrane characteristics and action potential amplitude and duration were observed. The exposure caused a temperature rise of the medium (of 3 °C) in which the sections were stored. Interestingly, a chronically induced Ca²⁺ blockade did not affect the MMW response.

Frequency Group 65.1–90 GHz

The studies in the frequency group of 65.1 to 90 GHz were performed both in vivo and in vitro in a total of 14 articles (four in vivo and 11 in vitro investigations). The studies vary widely, based on different hypotheses, biological endpoints, power densities, and exposure durations. In addition, some studies have used biological materials to identify physical properties such as dielectric properties and skin reflection coefficient. The latter studies are discussed in Section 4.2.

Four in vivo studies reported responses after MMW exposure. One study examined the dose of eye damage (especially damage to the corneal epithelium) [40]. The dose was calculated as DD_{50} (based on the results for which the probability of eye damage was 50%). The experiments were carried out on rats with an exposure of 75 GHz, the DD_{50} value being 143 mW/cm^2 .

Other in vivo studies were performed on rats and mice as well as on insects [27,42,57]. The study on mice used different frequencies of 37.5 to 70 GHz, with power densities of 0.01 and 0.3 mW/cm^2 for 20 to 40 min. A single whole-body exposure of the animals reduced both the footpad edema and local hyperthermia on average by 20% at the frequencies of 42.2, 51.8, and 65 GHz. Other frequencies had no influence.

The study on insects (*Chironomidae*) focused on DNA effects of giant chromosomes of the salivary glands of the animals with different frequencies (64.1–69.1, 67.2, 68.2 GHz) [42]. All frequencies, using power densities $<6 \text{ mW/cm}^2$, caused a reduction in the size of a particular area of the chromosome. This in turn led to the expression of certain secretory proteins of the salivary gland.

Different aspects were studied in the in vitro studies [18,28,39,50,64,72,83,89,94,106], where nerve cell function was investigated in three studies. Two studies used nerve cells from the snail *Lymnea* that were exposed at 75 GHz for a few minutes at very high SAR levels (up to 4200 W/kg , power density was not reported) [28,39]. The authors observed thermal effects on the ion currents and the firing rate of the action potentials. Another study also described thermal effects on transmembrane currents and ionic conductivity of the cell membrane. Again, the exposure was at very high SAR levels (2000 W/kg), and the authors emphasized the temperature dependence of the reaction.

Broadband frequencies (52–78 GHz) have been used in several publications, mainly investigating the effects on cell growth and cell morphology as well as the ultrastructure of different cell lines [50,72,83,94]. The values for the power densities were not given consistently but appear to have been very low (not higher than $1 \mu\text{W/cm}^2$). The results indicated the inhibition of cell growth, accompanied by changes in cell morphology.

Another group of studies used hamster fibroblasts, BHK cells, and exposed the cells at 65 to 75 GHz, with the power density reaching 450 mW/cm^2 [18,64,89]. The authors noted the inhibition of protein synthesis and cell proliferation as well as cell death at higher power densities. In a study using human dermal fibroblasts and human glioblastoma cells, no effects at the protein level (proliferation or cytotoxicity markers) were detected (70 GHz and higher, in 1 GHz increments; 3, 70 or 94 h) [106].

Power densities varied across frequencies, ranging from 1.27 $\mu\text{W}/\text{cm}^2$ in the lower frequency range to 0.38 $\mu\text{W}/\text{cm}^2$ at higher frequencies.

The in vitro studies in this group are similar to the in vivo studies in their diversity. The majority of studies in which responses were reported are thermal-effects due to MMW exposure. In three studies, responses at low power densities were described, but all results were from the same laboratory, and were not replicated by others. Moreover, the quality of these studies is questionable, as the quality criteria were not met.

Frequency Group 90.1–100 GHz

Eight out of eleven studies in the 90.1–100 GHz frequency group are in vitro studies [22,41,57,82,90,96,106]. The three in vivo investigations addressed a variety of issues including acute effects on muscle contraction, skin-reflection properties (which are more of a dose-related than health-related issue), and skin cancer [19,54,57]. The rat skin cancer study (one to two weekly, short-term exposures at 94 GHz, 1 W/kg; DMBA-initiated animals) did not show any positive outcome [54]. Another study examined the muscle contraction of mice and described some responses [19]. Again, 94 GHz was used, but power density or SAR values were not reported.

Seven of the eight in vitro studies showed responses after MMW exposure. In some studies, primary neurons were used to study the cytoskeleton (94 GHz, 31 mW/cm^2) [82] or specific electrophysiological parameters (90–160 GHz) [22]. In the latter study it was found that the observed responses were more likely due to interactions with the cell culture medium than with the cells, although the mechanisms of action were not clear. Other studies identified responses on the DNA integrity (100 GHz and higher) [41] or described changes in intracellular signaling pathways (94 GHz, 90–160 GHz) using different cell types [57,96]. The exposure time ranged from minutes to 24 h for partially unknown exposure values. In one study no cytotoxic influence at power density levels of a few $\mu\text{W}/\text{cm}^2$ was detected in either normal or in tumor cells.

3.1.2. Power Densities

All identified studies were analyzed as a function of the used power densities. The studies were grouped depending on the power density as follows: below 1; 1.1–10; 10.1 to 50; 50.1–100, and 100.1 mW/cm^2 or higher. Studies that do not provide information on power density or SAR values are not displayed in these groups. As shown in [\[pmc/articles/PMC6765906/figure/ijerph-16-03406-f002/\]](#)Figure 2, the vast majority of studies show responses regardless of the power density used.

[\[pmc/articles/PMC6765906/figure/ijerph-16-03406-f002/\]](#)

[\[pmc/articles/PMC6765906/figure/ijerph-16-03406-f002/\]](#)Figure 2

The number of publications as a function of power density. The black line represent the total number of publications, and bars represent the in vivo (dark blue) and in vitro (light blue) studies with biological responses.

3.1.3. Exposure Duration

Exposure duration of the studies was also grouped for data analysis ([/pmc/articles/PMC6765906/figure/ijerph-16-03406-f003/]Figure 3). The time groups were selected as seconds to 10 min; 10–30 min; 30–60 min; over 60 min—days and alternately/intermittently. The groups were selected so that the used exposure times and the number of studies are meaningfully summarized. Here, too, it becomes clear that the majority of all studies show responses regardless of the exposure time. Interestingly, longer exposure times (over 60 min—days) seemingly lead to fewer reactions than in the other groups.

[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f003/]
 [/pmc/articles/PMC6765906/figure/ijerph-16-03406-f003/]Figure 3

The number of publications as a function of exposure duration. The black line represent the total number of publications, and bars represent the in vivo (dark blue) and in vitro (light blue) studies with biological responses.

3.2. Studies without Responses

[/pmc/articles/PMC6765906/table/ijerph-16-03406-t003/]Table 3 shows the number of studies in which no responses were detected after or during MMW exposure. As “no response” also such investigations were referred to, which were considered by the authors themselves as such. This means that in some cases the observed effects were described as temperature-related and not as a non-thermal MMW effect.

Table 3

Studies without responses.

Frequency (GHz)	No Response	
	In Vivo	In Vitro
Up to 30	0	0
0.1–40	0	2
40.1–50	6	4
50.1–60	1	5
60.1–65	2	10
65.1–90	0	6
90.1–100	1	1

Few in vivo studies have shown no response at all. Noticeable is the frequency group 40.1–50 GHz, in which 6 studies were identified. These studies investigated immunosuppression, genotoxic effects,

changes in pain sensitivity, and changes in enzyme activity. One study was carried out on bacteria and fungi.

There are a variety of in vitro studies in which no responses were detected. Interestingly, studies on protein or gene expression levels often failed to detect any changes after MMW exposure. This could be due to the fact that in in vitro studies the possibility of non-thermal effects were specifically investigated, where cooling was used to counteract the temperature increase.

3.3. Quality Analysis

We analyzed the quality of the selected studies according to specific criteria [14]. The studies were categorized by the presence of sham/control, dosimetry, positive control, temperature control, and whether the study was blinded. The presence of these five criteria while performing an MMW study is the minimum requirement for qualifying as a study with sufficient technical quality.

Of the 45 in vivo studies, 78% (35) demonstrated biological responses after exposure to MMW. Of all studies, 73% were performed with sham/controls, 76% employed appropriate dosimetry, 44% used positive control, and 67% were done under temperature control conditions ([/pmc/articles/PMC6765906/figure/ijerph-16-03406-f004/]Figure 4). Unfortunately, only 16% of the studies were performed according to protocols that ensured blinding and only three publications were identified that met all five criteria [26,51,53]. If the blinding criterion was excluded, 13 studies could be identified that met the remaining four criteria. Considering three criteria only, namely sham, dosimetry, and temperature control, 40% (20 papers) were identified. Thus, the quality of the in vivo studies is unsatisfactory.

[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f004/]

[/pmc/articles/PMC6765906/figure/ijerph-16-03406-f004/]Figure 4

The quality of all publications: The number of in vivo (top) and in vitro (bottom) experiments (blue: no reaction, red: reaction) using the listed quality features (y-axis). The spider web shows the percentage of the quality characteristics in all examinations.

Out of the 53 in vitro studies, 31 showed biological responses. Only in 13 studies (42%) were three of the five quality criteria satisfied, namely the presence of sham/control, dosimetry, and temperature control ([/pmc/articles/PMC6765906/figure/ijerph-16-03406-f004/]Figure 4). Positive controls were used in 47% and only one study was performed with blinded protocol (2%).

These results show that the number of examinations and the quality criteria are insufficient for a statistical analysis. It should be stressed that this quality analysis covers all publications dealing with the responses/effects of exposure to 6 to 100 GHz MMW, irrespective of the endpoints tested. To perform a correlation analysis, a larger number of comparable studies (e.g., identical endpoints in a frequency group) would be required.

4. Discussion

The first relevant observation during the analysis of the studies is that in most publications the aim of the investigations has been to determine the effects of MMW exposure for medical purposes. This means that the exposure devices used primarily come from medical applications (therapy or diagnostics). Very few publications dealt with health-related issues after MMW exposure in general, or with the specific topic of 5G. Therefore, the 94 publications are very heterogeneous.

We divided the frequency bands into seven ranges and placed the studies in the relevant groups. All available information on physical and experimental parameters was collected, but the exact number of experiments in each study was not taken into account. (One publication can contain more than one experiment.) Therefore, it is the provided numbers of studies/publications that constitute the data set, not the exact numbers of experiments performed, which is significantly higher.

This report does not provide a statistical analysis of the correlation between the exposure conditions and the results, which was our original ambition. In the correlation study according to Simkó et al. [14] a frequency group was selected, with only one group of biological endpoints considered. About one hundred, exclusively in vitro, studies were identified and broken down into individual experiments in that paper. In this way, the number of experiments was sufficient to perform a correlation analysis. In the present review, the spread of biological endpoints in the individual frequency groups and the models used (in vivo and in vitro) is large and the number of studies is very low. Therefore, it was not possible to group the studies by specific endpoints and perform a statistical analysis.

Interestingly, more than half of the studies (53 publications) were conducted in the frequency bands 40.1–50 and 60.1–65 GHz (with different models and endpoints). One possible reason for this is that medical use of MMW has a long tradition in Eastern Europe. These applications use specific frequencies that fall in these two frequency groups. The studies were conducted with the aim of testing specific effects with medical relevance. In these two frequency groups, the “with responses” percentage was generally lower than in the other frequency bands (see [pmc/articles/PMC6765906/figure/ijerph-16-03406-f001/]Figure 1), where a majority of studies showed responses to exposure.

With regard to the power densities used, about half of the studies were carried out in the range up to 10 mW/cm² ([pmc/articles/PMC6765906/figure/ijerph-16-03406-f002/]Figure 2). This value is ten times higher than the current ICNIRP exposure guideline [10] for the general population. Based on available data, there is no indication that higher power densities cause more frequent responses, since the percentage of responses in all groups is already at 70% ([pmc/articles/PMC6765906/figure/ijerph-16-03406-f002/]Figure 2). One exception from this high response rate is the group 50.1–100 mW/cm², where the proportion of studies with reactions is slightly lower (54%). However, the total number of examinations (11) is relatively small in this group.

The results of some of the studies may suggest that exposure to power densities at or below the guideline recommendations induce biological effects. There are, however, some arguments against it. One of these is the apparent heterogeneity of the study design and the outcomes studied. There

are very few (if any) independent replication studies that confirm the reported results. It is also noteworthy that there is no trend towards a classic dose-response pattern where stronger or more frequent effects would be caused by higher exposure levels. Since the studies with conditions promoting tissue warming show no greater effect than below the guideline values (1 mW/cm^2), this would either mean that the same interactions are present at all power densities tested, or that experimental artifacts unknown to the scientists are present.

The most important physical experimental parameter is the temperature during exposure, therefore, the temperature must be consistently controlled. The need for stringent temperature control is not an insignificant or trivial matter and has been neglected or at least undervalued in many studies. Although some authors report that they performed specific temperature measurements during the experiments, this does not necessarily mean that this represents the actual temperature in the biological material. Measurements can be made, for example, in the surrounding medium but not in the exposed tissue or in the cell. It also has to be considered that the "bulk" heating (from outside to inside with a certain time course) can differ from a heating that occurs at a rather limited point ("hot spot"). In addition, the intensity of a short burst can be lost if the measurements are based on average exposure times. Such errors and problems are possible factors that have contributed to the questionable interpretation of "non-thermal effects" in some studies.

Effects after MMW exposure were shown at all exposure times with no clear time dependency. The data presented shows one exception, namely in the group ">60 min to days", where fewer reactions were detected ([\[pmc/articles/PMC6765906/figure/ijerph-16-03406-f003/\]Figure 3](#)). It has to be taken into account that 27 examinations were carried out in this group, 23 of which were in vitro studies. In vitro experiments can be carried out under cooling, therefore the results can be different (see further below).

Two research groups together provide 30 of the 94 publications in the data set, and could thus possibly have a large impact on the analysis of the outcomes. One group presented at least 21 publications (42.25 and 61.82 GHz; 10 to 30 mW/cm^2 ; with different exposure durations), with a variety of in vivo and in vitro studies, which mostly reported responses to exposure. The other group mainly studied gene and protein expressions (60 GHz; 5.4 to 20 mW/cm^2 ; exposure durations from minutes to days) and found mainly no responses. Studies from both groups adhered well to the quality criteria in our analysis.

4.1. Temperature Controls in In Vitro Studies

In vivo studies that are performed within or directly on the living organism have shown both thermal and purportedly non-thermal effects after or during MMW exposure. In vitro studies are carried out on cells and most experimental parameters can be accurately set and observed. Cell cultures can thus be very carefully controlled, e.g., an induced temperature increase can be counter-cooled. Many in vitro studies considered in this review were performed using cooling of the cell culture vessels and the authors did not detect any non-thermal effects in these studies. In in vivo studies counter-cooling is not possible, thus it is very difficult to differentiate between thermal and non-thermal

reactions. Therefore, in vivo and in vitro studies regarding the induced effects cannot be directly compared. An accurate dosimetry could solve this problem.

4.2. Dosimetry

It is important to know what the exposure of the MMW will be due to the expected introduction of a large number of 5G wireless communication devices. Given the novelty of the technology, it is currently unlikely that a large number of relevant exposure assessment studies will be available. However, an example from a recent study [110] shows that a "typical" office environment with wireless communication transmitters (5.50 GHz) leads to power densities well below the exposure guideline limits. Thus, the maximum power density was measured at $0.89 \mu\text{W}/\text{cm}^2$.

Partly ($n = 25$) the experimental studies on biological and health effects of MMW exposure are at or below the ICNIRP exposure guidelines. The power densities were often chosen so that the exposure caused no or very moderate tissue warming ($<1 \text{ }^\circ\text{C}$), namely in the range of 1 to $10 \text{ mW}/\text{cm}^2$. Since the penetration into the tissue of these frequencies are on the order of millimeters and below, it is important to study biological effects directly or indirectly related to skin and eyes exposure. As mentioned previously, the number of available studies in the 6–100 GHz frequency range is relatively low, which is in contrast to the number of studies for lower radio frequencies. Similarly, the number of tissue dosimetry studies (especially for the skin) is very limited. However, such studies are very relevant because they show how certain exposure parameters can influence the energy input and thus the thermal behavior of the skin.

Currently, both the ICNIRP guidelines and the IEEE standards are being revised to replace the SAR values with power density above 6 GHz. However, it has already been recognized that there is a reactive near field close to the transmitter (around the antennas). Here, the energy is not radiated, but the energy envelopes the antennas. The question is whether these "reactive near fields" are important for the energy delivery to a human body near the transmitter? If this is not the case, it is sufficient to comply with the existing exposure limits based on free space power density measurements. On the other hand, a strong reactive near field would considerably complicate the exposure situation [111]. Therefore, for dosimetry modeling of distances (from the antenna) below the wavelength of the MMW (mm), temperature measurements should rather be performed in suitable phantoms rather than direct measurements of the power densities in the free space [111].

The question is how reliably the power density (in free space) can be extrapolated to possible temperature increases in human tissue? For example, Neufeld et al. [112] found that 10 GHz "bursts" (considered "safe" by ICNIRP and IEEE) can cause temperature increases of $>1 \text{ }^\circ\text{C}$ if the burst duration is long enough. It was also discussed whether the average values of the power densities for the safety assessment are the right ones. In addition, the temperature increase by the MMW also depends on the size of the area. Thus, the factors such as the amplitude of the burst, the "averaging area" and the "averaging time" for the dosimetry would have to be considered.

Foster et al. [113] reviewed and modelled data on MMW-induced temperature increases in human skin. The model takes into account the frequencies of 3–100 GHz and smaller skin areas with the diameter of 1–2 cm. Available data on exposures lasting more than a few minutes, as well as areas of skin larger than 2 cm in diameter, were limited and made modeling difficult, but consistent with existing data. This means that this model, after appropriate evaluation for dosimetry, could use smaller areas of the skin. The authors also commented on the exposure guidelines for frequencies from 3 to 300 GHz in a separate article [114]. Based on “thermal modeling,” the authors considered the current guidelines to be conservative in terms of protection against temperature increases in the tissue. They also pointed out that the averaging time and average area provisions require further refinement and that the effects of short high intensity bursts may not be protected by the guidelines.

Zhadobov et al. [115] addressed the problem of accurate temperature measurement in in vitro MMW studies. They found that the type of thermal probe (thermocouples are better than fiber optic probes) and the size of the probe (smaller probes are more accurate) are relevant. In addition, they were able to show that the initial temperature rise during exposure is rapid (within seconds until a plateau is reached) and that the cells absorb very small amounts of energy, since most of the energy is already absorbed in the cell culture medium. Nevertheless, the authors have calculated that the exposure of 58.4 GHz with 10 mW/cm² leads to SAR values of more than 100 W/kg in a cell monolayer. This value is a fraction of the SAR values of the fluid surrounding the cells.

Several studies focused on the distribution of power density and the change in skin temperature as a result of exposure to MMW in the 6 to 100 GHz frequency range. The studies are experimental and/or modeling studies using previously published data. Alekseev et al. [116,117] investigated the absorption of the skin of mice and humans at frequencies between 30 and 82 GHz (10 mW/cm²). They found that in both species absorption into both the epidermis and the dermis occurs with a concomitant loss of power density in the deeper regions. An extended study from the same group [118] on human forearm skin showed that both temperature increase and SAR values depend on frequency (in the interval of 25 to 75 GHz; 25, 73.3 and 128 mW/cm²).

Frequency dependence for temperature increases was also observed in a modeling study with human facial skin [119]. Pulsed MMWs were used (6–100 GHz, 100 mW/cm², 200–10,000 ms pulse length) and the skin temperatures were modeled as the function of both pulse length and frequency. Peak skin temperature increased as a function of frequency up to 20 GHz, while above 20 GHz it proved to be dependent on “absorption hotspots”. In deeper regions (>2 mm), the temperature increases were very low and highest around 10 GHz.

In addition, certain skin constituents have been shown to affect energy absorption. It has been shown that the presence of sweat glands [120,121] and also capillaries in the dermis can cause locally elevated SAR levels [122]. The latter study showed that SAR levels in vessels could be up to 30 times higher than in the surrounding skin, depending on the diameter of the vessels.

Both [23] and [123] have reported that the dielectric properties of different areas of the skin differ. The first study found that so-called acupuncture points in healthy volunteers show different dielectric properties when exposed to MMW (50–75 GHz, 14 mW/cm²), while the second study even found differences between the epidermis and dermis (0–110 GHz).

These studies suggest that both the frequency and the specific condition and composition of the skin are relevant for tissue dosimetry. However, too few and very different studies are available to give a conclusive picture on dosimetry of 5G-relevant MMW exposures.

4.3. ICNIRP and other Exposure Recommendations

The guidelines for exposure limits for radiofrequency electromagnetic fields from 3 to 300 GHz in many countries are based on the recommendations of the International Commission on Non-Ionizing Radiation Protection (ICNIRP) [10]. However, there are also other organizations dealing with limit values such as the Institute of Electrical and Electronics Engineers, IEEE [11] or the US Federal Communications Commission, FCC [124].

The guidelines contain basic exposure limits that are indicated as SAR or power density. The limits for a given frequency differ only slightly, if at all, between the different guidelines. However, an important difference between the guidelines concerns frequency, as the SAR basic restriction values change to power density. This frequency (range) is currently set by ICNIRP at 10 GHz, while IEEE and FCC see this between 3–6 GHz. The current revision of these guidelines aims to harmonize these frequencies.

The exposure limits specified in the guidelines should protect against warming of tissue above 1 °C. The reason is that the perceived dangers of MMW energy are associated with excessive heating, called thermal effects. However, it must be considered that the guidelines mean a temperature increase of 1 °C relative to the starting temperature, regardless of the starting temperature. Elevations in temperature may cause pain in the skin when moderately increased, whereas at temperatures of 43–44 °C it may even induce burns [124,125].

At present, only thermal effects due to high-frequency electromagnetic fields are recognized as effects. This means that effects have a thermal component even if it is obviously not due to tissue that has been damaged by excessive heating. On the other hand, it has been suggested that the MMW exposure may also cause non-thermal effects. So far, however, no recognized expert committee has supported such an assertion.

4.4. Knowledge Gaps and Research Recommendations

Exposure of humans can occur through 5G devices with frequencies above 6 GHz, and may be primarily on the skin and, to a lesser extent, on the eyes. This is due to the very low penetration depth of this MMW. Therefore, it is important to investigate whether there are any health-related effects on the skin and/or effects associated with the skin. These include acute skin damage from tissue heating (burns), but possibly also less acute effects (such as inflammation, tumor

development, etc.). Such effects could appear after prolonged and repeated heating of superficial structures (the skin). This would mean that thermal effects occur that are not due to acute but to chronic damage.

It may also be that local exposure causes energy deposition in the dermis of the skin, which may be so great as to affect nerve endings and peripheral blood vessels through warming mechanisms. Such scenarios were proposed by Ziskin [9] based on a series of studies by his group. These studies typically used exposures around 60 GHz at a power density of 10 mW/cm² on the skin in the sternum area to produce systemic effects. The aim was to treat certain diseases and complaints. The idea was that the treatment induces the release of the body's own opioids and additionally stimulates the peripheral nerves. The stimulation would depend on a local thermal effect, which, due to the frequencies, induces locally high SAR values, even at low power densities, thus warming the tissue.

Due to the contradictory information from various lines of evidence that cannot be scientifically explained, and given the large gaps in knowledge regarding the health impact of MMW in the 6–100 GHz frequency range at relevant power densities for 5G, research is needed at many levels. It is important to define exact frequency ranges and power densities for possible research projects. There is an urgent need for research in the areas of dosimetry, in vivo dose-response studies and the question of non-thermal effects. It is therefore recommended that the following knowledge gaps should be closed by appropriate research (the list of research recommendations is not prioritized):

- Exact dosimetry with consideration of the skin for relevant frequency ranges, including the consideration of short intense pulses (bursts)
- Studies on inflammatory reactions starting from the skin and the associated tissues
- In vivo studies on the influence of a possible tissue temperature increase (e.g., nude mouse or hairless mouse model)
- In vivo dose-response studies of heat development
- Use of in vitro models (3D models) of the skin for molecular and cellular endpoints
- Clarification of the question about non-thermal effects (in vitro)

There are also questions about the environmental impact, with potential consequences for human health. Since many MMW devices will be installed in the environment, the impact of MMW on insects, plants, bacteria, and fungi is relevant to investigate. Particularly relevant is the question of temperature increase in very small organisms, as the depth of penetration of the MMW could warm the whole organism.

An unrealistic scenario, however, is that MMW exposures at realistic power densities could cause systemic body warming in humans. Any local heat exposure would be dissipated by the body's

normal heat regulation system. This is mainly due to convection caused by blood flow adjacent to the superficial skin areas where the actual exposure takes place.

In summary, it should be noted that there are knowledge gaps with respect to local heat developments on small living surfaces, e.g., on the skin or on the eye, which can lead to specific health effects. In addition, the question of any possibility of non-thermal effects needs to be answered.

5. Conclusions

Since the ranges up to 30 GHz and over 90 GHz are sparingly represented, this review mainly covers studies done in the frequency range from 30.1 to 65 GHz.

In summary, the majority of studies with MMW exposures show biological responses. From this observation, however, no in-depth conclusions can be drawn regarding the biological and health effects of MMW exposures in the 6–100 GHz frequency range. The studies are very different and the total number of studies is surprisingly low. The reactions occur both in vivo and in vitro and affect all biological endpoints studied.

There does not seem to be a consistent relationship between intensity (power density), exposure time, or frequency, and the effects of exposure. On the contrary, and strikingly, higher power densities do not cause more frequent responses, since the percentage of responses in most frequency groups is already at 70%. Some authors refer to their study results as having “non-thermal” causes, but few have applied appropriate temperature controls. The question therefore remains whether warming is the main cause of any observed MMW effects?

In order to evaluate and summarize the 6–100 GHz data in this review, we draw the following conclusions:

- Regarding the health effects of MMW in the 6–100 GHz frequency range at power densities not exceeding the exposure guidelines the studies provide no clear evidence, due to contradictory information from the in vivo and in vitro investigations.
- Regarding the possibility of “non-thermal” effects, the available studies provide no clear explanation of any mode of action of observed effects.
- Regarding the quality of the presented studies, too few studies fulfill the minimal quality criteria to allow any further conclusions.

Supplementary Materials

The following are available online at <https://www.mdpi.com/1660-4601/16/18/3406/s1>, Table S1: Master-table of the selected (in vivo and in vitro) studies and the extracted physical, biological, and quality parameters.

[/pmc/articles/PMC6765906/bin/ijerph-16-03406-s001.pdf]Click here for additional data file. ^(191K, pdf)

Author Contributions

M.S. and M.-O.M. have contributed equally to conceptualization, structuring, data collection and analysis, interpretation of data, and all aspects of writing of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

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[Comment] Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest Open Access

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Abstract

The fifth generation, 5G, of radiofrequency (RF) radiation is about to be implemented

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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 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

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Several Swiss citizens have brought to our attention that Associate Professor Martin Rösli 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 Rösli 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 Rösli, 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 Rösli, 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 Rösli 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 members

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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).

	<p>Table I.</p> <p>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).</p>
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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 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

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 Rössli (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/m² (Tables 5 and 7 in ref. no. 6) to 40 W/m² (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*

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, Emelie 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, Rööslö 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*

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.

'In 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.

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

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 Rösli *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 Rösli *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 Rösli 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 Rösli *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 Rösli *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 Rösli *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

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.

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 Rössli 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 foundations [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

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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Coates' Canons NC Local Government Law

Topping Off a Telecommunication Tower

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Author Name: David Owens

Cell tower construction is a hot zoning topic. The proliferation of connected devices has substantially increased the demand for wireless data transmission. Everyone wants good cell reception and high quality access for their mobile devices. But no one wants to look at a cell tower out their back door. This demand for more and higher quality wireless coverage, combined with widespread concern about the aesthetic impact of telecommunication towers, has sparked a considerable amount of legislation and litigation, not to mention many heated zoning hearings.

Both state and federal regulations attempt to reconcile these competing public interests. Federal statutes and FCC guidance affect an issue that increasingly arises with cell towers and comparable provisions have been written into state law. Suppose there is an existing, permitted cell tower that was built to the maximum height and size allowed by local zoning regulations. The tower owner now has an opportunity to lease space to a provider who wants to collocate a new antennae array on this tower, but there is not enough space on the tower for an additional antennae array.

If the tower owner applies for a permit modification to add height and new antennae to the tower, this new legislation dictates whether the local government has to approve it. Can the tower owner top off the tower? In many instances the answer is yes, the local government will have to approve the modification.

Initial Federal Provisions

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Federal law preemption of local zoning of telecommunication towers is not a new issue. There is a strong national interest in having an effective wireless network. Although some advocates recommended regulation of tower location and construction exclusively by the federal government, Congress recognized that the means of providing wireless service could have significant impact on important community interests. So a compromise was reached that allowed some local land use regulation of telecommunication towers, but with restrictions to protect national interests.

The Telecommunications Act of 1996 allows local regulation of the location of personal wireless services but provides for limited preemption of local ordinances in specified situations. 47 U.S.C. § 332(c)(7)(B) (2010). This law limits local regulation of cell towers as follows:

1. Local regulations based on the environmental health effects of radio frequency emissions are prohibited.
2. Local governments are required to act on permit applications for wireless telecommunication facilities within a reasonable time. The Federal Communications Commission (FCC) subsequently issued an order setting out “presumptively reasonable” times of 90 days to review a collocation application and 150 days to review an application for a new tower. 24 FCC Rcd 13994 (2009). See this [blog post](#) regarding this “shot clock” regulation and related state statutes.
3. Denials of applications for wireless telecommunication facilities must be supported by substantial evidence in the hearing record.
4. Local regulations may not unreasonably discriminate among providers of functionally equivalent wireless telecommunication facilities. Regulations may not set a preference for one type of wireless technology over another, may not favor publicly owned facilities over privately owned ones, or favor an initial provider of services over subsequent competitors.
5. Local regulations may not prohibit or have the effect of prohibiting the provision of personal wireless services. Prohibition includes not only a general ban on all towers in a jurisdiction, but also policies that will result in all possible sites in a given area being rejected.
6. Denials of applications for wireless telecommunication facilities must be in writing.

Initial State Legislation

In 2007 the General Assembly added state statutory restrictions on local government regulation of cell towers. G.S. 160D-930 to -938 allow local land use regulation of wireless telecommunication facilities, but add several limits on this authority:

1. A local land use regulation may not require information on an applicant’s “business decisions,” specifically including information about customer demand or quality of service. A local government is allowed to consider whether an existing or previously approved structure can reasonably be used to provide service, whether residential, historic, and designated scenic areas can be served from outside those areas, and whether the proposed tower height is necessary to provide the applicant’s designated service. **G.S. 160D-933(b)**.
2. Streamlined processing must be provided for qualified collocation applications. Decisions on these applications must be made within 45 days of receipt of a completed application. Decisions on all other applications must be made within a reasonable time consistent with other land use

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<https://canons.sog.unc.edu/2013/03/can-we-top-off-our-tower/>

applications. Collocations entitled to this streamlined process include those that meet a set of specified conditions, including no increase in the height or width of the supporting tower, no increase in ground space for the facility, and any new equipment meeting the weight limits for the structure. The new federal provisions noted below will supersede some of these limits. **G.S. 160D-934**.

3. Local governments are prohibited from requiring that wireless facilities be located on city- or county-owned towers or facilities. They are allowed to provide expedited processing for applications for wireless facilities proposed to be located on city- or county-owned property.
4. Local governments are allowed to charge an application fee that includes fees for consultants to assist in review of permit applications. These fees must be fixed in advance of the application and may not exceed the usual and customary costs of services provided. **G.S. 160D-933(f)**.
5. Local governments may add a condition to zoning approvals for new towers that prevents building permits for the tower being issued until the applicant provides documentation identifying parties intending to locate facilities on the tower. It also may require that permitted facilities be constructed within a reasonable time, provided that time is not less than twenty-four months. **G.S. 160D-933(e)**.

Updated Federal and State Legislation on Collocation and Small Cell Wireless

When Congress adopted the Middle Class Tax Relief and Job Creation Act of 2012 (Pub. L. 112-96), it included a number of other special provisions, including a section on wireless telecommunication that addressed spectrum management, facilities on federal lands, and public emergency technology.

Tucked into these special provisions was Section 6409(a), which broadens the federal preemption of local cell tower regulations. This law provides that state or local governments “shall approve” any eligible request to modify an existing wireless tower or base station that does not “substantially change” the tower or base station. Eligible requests include collocation of new transmission equipment and replacement of existing equipment.

This mandate raised the obvious question of just what constitutes a “substantial change” that must be approved. The FCC provided notice that it interprets the new law using the same standards for defining a “substantial modification” that were previously set in the context of reviewing collocation agreements and facilities in historic districts. Nationwide Programmatic Agreement for the Collocation of Wireless Antennas, § I.C, 47 C.F.R. Part 1, App. B.

The **FCC guidance** provided that it is not a substantial change if: (1) the height of the tower is not increased by more than 10%; (2) the addition will not extend more than 20 feet from the tower; (3) it will add no more than one equipment shelter or four equipment cabinets; and (4) it will not involve excavation outside the tower site or existing utility and access easements. Proposed modifications to existing towers that fall within these guidelines must be approved by local governments.

<https://canons.sog.unc.edu/2013/03/can-we-top-off-our-tower/>

The FCC guidance goes on to address several other questions raised by the new legislation. It interprets the law as applying to both telecommunication towers and to other structures that support or house an antennae and to include emerging technologies such as distributed antenna systems and small cells. It does not affect collocations on structures other than wireless towers or base stations. It provided that a local government may require an application for administrative approval, but that such applications must be approved within 90 days.

The General Assembly then amended state law to add provisions similar to the federal legislation. S.L. 2013-185 created **G.S. 160D-934** to require expedited review of collocations and minor modifications of existing towers. Minor modifications — include adding not more than 10% to the height or one additional antenna array, adding not more than 20 feet in width, or not more than 2,500 sq. ft. to the existing ground equipment compound — must be approved. The law also caps application fees for collocation reviews at \$1,000. Similarly, provisions for expedited review of small cell wireless facilities were in 2019 added to state law as **G.S. 160D-935**.

So, can a tower owner top off an existing tower that is already at the maximum height allowed by the zoning ordinance in order to install additional antennae?

Yes, provided the addition does not increase the tower height by more than 10%, protrude more than 20 feet from the tower, and involves only relatively modest ground work within the tower site. Eligible modifications must be approved “notwithstanding . . . any other provision of law,” thus overriding any applicable maximum height limit in a local zoning ordinance. An application can be required to verify that a proposed modification falls within these limits, but a qualifying, complete application must be approved in 90 days or less according to the FCC’s interpretation of the law.

There may be some exceptions to these limits, but the law and guidance leave some important questions unaddressed. Presumably applicants will need to document that they have legal authority to use or modify the tower from the tower owner before they can seek mandatory approval. Presumably a proposed tower modification that adds less than 10% to the height of a tower could be denied if the additional equipment would violate weight limits for the tower, fall zone buffer setbacks, or other safety requirements. But the law is not entirely clear about this and these presumptions may not be valid.

Local zoning ordinances should reflect the preempted modifications as permitted uses, not subject to special use permit reviews or variance procedures. Standards for new towers should be reviewed in light of the possibility of future modification requests that must be approved. A potential for a future

<https://canons.sog.unc.edu/2013/03/can-we-top-off-our-tower/>

10% increase in height or an additional 20 feet protrusion can be particularly significant where “stealth” design is involved, so this may need to be factored into standards for review of proposals for new towers.

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Fw: Montreat ordinance on wireless communications

Kayla DiCristina <kayla@landofsky.org>

Tue 11/8/2022 8:39 AM

To: Bill tucker <wotucker@gmail.com>;crawfordjohna@aol.com <crawfordjohna@aol.com>;danielbluedean@gmail.com <danielbluedean@gmail.com>;johndora@charter.net <johndora@charter.net>;julie19923@gmail.com <julie19923@gmail.com>;lhjohnson62@gmail.com <lhjohnson62@gmail.com>;sfstansill58@gmail.com <sfstansill58@gmail.com>;wdbmountainliving@gmail.com <wdbmountainliving@gmail.com>;wscheu@rtlaw.com <wscheu@rtlaw.com>

Cc: Ben Blackburn <bblackburn@townofmontreat.org>;Angela Murphy <amurphy@townofmontreat.org>

Good morning Board Members,

Below is an email received from Jane Warner regarding TA-2022-02. I will provide a copy of her email in your packets and you have access to the studies she cites digitally. Attached is also an article from the School of Government regarding zoning regulations on wireless communications towers and below is a link to the Telecommunications Act of 1996 (these are available digitally and will not be a part of your packets). In sum, the Telecommunications Act of 1996, prohibits local regulations based on the environmental health effects of radio frequency emissions. As a reminder, the scope of this text amendment is to bring this ordinance into compliance with General Statute revisions. Additional amendments should be considered at a later date.

<https://transition.fcc.gov/Reports/tcom1996.pdf>

Best,

Kayla DiCristina, AICP

(*For inquiries regarding the Town of Montreat, please see below)

Regional Planner | Economic and Community Development

Land of Sky Regional Council

339 New Leicester Hwy., Suite 140 • Asheville, NC 28806



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***Town of Montreat:** Inquiries regarding the Town of Montreat are answered in the order they are received during Montreat office hours every Tuesday through Thursday 8:00 am through 5:00 pm. For assistance, please call 828-669-8002, ext. 3030, or e-mail zoning@townofmontreat.org.

This institution is an equal opportunity provider and employer. All email correspondence to and from this address is subject to public review under the NC Public Records Law.

From: Jane Warner <skynetreegirl@mailfence.com>

Sent: Sunday, November 6, 2022 1:13 PM

To: thelms@townofmontreat.org <thelms@townofmontreat.org>

Cc: Commissioner Kitty Fouche <kfouche@townofmontreat.org>; Commissioner Mason Blake <mblake@townofmontreat.org>; Mayor Pro-Tem Kent Otto <kotto@townofmontreat.org>; Commissioner Jane Alexander <jalexander@townofmontreat.org>; Commissioner Tom Widmer <twidmer@townofmontreat.org>; julie19923@gmail.com <julie19923@gmail.com>; lhjohnson62@gmail.com <lhjohnson62@gmail.com>; sfstnsill58@gmail.com <sfstnsill58@gmail.com>; Bill Scheu <WScheu@rtlaw.com>; johndora@charter.net <johndora@charter.net>; danielbluedean@gmail.com <danielbluedean@gmail.com>; Kayla DiCristina <kayla@landofsky.org>; info@townofmontreat.org <info@townofmontreat.org>

Subject: Montreat ordinance on wireless communications

Be Advised: This email originated from outside Land of Sky

Dear Mayor, Commissioners, Zoning Administrator and zoning commission members,

I am writing in regard to the revisions of the Montreat ordinance on wireless communications equipment installation to bring it into alignment with the state of North Carolina. I find the approval process to be alarmingly beneficial to the telecommunications industry, and alarmingly oblivious of potential harm to residents. I would urge that Montreat take a more conservative and more healthful position with regard to such installations. I believe that it should not simply be that the zoning administrator can sign off on such, but that there should be robust community involvement in decisions to approve additional installations of wireless communication equipment.

I would urge a moratorium on new 5G installations.

I believe we all know that there is a major cell tower at South Carolina Inn.

Across the planet, there are varying levels of community concern about electromagnetic wave lengths, cell tower installations, etc. Some communities have chosen to freeze approval indefinitely for 5G installations. There is simply not enough known about health risks. In October, I shared a link to an appeal by scientist David Carpenter to the school board of Portland Oregon, requesting that Wi-Fi equipment be removed from schools and instead wired connections be installed. I've attached a pdf of that report. Dr. Carpenter attached approximately 400 scientific studies supporting this recommendation. I am also attaching here the report by the International Commission on the Biological Effects of Electromagnetic Fields, alerting to the dangers of Wi-Fi and electromagnetic fields. It's a long report, but their findings include the following:

- The limits set for radiofrequency radiation established by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) and the Federal Communications Commission (FCC) are based upon invalid assumptions and outdated science; they are not protective of human health.
- That there be an independent assessment of the dangers of radio frequency radiation based on scientific evidence from peer-reviewed studies conducted over the past 25 years. They are seeking health standards for workers and the public.
- That the public be informed of the health risks of EMF and encouraged to do everything they can to minimize exposures, especially for children, pregnant women and people who are hypersensitive.
- That there be an immediate moratorium on further rollout of 5G wireless technology until safety is actually demonstrated.

Individuals have widely differing responses to electromagnetic fields. For some it can bring about powerful acute health effects, for others a general malaise. It is quite true that the majority of people do not experience acute effects, however, as the studies I have provided show, they may experience significant adverse health effects over time.

I am going to share my own experience. I have been moderately to acutely sensitive to electromagnetic fields and wavelengths for about 18 years. I have difficulty being around other people's cell phones, Wi-Fi-connected computers, etc. It's inconvenient to say the very least.

Four or five years ago, my husband and I were in negotiations to purchase a house on South Carolina Terrace. I came to Montreat and stayed in my sisters' home on North Carolina Terrace. Because of my sensitivities, I repeatedly visited the house on South Carolina Terrace to see if it was a tolerable space for me. Over and over, I found that I was a bit dizzy when I was on the front porch, and it took some time for this feeling to subside after I left. I did not have an explanation for this until I was walking across the bridge by the Moore center and looked up and saw the cell tower at South Carolina Inn. I hadn't known it was there. That was the explanation. The house we were in negotiations to purchase was about 600 feet away. Needless to say, we did not purchase it.

I mention this to illustrate that the wavelengths of such towers are more than perceptible.

I am far from alone in my difficulty. When I wrote to the town Council in October, I heard back from another Montreater, Shelley Stevens, whose body is even more sensitive than mine. I have asked her to write up her experience and share it.

11/8/22, 8:41 AM

Mail - Kayla DiCristina - Outlook

I would like Montreat to continue to be a safe and healthful environment. Please help us by making Montreat's ordinance tougher than the state of North Carolina's. Sensitive people will thank you. And many people view us as the canaries in the coal mine regarding electromagnetic force fields. You will not be hurting your own health and likely you will be helping to preserve your physical well-being.

Thank you for reading this.

Jane Warner

346 Chapman Rd.

Montreat ordinance on wireless communications

Jane Warner <skylinetreegirl@mailfence.com>

Sun 11/6/2022 1:15 PM

To: thelms@townofmontreat.org <thelms@townofmontreat.org>

Cc: Commissioner Kitty Fouche <kfouche@townofmontreat.org>; Commissioner Mason Blake <mblake@townofmontreat.org>; Mayor Pro-Tem Kent Otto <kotto@townofmontreat.org>; Commissioner Jane Alexander <jalexander@townofmontreat.org>; Commissioner Tom Widmer <twidmer@townofmontreat.org>; julie19923@gmail.com <julie19923@gmail.com>; lhjohnson62@gmail.com <lhjohnson62@gmail.com>; sfstnsill58@gmail.com <sfstnsill58@gmail.com>; Bill Scheu <WScheu@rtlaw.com>; johndora@charter.net <johndora@charter.net>; danielbluedean@gmail.com <danielbluedean@gmail.com>; Kayla DiCristina <kayla@landofsky.org>; info@townofmontreat.org <info@townofmontreat.org>

📎 2 attachments (3 MB)

Carpenter EMF.pdf; ICBE-EMF.pdf;

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Thank you for reading this.

Jane Warner

346 Chapman Rd.

Wireless communications regulation

ltlredwagon <ltlredwagon@mailfence.com>

Mon 11/14/2022 8:56 PM

To: thelms@townofmontreat.org <thelms@townofmontreat.org>

Cc: Commissioner Kitty Fouche <kfouche@townofmontreat.org>; Commissioner Mason Blake <mblake@townofmontreat.org>; Mayor Pro-Tem Kent Otto <kotto@townofmontreat.org>; Commissioner Jane Alexander <jalexander@townofmontreat.org>; Commissioner Tom Widmer <twidmer@townofmontreat.org>; Julie Schell <julie19923@gmail.com>; wscheu@rtlaw.com <wscheu@rtlaw.com>; Kayla DiCristina <kayla@landofsky.org>; sfstansill58@gmail.com <sfstansill58@gmail.com>; Angela Murphy <amurphy@townofmontreat.org>

 7 attachments (10 MB)

Are Contaminants Silencing Our Genes - Scientific American.pdf; Carpenter EMF.pdf; ICBE-EMF.pdf; EMF Genetic Effects.pdf; Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest.pdf; Oncology Letters.pdf; Why Most Published Research Findings Are False - PMC.pdf;

You don't often get email from ltlredwagon@mailfence.com. [Learn why this is important](#)

Be Advised: This email originated from outside Land of Sky

Dear Mayor and Commissioners of Montreat,

My wife, Jane, recently wrote to you regarding regulations governing wireless communication installations in Montreat. My understanding is that there are no equipment installations planned for the immediate future and that this matter may be examined again in the new year. Perhaps this email can be set aside for review at that time.

Whenever you do take up this matter, I would like to offer the following data for your consideration. I support the recommendation of my wife that Montreat take a more proactive stance to protect Montreaters from electromagnetic frequency radiation (EMF).

Here is the fundamental problem as I see it. Some may disagree, but I would say that history reveals humans to be fantastically creative and capable as problem solvers, but often expedient, even careless at times, in getting things done. I'll provide a few examples below.

In seeking to understand a topic it is sometimes helpful to make comparisons to something which is more familiar. EMF radiation, as a global phenomenon, is a very recent concern. It was only 30 years ago that reduced cell phone size enormously increased the popularity of cell phone usage. Complete internet service on the mobile web has only been available for a little over 20 years – mere seconds when it comes to biological research.

But the phenomenon is not without precedent. A similar situation existed in the mid-20th century with regard to skyrocketing chemical use in agriculture and industry. (In the 1980s I spent about 8 years writing for various trade journals – agriculture, industry, police, firefighting – on human exposure to toxic chemicals; I'm no expert, but I know a bit about the subject.) At that time the general population was being exposed to thousands of toxic chemicals and there were no studies which had examined the effects of prolonged exposure to such chemicals over a lifetime (particularly at low levels) and, through placental blood transfer and other factors, over many generations. The renowned environmental scientist and pathologist, Dr. Rene Dubos of the Harvard Medical School, sounded the alarm in 1968 in the journal *Environmental Scientist*, writing, "The greatest danger of pollution may well be that we shall tolerate levels of it so low as to have no acute nuisance value, but sufficiently high, nevertheless, to cause delayed disease and spoil the quality of life." Dubos is credited with popularizing the environmental maxim, "Think globally, act locally".

Dubos words were prophetic. The US Environmental Protection Agency's National Human Adipose Tissue Survey (NHATS) has found dozens of carcinogenic chemicals (herbicides, pesticides, industrial chemicals such as PCBs) at low levels in virtually every human ever tested. According to a report in the journal, *Environment International*, these chemicals "concentrate in fatty

tissues and bioaccumulate as they move up the food chain; travel long distances in global air and water currents; and have been linked with serious health effects in humans, even at low exposures" (*Environment International*, Vol. 39, Issue 1, Feb., 2012).

We are now in the middle of a decades-long human experiment with low-level chemical exposure on a global scale, the effects of which we simply have not been able to fully study. There are so many substances which were tested and "proven" safe at given levels by top scientists decades ago. Now we know more. If you will allow me to give just one example. This year the EPA reported that PFAS (per- and poly-fluoroalkyl substances), carcinogenic chemicals in use since the 1940s for nonstick cookware, fabrics and flame-retardant equipment, are far more dangerous than previously known. The EPA has set new levels which are 3,000 to 17,000 times lower than previous standards (<https://www.washingtonpost.com/climate-environment/2022/06/15/epa-pfas-forever-chemicals/>). PFAS are in the drinking water of a majority of Americans and in the blood of almost everyone, but one component of the environmental and internal chemical "soup" we now live with. It's worth pausing to consider this: the adverse effects of a chemical in use for 80 years are only now being understood. (Teflon pans – never caused me any problems, right?)

There are also concerns that "chemicals in our environment and in our food can alter genes, leaving people vulnerable to a variety of diseases and disorders." ("Are Contaminants Silencing our Genes?", *Scientific American*, Aug 3., 2009.)

It is my opinion that a very similar global experiment is now occurring with electromagnetic frequency radiation (EMF). My wife has forwarded to you the extensive review of scientific literature on this subject which David Carpenter, M.D. provided when the Portland Public Schools were considering the effects of EMF on children. Dr. Carpenter, a graduate of the Harvard Medical School, is the Director of the Institute for Health and the Environment at the University of Albany. Dr. Carpenter received no compensation for his testimony.

Dr. Carpenter concluded:

"Chronic exposure to PM MW [pulse-modulated microwave] radiation harms every individual in a population in some ways, even if these are not always detectable by the individual or consciously attributed to the responsible RF/MW [radiofrequency/microwave] radiation sources. This Agent injures some individuals into a condition in which symptoms will be more easily retriggered with subsequent exposure."

This year the International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF) published a peer-reviewed paper entitled: "Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G" (*Environmental Health* (2022) 21:92 <https://doi.org/10.1186/s12940-022-00900-9>). My wife has already summarized the findings, as follows:

- **The limits set for radiofrequency radiation established by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) and the Federal Communications Commission (FCC) are based upon invalid assumptions and outdated science; they are not protective of human health.**
- **That there be an independent assessment of the dangers of radio frequency radiation based on scientific evidence from peer-reviewed studies conducted over the past 25 years. They are seeking health standards for workers and the public.**
- **That the public be informed of the health risks of EMF and encouraged to do everything they can to minimize exposures, especially for children, pregnant women and people who are hypersensitive.**
- **That there be an immediate moratorium on further rollout of 5G wireless technology until safety is actually demonstrated.**

As with chemical pollutants, there is also concern over the genetic effects of EMF. The journal, *Electromagnetic Biology and Medicine* (Electromag Biol Med. 2021 Apr 3;40(2):264-273. doi: 10.1080/15368378.2021.1881866. Epub 2021 Feb 4) reported last year:

"Genetic effects of EMF depend on various factors, including field parameters and characteristics (frequency, intensity, wave-shape), cell type, and exposure duration. The types of gene expression affected (e.g., genes involved in cell cycle arrest, apoptosis and stress responses, heat-shock proteins) are consistent with the findings that EMF causes genetic damages. Many studies reported effects in cells and animals after exposure to EMF at intensities similar to those in the public and occupational environments. The mechanisms by which effects are induced by EMF are basically unknown."

You may also wish to review a study published in the journal, *Oncology Letters*, in 2020 (*Oncology Letters*, 2020 October; 20(4): 15), entitled, "Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest". The researchers wrote:

"The fifth generation, 5G, of radio frequency 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, the 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.”

With regard to this last report, I would particularly like to draw your attention to two important peer-reviewed studies. In 2016, 40% of scientists surveyed by the journal, *Nature* (1500 scientists) believed that fraud was always or often a factor in research. This is the scientific community critiquing itself. An astonishing 70% cited the bias of “selective reporting”, the suppression of undesirable facts and findings (*Nature*, Vol. 533, pages 452–454, 2016). John Ioannidis, M.D., of the Stanford University School of Medicine reached a similar conclusion. Ioannidis is an internationally recognized expert in the study of scientific research. In 2005 he published a paper entitled, “Why most published research findings are false” (*PLoS Med.* 2005 Aug;2(8):e124. doi:10.1371/journal.pmed.0020124). Ioannidis reported:

“The greater the financial and other interests and prejudices in a scientific field, the less likely the research findings are to be true. Conflicts of interest are very common in biomedical research, and typically they are inadequately and sparsely reported.”

The question for Montreat is whether or not it will, by default, side with industry and continue the EMF experiment, or whether it will act now to reduce the exposure of Montreaters to EMF. Will there be, to quote Dubos, very little in the way of “acute nuisance value” but eventual “delayed disease”? If you research this field you will likely encounter those who will tell you that there are no studies which have proven long-term adverse health effects on populations exposed to EMF. Of course, they are absolutely right! But those who wish to act responsibly must then ask themselves the *other* question: **While most people do not experience, or do not notice, any acute effects, are there any studies which prove that there are NO long-term adverse health effects from prolonged exposure to EMF radiation?** Who has the burden to answer that question? Industry, yes; medicine, yes. But you and I as well.

Look at our oceans and rivers, our air, our soil, our food supply. It’s hard to argue the charge that humans tend to be expedient: push ahead now, “get it done” – ask questions later. Are we making the same mistake with EMF?

I believe that there is more than enough evidence in the scientific research literature to support a decision by the government of Montreat to place a moratorium on all 5G installations in Montreat until their apparent dangers are fully researched. I hope the government of Montreat will place the safety of its citizens above all other concerns.

Sincerely,
Bob Warner
346 Chapman Road

ATTACHMENTS:

1. “Are Contaminants Silencing our Genes?”, *Scientific American*, Aug 3., 2009
2. David O. Carpenter, M.D., Declaration on EMF United States District Court, Portland, Oregon, 2011
3. “Scientific evidence invalidates health assumptions underlying the FCC and ICNIRP exposure limit determinations for radiofrequency radiation: implications for 5G” (*Environmental Health* (2022) 21:92 <https://doi.org/10.1186/s12940-022-00900-9>).
4. “Genetic effects of non-ionizing electromagnetic fields,” *Electromagnetic Biology and Medicine*, Vol. 40, 2021 - Issue 2
5. “Health risks from radiofrequency radiation, including 5G, should be assessed by experts with no conflicts of interest”, *Oncology Letters*, 2020 October; 20(4):15)
6. “1500 scientists lift the lid on reproducibility,” *Nature* 533, 452-454 (2016)
7. “Why Most Published Research Findings Are False,” John Ioannidis, M.D., *PLoS Med.* 2005 Aug; 2(8): e124

Fwd: Urgent message re: wireless communication Installations, Community concerns

Angela Murphy <amurphy@townofmontreat.org>

Thu 10/13/2022 10:05 AM

To: Kayla DiCristina <kayla@landofsky.org>

 1 attachments (226 KB)

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From: skylinetreegirl <skylinetreegirl@mailfence.com>**Sent:** Thursday, October 13, 2022 10:03:05 AM**To:** Angela Murphy <amurphy@townofmontreat.org>**Subject:** Urgent message re: wireless communication Installations, Community concerns

Dear Planning and Zoning board members, and Board of Commissioners members,

I see that The wireless communications ordinance is to be addressed today in the Planning and Zoning meeting. I want to bring up the importance of keeping Montreat a safe place for all.

There are many health concerns with regard to wireless communication. Attached please find a PDF submitted to the town of Portland regarding Wi-Fi in schools, raising an alarm supported by 1500 scientific studies about the potential adverse effects.

I know that today, Planning and Zoning is simply addressing changes of wording and updating the town ordinance to align with the state of North Carolina's regulations. However, I think it's very important to understand that wireless communication equipment, which might be casually approved, potentially causes health effects broadcast over the entire town.

There is much information about communities all over the world which are not permitting such installations due to health concerns and studies revealing adverse effects on a community. I feel that there should be a robust opportunity for community input whenever there is something relating to Radio Frequency (RF) radiation being installed in our town.

I will do further research and forward more information.

Thank you very much.

Jane Warner

346 Chapman Rd.

Why Most Published Research Findings Are False

[John P. A. Ioannidis](#)

Summary

There is increasing concern that most current published research findings are false. The probability that a research claim is true may depend on study power and bias, the number of other studies on the same question, and, importantly, the ratio of true to no relationships among the relationships probed in each scientific field. In this framework, a research finding is less likely to be true when the studies conducted in a field are smaller; when effect sizes are smaller; when there is a greater number and lesser preselection of tested relationships; where there is greater flexibility in designs, definitions, outcomes, and analytical modes; when there is greater financial and other interest and prejudice; and when more teams are involved in a scientific field in chase of statistical significance. Simulations show that for most study designs and settings, it is more likely for a research claim to be false than true. Moreover, for many current scientific fields, claimed research findings may often be simply accurate measures of the prevailing bias. In this essay, I discuss the implications of these problems for the conduct and interpretation of research.

Published research findings are sometimes refuted by subsequent evidence, with ensuing confusion and disappointment. Refutation and controversy is seen across the range of research designs, from clinical trials and traditional epidemiological studies [[1–3](#)] to the most modern molecular research [[4,5](#)]. There is increasing concern that in modern research, false findings may be the majority or even the vast majority of published research claims [[6–8](#)]. However, this should not be surprising. It can be proven that most claimed research findings are false. Here I will examine the key factors that influence this problem and some corollaries thereof.

Modeling the Framework for False Positive Findings

Several methodologists have pointed out [[9–11](#)] that the high rate of nonreplication (lack of confirmation) of research discoveries is a consequence of the convenient, yet ill-founded strategy of claiming conclusive research findings solely on the basis of a single study assessed by formal statistical significance, typically for a p -value less than 0.05. Research is not most appropriately represented and summarized by p -values, but, unfortunately, there is a widespread notion that medical research articles should be interpreted based only on p -values. Research findings are defined here as any relationship reaching formal statistical signifi-

cance, e.g., effective interventions, informative predictors, risk factors, or associations. “Negative” research is also very useful. “Negative” is actually a misnomer, and the misinterpretation is widespread. However, here we will target relationships that investigators claim exist, rather than null findings.

It can be proven that most claimed research findings are false

As has been shown previously, the probability that a research finding is indeed true depends on the prior probability of it being true (before doing the study), the statistical power of the study, and the level of statistical significance [10,11]. Consider a 2×2 table in which research findings are compared against the gold standard of true relationships in a scientific field. In a research field both true and false hypotheses can be made about the presence of relationships. Let R be the ratio of the number of “true relationships” to “no relationships” among those tested in the field. R is characteristic of the field and can vary a lot depending on whether the field targets highly likely relationships or searches for only one or a few true relationships among thousands and millions of hypotheses that may be postulated. Let us also consider, for computational simplicity, circumscribed fields where either there is only one true relationship (among many that can be hypothesized) or the power is similar to find any of the several existing true relationships. The pre-study probability of a relationship being true is $R/(R + 1)$. The probability of a study finding a true relationship reflects the power $1 - \beta$ (one minus the Type II error rate). The probability of claiming a relationship when none truly exists reflects the Type I error rate, α . Assuming that c relationships are being probed in the field, the expected values of the 2×2 table are given in Table 1. After a research finding has been claimed based on achieving formal statistical significance, the post-study probability that it is true is the positive predictive value, PPV. The PPV is also the complementary probability of what Wacholder et al. have called the false positive report probability [10]. According to the 2×2 table, one gets $PPV = (1 - \beta)R/(R - \beta R + \alpha)$. A research finding is thus more likely true than false if $(1 - \beta)R > \alpha$. Since usually the vast majority of investigators depend on $\alpha = 0.05$, this means that a research finding is more likely true than false if $(1 - \beta)R > 0.05$.

Table 1

Research Findings and True Relationships

Research Finding	True Relationship		Total
	Yes	No	
Yes	$c(1 - \beta)R/(R + 1)$	$c\alpha/(R + 1)$	$c(R + \alpha - \beta R)/(R + 1)$
No	$c\beta R/(R + 1)$	$c(1 - \alpha)/(R + 1)$	$c(1 - \alpha + \beta R)/(R + 1)$
Total	$cR/(R + 1)$	$c/(R + 1)$	c

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What is less well appreciated is that bias and the extent of repeated independent testing by different teams of investigators around the globe may further distort this picture and may lead to even smaller probabilities of the research findings being indeed true. We will try to model these two factors in the context of similar 2×2 tables.

First, let us define bias as the combination of various design, data, analysis, and presentation factors that tend to produce research findings when they should not be produced. Let u be the proportion of probed analyses that would not have been “research findings,” but nevertheless end up presented and reported as such, because of bias. Bias should not be confused with chance variability that causes some findings to be false by chance even though the study design, data, analysis, and presentation are perfect. Bias can entail manipulation in the analysis or reporting of findings. Selective or distorted reporting is a typical form of such bias. We may assume that u does not depend on whether a true relationship exists or not. This is not an unreasonable assumption, since typically it is impossible to know which relationships are indeed true. In the presence of bias ([Table 2](#)), one gets $PPV = ([1 - \beta]R + u\beta R)/(R + \alpha - \beta R + u - u\alpha + u\beta R)$, and PPV decreases with increasing u , unless $1 - \beta \leq \alpha$, i.e., $1 - \beta \leq 0.05$ for most situations. Thus, with increasing bias, the chances that a research finding is true diminish considerably. This is shown for different levels of power and for different pre-study odds in [Figure 1](#). Conversely, true research findings may occasionally be annulled because of reverse bias. For example, with large measurement errors relationships are lost in noise [[12](#)], or investigators use data inefficiently or fail to notice statistically significant relationships, or there may be conflicts of interest that tend to “bury” significant findings [[13](#)]. There is no good large-scale empirical evidence on how frequently such reverse bias may occur across diverse research fields. However, it is probably fair to say that reverse bias is not as common. Moreover measurement errors and inefficient use of data are probably becoming less frequent problems, since measurement error has decreased with technological advances in the molecular era and investigators are becoming increasingly sophisticated about their data. Regardless, reverse bias may be modeled in the same way as bias above. Also reverse bias should not be confused with chance variability that may lead to missing a true relationship because of chance.

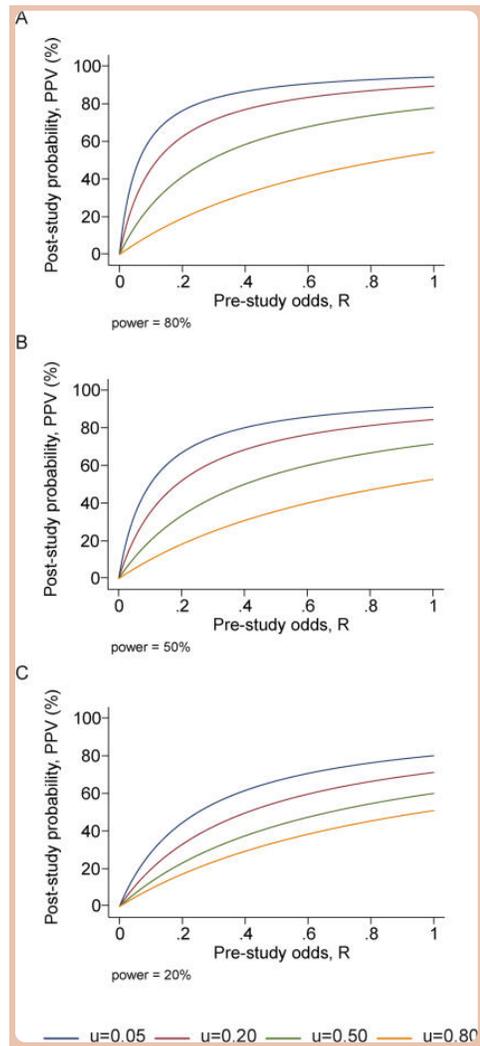


Figure 1

PPV (Probability That a Research Finding Is True) as a Function of the Pre-Study Odds for Various Levels of Bias, u

Panels correspond to power of 0.20, 0.50, and 0.80.

Table 2

Research Findings and True Relationships in the Presence of Bias

Research Finding	True Relationship		
	Yes	No	Total
Yes	$\frac{c[1 - \beta]R + u\beta R}{R + 1}$	$\frac{c\alpha + u(1 - \alpha)}{R + 1}$	$\frac{c(R + \alpha - \beta R + u - u\alpha + u\beta R)}{R + 1}$
No	$\frac{(1 - u)c\beta R}{R + 1}$	$\frac{(1 - u)c(1 - \alpha)}{R + 1}$	$\frac{c(1 - u)(1 - \alpha + \beta R)}{R + 1}$
Total	$\frac{cR}{R + 1}$	$\frac{c}{R + 1}$	c

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Testing by Several Independent Teams

Several independent teams may be addressing the same sets of research questions. As research efforts are globalized, it is practically the rule that several research teams, often dozens of them, may probe the same or similar questions. Unfortunately, in some areas, the prevailing mentality until now has been to focus on isolated discoveries by single teams and interpret research experiments in isolation. An increasing number of questions have at least one study claiming a research finding, and this receives unilateral attention. The probability that at least one study, among several done on the same question, claims a statistically significant research finding is easy to estimate. For n independent studies of equal power, the 2×2 table is shown in [Table 3](#): $PPV = R(1 - \beta^n)/(R + 1 - [1 - \alpha]^n - R\beta^n)$ (not considering bias). With increasing number of independent studies, PPV tends to decrease, unless $1 - \beta < \alpha$, i.e., typically $1 - \beta < 0.05$. This is shown for different levels of power and for different pre-study odds in [Figure 2](#). For n studies of different power, the term β^n is replaced by the product of the terms β_i for $i = 1$ to n , but inferences are similar.

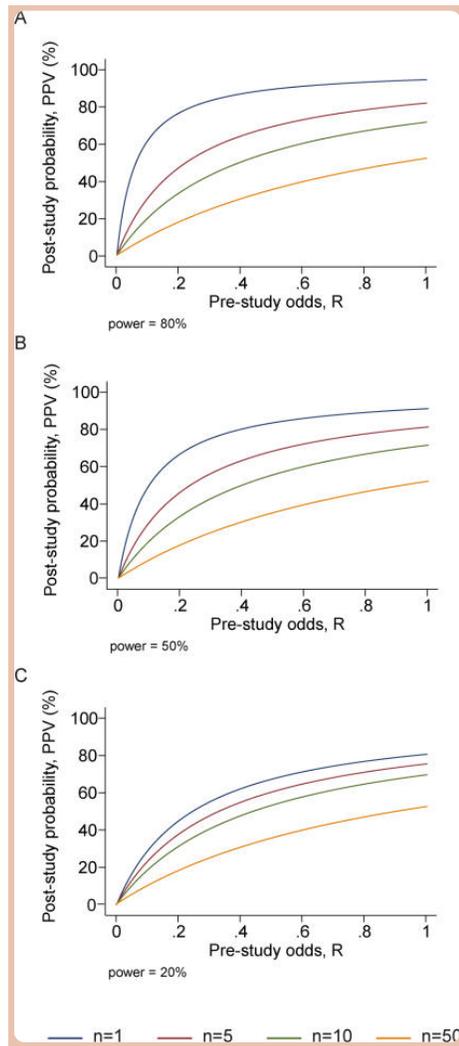


Figure 2

PPV (Probability That a Research Finding Is True) as a Function of the Pre-Study Odds for Various Numbers of Conducted Studies, n

Panels correspond to power of 0.20, 0.50, and 0.80.

Table 3

Research Findings and True Relationships in the Presence of Multiple Studies

Research Finding	True Relationship		
	Yes	No	Total
Yes	$cR(1 - \beta^n)/(R + 1)$	$c(1 - [1 - \alpha]^n)/(R + 1)$	$c(R + 1 - [1 - \alpha]^n - R\beta^n)/(R + 1)$
No	$cR\beta^n/(R + 1)$	$c(1 - \alpha)^n/(R + 1)$	$c([1 - \alpha]^n + R\beta^n)/(R + 1)$
Total	$cR/(R + 1)$	$c/(R + 1)$	c

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A practical example is shown in [Box 1](#). Based on the above considerations, one may deduce several interesting corollaries about the probability that a research finding is indeed true.

Box 1. An Example: Science at Low Pre-Study Odds

Let us assume that a team of investigators performs a whole genome association study to test whether any of 100,000 gene polymorphisms are associated with susceptibility to schizophrenia. Based on what we know about the extent of heritability of the disease, it is reasonable to expect that probably around ten gene polymorphisms among those tested would be truly associated with schizophrenia, with relatively similar odds ratios around 1.3 for the ten or so polymorphisms and with a fairly similar power to identify any of them. Then $R = 10/100,000 = 10^{-4}$, and the pre-study probability for any polymorphism to be associated with schizophrenia is also $R/(R + 1) = 10^{-4}$. Let us also suppose that the study has 60% power to find an association with an odds ratio of 1.3 at $\alpha = 0.05$. Then it can be estimated that if a statistically significant association is found with the p -value barely crossing the 0.05 threshold, the post-study probability that this is true increases about 12-fold compared with the pre-study probability, but it is still only 12×10^{-4} .

Now let us suppose that the investigators manipulate their design, analyses, and reporting so as to make more relationships cross the $p = 0.05$ threshold even though this would not have been crossed with a perfectly adhered to design and analysis and with perfect comprehensive reporting of the results, strictly according to the original study plan. Such manipulation could be done, for example, with serendipitous inclusion or exclusion of certain patients or controls, post hoc subgroup analyses, investigation of genetic contrasts that were not originally specified, changes in the disease or control definitions, and various combinations of selective or distorted reporting of the results. Commercially available “data mining” packages actually are proud of their ability to yield statistically significant results through data dredging. In the presence of bias with $u = 0.10$, the post-study probability that a research finding is true is only 4.4×10^{-4} . Furthermore, even in the absence of any bias, when ten independent research teams perform similar experiments around the world, if one of them finds a formally statistically significant association, the probability that the research finding is true is only 1.5×10^{-4} , hardly any higher than the probability we had before any of this extensive research was undertaken!

Corollary 1: The smaller the studies conducted in a scientific field, the less likely the research findings are to be true. Small sample size means smaller power and, for all functions above, the PPV for a true research finding decreases as power decreases towards $1 - \beta = 0.05$. Thus, other factors being equal, research findings are more likely true in scientific fields that undertake large studies, such as randomized controlled trials in cardiology (several thousand subjects randomized) [14] than in scientific fields with small studies, such as most research of molecular predictors (sample sizes 100-fold smaller) [15].

Corollary 2: The smaller the effect sizes in a scientific field, the less likely the research findings are to be true. Power is also related to the effect size. Thus research findings are more likely true in scientific fields with large effects, such as the impact of smoking on cancer or cardiovascular disease (relative risks 3–20), than in scientific fields where postulated effects are small, such as genetic risk factors for multigenetic diseases (relative risks 1.1–1.5) [7]. Modern epidemiology is increasingly obliged to target smaller effect sizes [16]. Consequently, the proportion of true research findings is expected to decrease. In the same line of thinking, if the true effect sizes are very small in a scientific field, this field is likely to be plagued by almost ubiquitous false positive claims. For example, if the majority of true genetic or nutritional determinants of complex diseases confer relative risks less than 1.05, genetic or nutritional epidemiology would be largely utopian endeavors.

Corollary 3: The greater the number and the lesser the selection of tested relationships in a scientific field, the less likely the research findings are to be true. As shown above, the post-study probability that a finding is true (PPV) depends a lot on the pre-study odds (R). Thus, research findings are more likely true in confirmatory designs, such as large phase III randomized controlled trials, or meta-analyses thereof, than in hypothesis-generating experiments. Fields considered highly informative and creative given the wealth of the assembled and tested information, such as microarrays and other high-throughput discovery-oriented research [4,8,17], should have extremely low PPV.

Corollary 4: The greater the flexibility in designs, definitions, outcomes, and analytical modes in a scientific field, the less likely the research findings are to be true. Flexibility increases the potential for transforming what would be “negative” results into “positive” results, i.e., bias, u . For several research designs, e.g., randomized controlled trials [18–20] or meta-analyses [21,22], there have been efforts to standardize their conduct and reporting. Adherence to common standards is likely to increase the proportion of true findings. The same applies to outcomes. True findings may be more common when outcomes are unequivocal and universally agreed (e.g., death) rather than when multifarious outcomes are devised (e.g., scales for schizophrenia outcomes) [23]. Similarly, fields that use commonly agreed, stereotyped analytical methods (e.g., Kaplan-Meier plots and the log-rank test) [24] may yield a larger proportion of true findings than fields where analytical methods are still under experimentation (e.g., artificial intelligence methods) and only “best” results are reported. Regardless, even in the most stringent research designs, bias seems to be a major problem. For example, there is strong evidence that selective outcome reporting, with manipulation of the outcomes and analyses reported, is a common problem even for randomized trials [25]. Simply abolishing selective publication would not make this problem go away.

Corollary 5: The greater the financial and other interests and prejudices in a scientific field, the less likely the research findings are to be true. Conflicts of interest and prejudice may increase bias, u . Conflicts of interest are very common in biomedical research [26], and typically they are inadequately and sparsely reported [26,27]. Prejudice may not necessarily have financial roots. Scientists in a given field may be prejudiced purely because of their belief in a scientific theory or commitment to their own findings. Many otherwise seemingly independent, university-based studies may be conducted for no other reason than to give physicians and researchers qualifications for promotion or tenure. Such nonfinancial conflicts may also lead to distorted reported results and interpretations. Prestigious investigators may suppress via the peer review process the appearance and dissemination of findings that refute their findings, thus condemning their field to perpetuate false dogma. Empirical evidence on expert opinion shows that it is extremely unreliable [28].

Corollary 6: The hotter a scientific field (with more scientific teams involved), the less likely the research findings are to be true. This seemingly paradoxical corollary follows because, as stated above, the PPV of isolated findings decreases when many teams of investigators are involved in the same field. This may explain why we occasionally see major excitement followed rapidly by severe disappointments in fields that draw wide attention. With many teams working on the same field and with massive experimental data being produced, timing is of the essence in beating competition. Thus, each team may prioritize on pursuing and disseminating its most impressive “positive” results. “Negative” results may become attractive for dissemination only if some other team has found a “positive” association on the same question. In that case, it may be attractive to refute a claim made in some prestigious journal. The term Proteus phenomenon has been coined to describe this phenomenon of rapidly alternating extreme research claims and extremely opposite refutations [29]. Empirical evidence suggests that this sequence of extreme opposites is very common in molecular genetics [29].

These corollaries consider each factor separately, but these factors often influence each other. For example, investigators working in fields where true effect sizes are perceived to be small may be more likely to perform large studies than investigators working in fields where true effect sizes are perceived to be large. Or prejudice may prevail in a hot scientific field, further undermining the predictive value of its research findings. Highly prejudiced stakeholders may even create a barrier that aborts efforts at obtaining and disseminating opposing results. Conversely, the fact that a field is hot or has strong invested interests may sometimes promote larger studies and improved standards of research, enhancing the predictive value of its research findings. Or massive discovery-oriented testing may result in such a large yield of significant relationships that investigators have enough to report and search further and thus refrain from data dredging and manipulation.

Most Research Findings Are False for Most Research Designs and for Most Fields

In the described framework, a PPV exceeding 50% is quite difficult to get. [Table 4](#) provides the results of simulations using the formulas developed for the influence of power, ratio of true to non-true relationships, and bias, for various types of situations that may be characteristic of specific study designs and settings. A finding from a well-conducted, adequately powered randomized controlled trial starting with a 50% pre-study chance that the intervention is effective is eventually true about 85% of the time. A fairly similar performance is expected of a confirmatory meta-analysis of good-quality randomized trials: potential bias probably increases, but power and pre-test chances are higher compared to a single randomized trial. Conversely, a meta-analytic finding from inconclusive studies where pooling is used to “correct” the low power of single studies, is probably false if $R \leq 1:3$. Research findings from underpowered, early-phase clinical trials would be true about one in four times, or even less frequently if bias is present. Epidemiological studies of an exploratory nature perform even worse, especially when underpowered, but even well-powered epidemiological studies may have only a one in five chance being true, if $R = 1:10$. Finally, in discovery-oriented research with massive testing, where tested relationships exceed true ones 1,000-fold (e.g., 30,000 genes tested, of which 30 may be the true culprits) [30,31], PPV for each claimed relationship is extremely low, even with considerable standardization of laboratory and statistical methods, outcomes, and reporting thereof to minimize bias.

Table 4

PPV of Research Findings for Various Combinations of Power ($1 - \beta$), Ratio of True to Not-True Relationships (R), and Bias (u)

$1 - \beta$	R	u	Practical Example	PPV
0.80	1:1	0.10	Adequately powered RCT with little bias and 1:1 pre-study odds	0.85
0.95	2:1	0.30	Confirmatory meta-analysis of good-quality RCTs	0.85
0.80	1:3	0.40	Meta-analysis of small inconclusive studies	0.41
0.20	1:5	0.20	Underpowered, but well-performed phase I/II RCT	0.23
0.20	1:5	0.80	Underpowered, poorly performed phase I/II RCT	0.17
0.80	1:10	0.30	Adequately powered exploratory epidemiological study	0.20
0.20	1:10	0.30	Underpowered exploratory epidemiological study	0.12
0.20	1:1,000	0.80	Discovery-oriented exploratory research with massive testing	0.0010
0.20	1:1,000	0.20	As in previous example, but with more limited bias (more standardized)	0.0015

The estimated PPVs (positive predictive values) are derived assuming $\alpha = 0.05$ for a single study.
 RCT, randomized controlled trial.
 DOI: 10.1371/journal.pmed.0020124.t004

The estimated PPVs (positive predictive values) are derived assuming a $\alpha = 0.05$ for a single study.
 RCT, randomized controlled trial.

Claimed Research Findings May Often Be Simply Accurate Measures of the Prevailing Bias

As shown, the majority of modern biomedical research is operating in areas with very low pre- and post-study probability for true findings. Let us suppose that in a research field there are no true findings at all to be discovered. History of science teaches us that scientific endeavor has often in the past wasted effort in fields with absolutely no yield of true scientific information, at least based on our current understanding. In such a “null field,” one would ideally expect all observed effect sizes to vary by chance around the null in the absence of bias. The extent that observed findings deviate from what is expected by chance alone would be simply a pure measure of the prevailing bias.

For example, let us suppose that no nutrients or dietary patterns are actually important determinants for the risk of developing a specific tumor. Let us also suppose that the scientific literature has examined 60 nutrients and claims all of them to be related to the risk of developing this tumor with relative risks in the range of 1.2 to 1.4 for the comparison of the upper to lower intake tertiles. Then the claimed effect sizes are simply measuring nothing else but the net bias that has been involved in the generation of this scientific litera-



ture. Claimed effect sizes are in fact the most accurate estimates of the net bias. It even follows that between “null fields,” the fields that claim stronger effects (often with accompanying claims of medical or public health importance) are simply those that have sustained the worst biases.

For fields with very low PPV, the few true relationships would not distort this overall picture much. Even if a few relationships are true, the shape of the distribution of the observed effects would still yield a clear measure of the biases involved in the field. This concept totally reverses the way we view scientific results. Traditionally, investigators have viewed large and highly significant effects with excitement, as signs of important discoveries. Too large and too highly significant effects may actually be more likely to be signs of large bias in most fields of modern research. They should lead investigators to careful critical thinking about what might have gone wrong with their data, analyses, and results.

Of course, investigators working in any field are likely to resist accepting that the whole field in which they have spent their careers is a “null field.” However, other lines of evidence, or advances in technology and experimentation, may lead eventually to the dismantling of a scientific field. Obtaining measures of the net bias in one field may also be useful for obtaining insight into what might be the range of bias operating in other fields where similar analytical methods, technologies, and conflicts may be operating.

How Can We Improve the Situation?

Is it unavoidable that most research findings are false, or can we improve the situation? A major problem is that it is impossible to know with 100% certainty what the truth is in any research question. In this regard, the pure “gold” standard is unattainable. However, there are several approaches to improve the post-study probability.

Better powered evidence, e.g., large studies or low-bias meta-analyses, may help, as it comes closer to the unknown “gold” standard. However, large studies may still have biases and these should be acknowledged and avoided. Moreover, large-scale evidence is impossible to obtain for all of the millions and trillions of research questions posed in current research. Large-scale evidence should be targeted for research questions where the pre-study probability is already considerably high, so that a significant research finding will lead to a post-test probability that would be considered quite definitive. Large-scale evidence is also particularly indicated when it can test major concepts rather than narrow, specific questions. A negative finding can then refute not only a specific proposed claim, but a whole field or considerable portion thereof. Selecting the performance of large-scale studies based on narrow-minded criteria, such as the marketing promotion of a specific drug, is largely wasted research. Moreover, one should be cautious that extremely large studies may be more likely to find a formally statistical significant difference for a trivial effect that is not really meaningfully different from the null [32–34].

Second, most research questions are addressed by many teams, and it is misleading to emphasize the statistically significant findings of any single team. What matters is the totality of the evidence. Diminishing bias through enhanced research standards and curtailing of prejudices may also help. However, this may require a change in scientific mentality that might be difficult to achieve. In some research designs, efforts may also be more successful with upfront registration of studies, e.g., randomized trials [35]. Registration would pose a challenge for hypothesis-generating research. Some kind of registration or networking of data collections or investigators within fields may be more feasible than registration of each and every hypothe-

sis-generating experiment. Regardless, even if we do not see a great deal of progress with registration of studies in other fields, the principles of developing and adhering to a protocol could be more widely borrowed from randomized controlled trials.

Finally, instead of chasing statistical significance, we should improve our understanding of the range of R values—the pre-study odds—where research efforts operate [10]. Before running an experiment, investigators should consider what they believe the chances are that they are testing a true rather than a non-true relationship. Speculated high R values may sometimes then be ascertained. As described above, whenever ethically acceptable, large studies with minimal bias should be performed on research findings that are considered relatively established, to see how often they are indeed confirmed. I suspect several established “classics” will fail the test [36].

Nevertheless, most new discoveries will continue to stem from hypothesis-generating research with low or very low pre-study odds. We should then acknowledge that statistical significance testing in the report of a single study gives only a partial picture, without knowing how much testing has been done outside the report and in the relevant field at large. Despite a large statistical literature for multiple testing corrections [37], usually it is impossible to decipher how much data dredging by the reporting authors or other research teams has preceded a reported research finding. Even if determining this were feasible, this would not inform us about the pre-study odds. Thus, it is unavoidable that one should make approximate assumptions on how many relationships are expected to be true among those probed across the relevant research fields and research designs. The wider field may yield some guidance for estimating this probability for the isolated research project. Experiences from biases detected in other neighboring fields would also be useful to draw upon. Even though these assumptions would be considerably subjective, they would still be very useful in interpreting research claims and putting them in context.

Abbreviation

PPV positive predictive value

Footnotes

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