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2G radiation affects brain development

Altered development in rodent brain cells after 900 MHz radiofrequency exposure

Bodin R, Godin L, Mougin C, Lecomte A, Larrigaldie V, Feat-Vetel J et al. (2025). Altered development in rodent brain cells after 900 MHz radiofrequency exposure. Neurotoxicology, 111: 103312. https://doi.org/10.1016/j.neuro.2025.103312

The ICNIRP has established exposure guidelines for radiofrequency (RF) radiation: 0.08 W/kg for the general population and 0.4 W/kg for occupationally exposed persons. However, it is unclear whether these guidelines provide adequate protection. Previous studies have shown that exposure to comparable levels can cause oxidative stress and altered growth in rodents. These biological effects may occur at these thresholds, especially during sensitive developmental stages. Thus, this study examined the impact of 2G radiofrequency (RF) exposure on the developing brain by using in vivo and in vitro rat models. The ICNIRP limits were adhered to. Most synapses in rats form between postnatal days (PND) 0 and 21, with synaptogenesis (the formation of synapses) peaking during PND 14. In human children, this peak occurs between the ages of 1 and 2. This study focused on proteomic changes and cellular parameters, including proliferation, synaptogenesis, and differentiation of neural cells.

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Study design and implementation:

The male Sprague Dawley rats were examined after being exposed in utero from the eighth day of gestation until the seventh postnatal day at most. The rats were exposed to 900 MHz radiation at either the public exposure limit (30.2 V/m; whole-body SAR 0.08 W/kg) or the occupational exposure limit (67.5 V/m; whole-body SAR 0.4 W/kg). The controls were sham-exposed. A total of 9 control animals (n = 9) and 8 exposed animals (n = 8) were exposed. Examined endpoints included proteomic analyses of the hippocampus and cortex immediately after birth; quantification of brain-derived neurotrophic factor (BDNF), which is essential for maintaining and developing neurons and synapses, as well as memory processes; cell proliferation; the number of synapses and the balance between excitatory and inhibitory synapses; and oxidative stress markers in the hippocampus and cortex on the postnatal days (PND) 8 and 17. (Due to the different time points, the number of animals available for respective endpoint analyses varied from n = 3 to n = 9; editor's note.) These in vivo studies were supplemented by in vitro analyses of neural stem cells. The cells were exposed to 900 MHz radiation and 0.08 W/kg for 3 or 7 days. The scientists evaluated cell differentiation, cell proliferation, and DNA damage.

Results:

Proteomic analysis on postnatal day (PND) 0 revealed different expression levels of 10 proteins in the exposed groups compared to the sham-exposed control group. These changes primarily affected proteins associated with synapse formation and cell growth. A significant decrease in the number of synapses was observed in the hippocampus of young animals exposed on PND 8. The balance between excitatory and inhibitory synapses shifted significantly toward inhibitory synapses in both exposed groups. Both effects leveled off by PND 17. There were no significant differences in the number of synapses in the cortex. However, the balance shifted significantly in the more heavily exposed group on PND 17. In the cortex, but not the hippocampus, there were significantly fewer proliferating cells on PND 8. A significant decrease in BDNF levels was also observed on PND 17. Examination of oxidative stress markers showed no significant changes. In vitro, there were significantly more apoptotic and proliferating cells, as well as DNA double-strand breaks. In addition, there was a statistically significant change in the differentiation pattern of neural stem cells.

Conclusions:

The study data suggest that exposure within regulatory limits for the general population may adversely impact brain development. These changes include reduced cell proliferation, BDNF levels, and synapse formation in vivo, as well as changes in neural stem cell differentiation in vitro. The authors hypothesize that the increase in inhibitory synapses may result from increased neuronal excitability of the organism. Changes in this balance have been linked to developmental disorders of the nervous system, such as autism spectrum disorder. In summary, the scientists' hypothesis that developing organisms are susceptible to RF radiation is confirmed. They recommend exercising caution when using mobile devices, especially around pregnant women and young children.

Editor's note:

The study design is impressive thanks to its combination of in vivo and in vitro experiments and a multimodal approach that includes proteomics, immunohistochemistry, and functional analyses. The study's significance is also enhanced by its use of low field strengths that fall within regulatory limits for the general population. However, there are areas for improvement. For example, the total number of experimental animals is small, and the number varies depending on the endpoints investigated. While the chosen study times are well-selected in terms of neural development, an additional study including adult animals would have been desirable to analyze functional consequences that may persist into adulthood. As with any animal model, the question of transferability to humans arises. Nevertheless, the data suggest that regulatory limits may not be safe at all stages of development. (RH)

Bluetooth affects human neural stem cells

Radiofrequency regulates the BET-mediated pathways in radial glia differentiation in human cortical development

Cakir B, Tanaka Y, Choe MS, Kiral FR, Kim J, Micali N et al. (2025). Radiofrequency regulates the BET-mediated pathways in radial glia differentiation in human cortical development. Cell Reports, 44(10): 116238. https://doi.org/10.1016/j.celrep.2025.116238

The development of the human brain is controlled by a complex genetic program. Several genes have been identified as being associated with neurological developmental disorders, including autism spectrum disorder (ASD), intellectual disability, learning disorders, and neuropsychiatric disorders. Researchers have recently proven that non-genetic factors, such as prenatal stress, infections, and nutrition, also influence brain development. Another environmental factor suspected of influencing brain development is radiofrequency (RF) radiation. The working group uses cortical organoids derived from human embryonic stem cells to study the impact of 2.4 GHz RF radiation on brain development. These cortical organoids exhibit complex 3D architectures and cellular diversity. Their developmental pattern reflects the stages and characteristics of human corticogenesis. This includes the formation of apical radial glial (aRG) cells, which differentiate into excitatory neurons.

Study design and implementation:

Cortical organoids (hCOs) were grown from human embryonic stem cells to serve as 3D models of the developing human brain in its earliest stages. Beginning on day 10 of the patterning stage, the cells were exposed to RF radiation for 12 hours per day or continuously. The field source was a Bluetooth module operating at 2.4 GHz (Bluetooth V2.0 + EDR), which corresponds to the transmission power of low-power Bluetooth devices of 4 dBm (2.4 mW). The maximum measured field strength was 2.5 mW/m^2. Control hCOs were cultivated in separate incubators, and ambient RF radiation levels were measured. The authors examined a variety of endpoints using various methods, including immunohistochemistry, single-cell RNA sequencing (scRNA-seq), epigenetic methods (single-cell ATAC sequencing to detect chromatin accessibility), electrophysiological recordings (whole-cell patch clamp), calcium imaging, and quantitative PCR (reverse transcription quantitative polymerase chain reaction / RT-qPCR).

Results:

Eight days after exposure began, the scientists observed a significant reduction in the size of the hCOs, as well as an irregular surface. Examination of differentiation markers revealed that Bluetooth exposure favors the self-renewal of apical radial glial (aRG) cells (symmetrical cell division). This occurs at the expense of other cell populations, such as deep layer neurons. These findings suggest impairment in the early development of the cerebral cortex. These observations were confirmed using neural stem cells from rhesus monkeys in vitro. Statistically significant transcriptomic changes were observed in hCOs exposed to Bluetooth radiation. Genes associated with neurodevelopment and neuronal projection were among those affected, suggesting that RF radiation may influence the early development of the human brain. Genes associated with autism spectrum disorder (ASD) (including FOXG1, AUTS2, and CPEB4) were also overexpressed in the exposed hCOs. This finding was confirmed using three methods: scRNA-seq, RT-qPCR, and immunohistochemistry. Retrotransposons associated with ASD increased statistically significantly after exposure. In summary, these results suggest that RF radiation from mobile devices or Bluetooth can increase the risk of ASD by increasing ASD-risk genes and retrotransposons associated with ASD. However, these effects were reduced or almost completely prevented by inserting a physical barrier in the form of aluminum foil or Faraday shielding. RF radiation also significantly impacted the organoids morphologically and functionally. A higher synapse density accompanied by increased neuronal activity was documented. Pharmacological inhibition of bromodomain and extraterminal (BET) proteins restored a wide range of Bluetooth-induced damage, including morphological changes, hCO size, ASD-associated genes, differentiation markers, and proliferation dynamics. This "rescue" of the phenotype indicates that the RF effect is mediated by epigenetic signaling pathways controlled by histone acetylation. Further analyses revealed that RF radiation altered chromatin accessibility, presumably through the transcription regulator NRF1.

Conclusions:

This study shows that Bluetooth radiation can harm cortical organoids (hCOs), which are excellent models for studying the effects of environmental factors, such as viral infections, ethanol, nicotine, and cannabis, on brain development. Damaging effects of RF radiation include altered differentiation of neural stem cells and characteristics associated with ASD. These effects could be mitigated by physical barriers or pharmacological treatment. These effects are consistent with observations in animal models in which intrauterine RF exposure caused cognitive and behavioral abnormalities such as hyperactivity and memory impairment (Aldad et al., 2012). According to the authors, hCO models bridge the gap between animal models and human neurodevelopment.

Editor's note:

The broad scope of methodologies and endpoints examined in this study enhances its significance. This is particularly evident for endpoints confirmed by various molecular biological methods, such as RT-qPCR, scRNA-seq, and immunohistochemistry, as well as for endpoints observed across different species. The significant improvement in phenotype following pharmacological treatment strengthens the study's findings and provides insight into possible mechanistic relationships. The authors clearly state the study's limitations, including the absence of physical barriers, such as a skull, and the limitations of organoid models compared to in vivo situations. Nevertheless, the meaningful hCO model demonstrates that low field strengths (2.5 mW/m^2) impair early neural development. (RH)

Aldad TS, Gan G, Gao XB, Taylor HS (2012). Fetal radiofrequency radiation exposure from 800-1900 Mhz-rated cellular telephones affects neurodevelopment and behavior in mice. Scientific Reports, 2: 312. https://doi.org/10.1038/srep00312

Wi-Fi and Alzheimer's?

Review of the evidence on the influence of Wi-Fi 2.4 GHz radiation on oxidative stress and its possible relationship with Alzheimer's disease

Laván D, Lluncor A, Moyano J, Ubillus J, Peña M, Paredes M et al. (2025). Review of the evidence on the influence of Wi-Fi 2.4 GHz radiation on oxidative stress and its possible relationship with Alzheimer's disease. Frontiers in Neurology, 16: 1616435. https://doi.org/10.3389/fneur.2025.1616435

A new study by a Peruvian research group examined the effects of 2.4 GHz Wi-Fi radiation, with a particular focus on its role in oxidative stress and its potential link to Alzheimer's disease (AD). Alzheimer's disease is multifactorial, developing as a result of the complex interaction of genetic, environmental, and lifestyle factors.

Study design and implementation:

The authors conducted a literature search in accordance with PRISMA guidelines. The review addressed 3 questions:

> 1) How does exposure to 2.4 GHz electromagnetic fields affect gene expression related to oxidative stress response, genomic stability, metabolism, and cellular regulation?

> 2) How does the regulation of oxidative stress and protein homeostasis influence the progression of neurodegeneration in Alzheimer's disease?

> 3) Can genes associated with Alzheimer's disease interact functionally with genes involved in DNA repair, metabolism, and cellular regulation?

Results:

A total of 49 studies were used to synthesize and map genetic relationships. Several genes, including APOE and GSK3B, have been directly linked to Alzheimer's disease (AD). Beyond their role in AD pathogenesis, genes such as MAGED4, HSPB8, HSPB6, HSF1, HSPA4, HSPB1, and MAPKAPK5 play a crucial role in the cellular response to oxidative stress, which is a key factor in the pathogenesis of various diseases. These genes contribute to cell protection by regulating protein homeostasis, stabilizing misfolded proteins and preventing apoptosis induced by reactive oxygen species (ROS). This highlights their importance in preventing diseases related to oxidative stress. Furthermore, exposure to electromagnetic fields (EMFs) at a frequency of 2.4 GHz has been shown to alter the expression of several genes associated with DNA replication, cellular homeostasis, and metabolism. These include POLD4 and FEN1, both of which are essential for DNA replication and repair and whose altered expression could compromise genomic stability. In addition, EXOG, which is involved in mitochondrial DNA repair, exhibits changes that suggest a potential disruption to mitochondrial function. Taken together, these findings suggest that exposure to Wi-Fi radiation can significantly alter genomic stability, metabolism, and cellular regulation. Genes associated with Alzheimer's disease, including ABCA7, PICALM, CLU, TREM2, APOE, SORL1, GSK3B, and PRNP, intersect with HSP27, HSPB1, HSP70, and HSPA4. The latter genes are all part of the "heat shock protein (HSP)" family and regulate oxidative stress. The genes involved in regulating Alzheimer's disease also intersect with POLD4, FEN1, and EXOG, among others, which showed reduced expression after exposure to 2.4 GHz Wi-Fi radiation.

Conclusions:

Recent research on Alzheimer's disease (AD) has identified several genes that contribute to its pathogenesis. These genes are located in a highly dynamic biological environment in which oxidative stress plays a central role in the cell damage associated with neurodegeneration. The activation of heat shock proteins (HSPs), such as HSPB1 and HSPA4, is of particular interest in Alzheimer's disease because these proteins protect cells from oxidative stress. Several studies suggest that exposure to Wi-Fi radiation can alter the expression of genes involved in DNA replication and repair processes. This indicates that exposure to this frequency of RF radiation could compromise genomic stability and mitochondrial function. While the authors could not find direct evidence of a link between Wi-Fi exposure and AD, the indirect link via oxidative stress is concerning and warrants further investigation. (AT)

High voltage and cognition

Environmental effect of high-voltage towers on the cerebellum and cognitive impairments in the monkey

Aliyari H, Sahraei H, Menhaj MB, Kazemi M, Vahidi B, Hosseinian SH (2024). Environmental effect of high-voltage towers on the cerebellum and cognitive impairments in the monkey. Basic and Clinical Neuroscience, 15(2): 185–198. https://doi.org/10.32598/bcn.2021.1340.5

High-voltage power lines create a harmful environment for people who live or work near them. Previous research and epidemiological studies have shown that the extremely low-frequency (ELF) electromagnetic fields around these lines significantly harm the brains and cognitive functions of animals and humans. To study the direct physiological effects of exposure to high voltage (HV), researchers exposed a male rhesus monkey to an artificially generated voltage of 3 kV/m in a laboratory setting.

Study design and implementation:

In this experiment, 2 adult rhesus monkeys were used: 1 as the test subject and 1 as the control. The test monkey was exposed to a simulated high-voltage field of 3 kV/m for 4 hours per day for 1 month. This corresponds to the field strength at a distance of approximately 20 meters from a 275 kV transmission line. Behavioral tests were performed before and after exposure using a specially developed and custom made device. Two tests were administered: a visual learning test and a visual memory test. Blood levels of adrenocorticotropic hormone (ACTH) were analyzed before and after exposure. Expression of the glucocorticoid receptor (GR) gene was also examined. Changes in the anatomy of the cerebellum were examined using magnetic resonance imaging (MRI). All tests were performed before and after the study period and compared with the results for the control monkey.

Results:

The results of the behavioral study showed that the exposed monkey performed worse on visual learning tests in an HV field than before the test. Meanwhile, there were no significant changes in the control group. The exposed monkey demonstrated reduced visual memory performance in the HV field compared to its previous state. ACTH levels increased in the exposed monkey, while the control group showed no significant change. Notably, these hormone levels decreased and returned to baseline within 1 month after the recovery period. The expression of the glucocorticoid receptor gene decreased significantly in the exposed monkey, but it also returned to baseline during the 1-month recovery period. Magnetic resonance imaging (MRI) scans of the cerebellum revealed no significant changes in volume. However, bleeding was detected on the left side of the exposed monkey's cerebellum, but only after the 1-month exposure period.

Conclusions:

The study results show that the monkey exposed to HV fields exhibited significant cognitive and behavioral changes. Before the test, the monkey was intelligent, alert, lively, and energetic. However, significant changes occurred after the testing period. It became inattentive, lethargic, inactive, listless, and depressed, and lost approximately 1 kg of weight. The monkey showed elevated ACTH levels after the test. ACTH regulates cortisol, a hormone that plays an important role in cognitive functions, such as memory and learning, in the central nervous system. Increased ACTH levels reduce cortisol secretion, and abnormal cortisol secretion can lead to mental disorders. Conversely, cortisol plays an essential role in the expression of the glucocorticoid receptor (GR) gene. GRs and N-methyl-D-aspartate (NMDA) receptors, which play an important role in memory and learning processes, are distributed throughout the central nervous system. The highest concentrations of these receptor genes are found in the hippocampus, prefrontal cortex, amygdala, and cerebellum. According to the results of this study, high-voltage transmission lines pose a serious threat to the cognitive abilities of residents in the surrounding area, necessitating further research.

Editor's note:

This was a well-conducted semifield study because it used a realistic high-voltage simulation. The behavioral findings were physiologically confirmed and plausibly explained. However, using only 1 test animal instead of 10 or 30 yielded weaker statistical results. However, when monkeys are used as realistic test animals, which is expensive and raises ethical questions, even a single test animal (with a control) is significant. (AT)

High-voltage transmission lines and honeybees

Weak anthropogenic electric fields affect honeybee foraging

Mallinson VJ, Woodburn FA, O'Reilly LJ (2025). Weak anthropogenic electric fields affect honeybee foraging. iScience, 28(6): 112550. https://doi.org/10.1016/j.isci.2025.112550

Bees tend to acquire a positive electrostatic charge while flying. In contrast, flowers are usually negatively charged due to their electrical connection to the ground and the generally positive atmospheric potential gradient. These charge dynamics create an electrical environment that provides foraging signals and facilitates pollen transfer. Animal pollination is responsible for approximately 75 % of food crops and 35 % of global food production, which is estimated to have an annual economic value of between 235 billion and 577 billion US dollars. The following study investigated how visits by honeybees to flowers are influenced by small electric fields generated on site, making this a semifield study.

Study design and implementation:

Field experiments were conducted in Bristol, UK, to study the impact of weak anthropogenic electric fields on pollinator behavior. Researchers manipulated the electrical environment of flowers by exposing them to electric fields that mimicked the alternating and direct current voltages of transmission lines. Then, they observed how honeybees landed. The electric fields were generated near 2 catnip flowers (1.2 meters apart) using a portable, battery-powered function generator. The researchers simultaneously observed the foraging behavior of the honeybees at 2 flowers, focusing on how they landed on the exposed flower and the (sham-exposed) control flower.

Results:

In an experiment in which electric fields were altered, the average number of bees that landed on flowers exposed to alternating or direct current fields decreased by 70.8 % and 52.6 %, respectively, compared to flowers in the control group. In contrast, no difference in landing numbers was observed with negative DC treatment. Regarding temporal variation in observed behavior, the number of bees that landed on flowers exposed to AC treatment during the first 10 minutes was 5.9 % of what was observed for the paired control flower. This figure increased to 58.8 % in the subsequent 10 minutes. This effect was less pronounced for DC fields. The AC field used in this experiment corresponds to ambient electric fields at a distance of about 100 meters from a 275 kV high-voltage transmission line.

Conclusions:

This study shows that weak, locally limited anthropogenic electric fields with strengths comparable to those measured 50 to 150 meters away from high-voltage transmission lines alter the foraging behavior of honeybees. Therefore, it is conceivable that such fields could influence the foraging behavior of pollinators and their interactions with flowers. However, the experimental electric fields used in this study were constant in strength and dynamics, representing a stable stimulus. In contrast, anthropogenic electric fields often fluctuate considerably in strength due to variable power consumption throughout the day. In addition, these results focus on short-term effects. Further research is needed to understand how these responses can be applied to different pollinators, plant species, and broader ecological networks. (AT)

900 MHz exposure damages bone cells in vitro

Activating transcription factor 4 regulation of radiofrequency radiation-induced ferroptosis in osteoblasts

Wang H, Zou W, Ding C, Cao Y (2025). Activating Transcription Factor 4 regulation of radiofrequency radiation-induced ferroptosis in osteoblasts. Electromagnetic Biology and Medicine, 44(4): 551–565. https://doi.org/10.1080/15368378.2025.2547799

A growing body of research suggests a link between radiofrequency (RF) radiation and biological responses, including oxidative stress, DNA damage, and apoptosis. Osteoblasts, which are essential for bone formation, appear to be susceptible to damage from RF radiation. This effect may be mediated by ferroptosis, a process regulated by 3 signaling pathways: glutathione/glutathione peroxidase 4 (GSH/GPX4), iron metabolism, and lipid metabolism (peroxidation of polyunsaturated fatty acids). Using bioinformatic analyses, the research group identified AtfPX4 as a central gene at the intersection of RF radiation and ferroptosis. The researchers then examined the relationship between 900 MHz exposure, ferroptosis, and ATF4 (activating transcription factor 4) in osteoblasts in vitro.

Study design and implementation:

First, a database analysis was performed to identify candidates that were potentially altered by radiofrequency electromagnetic fields (RF-EMF) associated with ferroptosis. The MC3T3-E1 murine osteoblast cell line was exposed to 900 MHz radiation at 3 different intensities (50, 150, and 450 μW/cm²) for 4 hours per day over 5 days. These values were based on the ICNIRP's recommended limit for the general population (200 μW/cm²). The controls were sham-exposed. The endpoints examined were cell viability, lipid peroxidation (MDA), glutathione (GSH), reactive oxygen species (ROS), iron homeostasis, mitochondrial function, and ferroptosis-associated gene expression. The researchers analyzed the role of ATF4 using knockdown (siRNA) and overexpression, as well as pharmacological ferroptosis inhibitors.

Results:

Experiments showed that RF exposure to an intensity of 150 μW/cm² at 900 MHz significantly reduced the viability of the osteoblast cell line. There was also a significant increase in lipid peroxidation, intracellular iron content, and mitochondrial damage. These changes were accompanied by a disrupted antioxidant protection mechanism, including reduced levels of glutathione (GSH) and glutathione peroxidase 4 (GPX4). These results suggest that RF exposure induces ferroptosis. Pharmacological inhibition of ferroptosis restored the aforementioned endpoints to levels observed in the sham-exposed controls. Knockdown of the Atf4 hub gene increased ferroptosis markers, albeit inefficiently (RT-qPCR revealed a reduction of approximately 25 %; editor's note). In contrast, overexpression provided significant protection to the exposed cells against ferroptosis.

Conclusions:

The scientists concluded that exposure to continuous wave RF radiation at 900 MHz (without pulse modulation) can induce ferroptosis in bone-forming cells in vitro, even at levels below of those recommended by the International Commission on Non-Ionizing Radiation Protection (ICNIRP). They consider the non-linear dose–response relationship to be a significant finding and assume a "window effect" (wherein an effect only occurs within a certain range). This highlights the complexity of biological interactions with RF radiation. The authors argue that ATF4's protective role suggests a mechanistic link between RF radiation and bone diseases, such as osteoporosis.

Editor's note:

The study's significance is underscored by consistent results obtained through various molecular biological methods, which include "rescuing" ferroptosis with inhibitors and overproducing ATF4. However, the data were generated only in vitro and with a single cell line. The authors acknowledge these limitations and suggest verifying the data in vivo by investigating downstream ATF4 molecules, among other steps. Nevertheless, evidence suggests that RF-EMF can damage bones in vivo (Bektas et al., 2023; see our review in ElektrosmogReport 3-2023). (RH)

Bektas H, Nalbant A, Akdag MB, Demir C, Kavak S, Dasdag S (2023). Adverse effects of 900, 1800 and 2100 MHz radiofrequency radiation emitted from mobile phones on bone and skeletal muscle. Electromagnetic Biology and Medicine, 42(1): 12–20. https://doi.org/10.1080/15368378.2023.2179065

RF radiation changes metabolism in testicular cells

Differential metabolic responses of mouse Leydig and spermatogonia cells to radiofrequency electromagnetic field exposure

Miao X, Lin Y, Guo J, Lin J, Gao P, Zhang W et al. (2025). Differential metabolic responses of mouse Leydig and spermatogonia cells to radiofrequency electromagnetic field exposure. Frontiers in Public Health, 13: 1623701. https://doi.org/10.3389/fpubh.2025.1623701

Several studies suggest that exposure to radiofrequency electromagnetic fields (RF-EMF) can adversely affect male fertility. Potential consequences include reduced sperm quality and testosterone production, as well as genotoxic effects. Metabolomics, the study of metabolites, can identify and quantify disrupted metabolic pathways, proteins, and genes, thereby providing insight into the underlying mechanisms. This study uses metabolomics analysis to investigate the effects of RF radiation on the metabolism of mouse Leydig cells (TM3 cell line) and mouse spermatogonia (GC-1 cell line).

Study design and implementation:

The TM3 and GC-1 cell lines were exposed to an unmodulated frequency of 1950 MHz and a specific absorption rate (SAR) of 3 W/kg for 24 hours. The exposure occurred either continuously or in 1-hour intervals of 1 hour of exposure followed by 1 hour without exposure, while the controls were sham-exposed. The temperature was kept constant at 37°C. After exposure, metabolites were extracted and separated by liquid chromatography and analyzed by mass spectrometry. Differential metabolites were examined using the KEGG database to determine enriched metabolic pathways. In addition, glutathione concentration was determined by ELISA.

Results:

The RF effect depended on the level of exposure and the type of cell. Statistically significant changes in metabolites in continuously exposed TM3 cells indicated substantial disturbances in amino acid metabolism and the citric acid cycle. In contrast, changes in intermittently exposed TM3 cells indicated impairment in energy metabolism and nucleotide signaling pathways. KEGG analysis revealed significant accumulations of differential metabolites in ABC transporters, other membrane transporters, and glutathione metabolism during continuous exposure. Consistent with this finding, significantly lower levels of glutathione were observed in continuously exposed cells. Disturbances in purine metabolism and lysine degradation were observed in both exposure types. The key molecule adenosine diphosphate (ADP) was among those affected. In the spermatogonia cell line (GC-1), the RF effect was significantly lower. Changes in lipid metabolism were only detected with on/off exposure, which may indicate an altered membrane integrity.

Conclusions:

The study revealed significant metabolic changes in the TM3 Leydig cell line after exposure to 1950 MHz radiation. Reduced GSH levels confirm a change in glutathione metabolic components during continuous exposure. The authors suggest that the accumulation of differential metabolites from various metabolic transport processes may indicate an impaired ability to detoxify and excrete toxic substances. Since spermatogenesis is an energy-intensive process, disturbances in energy metabolism could negatively impact sperm production. The authors interpret the different effects of exposure on specific cell types as being closely related to the cells' functional roles and metabolic requirements.

Editor's note:

The study uses modern chromatographic and mass spectrometric methods, multivariate analyses, and supplementary validation by ELISA validation. The authors acknowledge one limitation: the use of a purely in vitro system. Using cell lines is also a negative factor. Although the influence of temperature increases can practically be ruled out due to temperature monitoring, the SAR value of 3 W/kg is rather high. Nevertheless, this study provides valuable information for future in vivo and dose–response studies because it offers reliable evidence of how reproductive cells metabolize RF radiation. (RH)

Review of genotoxicity

A scoping review and evidence map of radiofrequency field exposure and genotoxicity: assessing in vivo, in vitro, and epidemiological data

Weller SG, McCredden JE, Leach VA, Chu C, Lam AK (2025). A scoping review and evidence map of radiofrequency field exposure and genotoxicity: assessing *in vivo, in vitro*, and epidemiological data. Frontiers in Public Health, 13: 1613353. https://doi.org/10.3389/fpubh.2025.1613353

The world is facing a cancer pandemic, with many types of cancer increasing exponentially. While an ageing population undoubtedly contributes to this trend, it cannot account for all of the observed increase in certain types of cancer. This suggests that environmental and/or lifestyle factors also play a role. One possible factor is the global increase in background levels of radiofrequency electromagnetic fields (RF-EMF), which are generated by humans and have been shown to correlate with rising cancer incidence rates in recent decades. Genetic changes are a known trigger for the development of cancer. However, studies investigating the genotoxic effects of RF-EMF exposure have employed various parameters and produced inconsistent results. A systematic mapping of existing research is necessary to identify emerging patterns and inform future research and policy. The aim of this review was to determine whether exposure to RF-EMF can damage DNA and thus potentially contribute to the global increase in cancer incidence.

Study design and implementation:

The protocol for creating the evidence map and associated data synthesis adheres to the PRISMA guidelines for scoping reviews (PRISMA-ScR). Studies underwent a quality assessment based on specific criteria. These criteria were recommended by Vijayalaxmi and Prihoda (Prihoda, 2019). Data were synthesized according to the following criteria: study type, organism type, exposure level and duration, biological markers (genotoxicity, cellular stress, and apoptosis), radiofrequency electromagnetic field (RF-EMF) signal characteristics, and funding source. Quantitative aggregation was used to create tables, graphs, and heat maps.

Results:

Of the 3,430 candidate studies, 517 were identified as relevant. The relevant studies were classified into 3 categories: in vitro (53 %), in vivo (37 %), and epidemiological (10 %) studies. Overall, the balance of evidence for DNA damage was 59 % "effect" versus 41 % "no effect."

The results of the in vitro studies leaned slightly toward "no effect" (55 %), whereas most of the in vivo (75 %) and epidemiological (75 %) studies reported statistically significant DNA damage. In higher-quality studies, the balance shifted slightly toward "no effect" (52 %) versus "effect" (48 %). The simplified environment of in vitro studies may lead to an underestimation of RF-induced genotoxic effects. To fully understand the biological effects of RF exposure, in vitro studies must be interpreted with caution and validated by in vivo research. Human studies showed an almost even split (51 % versus 49 %) in the evidence. In vivo and epidemiological studies consistently reported statistically significant DNA damage, while in vitro studies tended to show negative results. Similar trends were observed in rats (75 % "effect" studies) and other mammals (73 %). All studies on plants, worms, birds, and amphibians reported statistically significant DNA damage. Similarly, 71 % of insect studies identified statistically significant DNA damage. Approximately 80–100 % of non-mammal studies showed statistically significant effects, particularly DNA base damage and oxidative stress. Spindle disruption, a potential mechanism of DNA damage, was found in 100 % of mammalian and plant studies. The highest proportion of genotoxic effects (81 % of 21 studies) was reported in studies examining extremely low exposure levels (< 0.001 W/kg). Furthermore, the proportion of statistically significant DNA damage findings decreased with increasing intensity. However, at extremely high intensities (> 10 W/kg) exceeding ICNIRP limits, the proportion of statistically significant DNA damage effects increased again (58 % of 59 studies). These results suggest that DNA damage does not follow a linear dose–response pattern but rather a U-shaped curve and that non-thermal mechanisms likely play a significant role in RF-induced DNA damage.

In addition, the effects varied in a U-shaped pattern depending on the duration of the study's exposure. The highest proportion of effects occurred in long-term studies (> 3 months), followed by medium-term studies (1–3 months). The fewest effects occurred in short-term studies (1–24 hours), while a higher proportion of effects occurred in acute studies (< 1 hour). This complex interaction confirms Lai and Levitt's (2022) earlier evidence of non-linear response patterns in terms of both intensity and duration. Linear models are not suitable for describing the results in this area. These results challenge overly simplistic conclusions from earlier reviews that found no effects. Elevated concentrations of reactive oxygen species have been linked to DNA damage. In response to cellular stress, cells employ various protective mechanisms, including the upregulation of DNA repair genes, heat shock proteins, and enzymes that mitigate oxidative stress. Gene expression is a sequential, time-consuming process that begins with the activation of signaling pathways and the binding of transcription factors. This is followed by mRNA processing and protein synthesis. These adaptive mechanisms likely explain the observed U-shaped curves for EMF exposure duration. Real-world RF exposure is usually chronic and variable, raising concerns about its cumulative effects. Although repair mechanisms appear to resolve much of the damage in the short term, prolonged or repeated exposure may overwhelm these defense mechanisms, resulting in permanent genomic changes.

A comprehensive analysis examining potential biases, such as authors' affiliations, funding sources, and journals used for publication, revealed that these factors strongly influence reported outcomes. Studies funded by interest groups (e.g. industry, government telecommunications regulators, and the U.S. Air Force) were more likely to conclude that there was "no DNA damage" than studies funded by governments, institutions, or private/public sources.

Industry- or military-funded research primarily consisted of in vitro studies: 30 % of the 142 in vitro studies reported statistically significant results compared to 26 % of the 31 in vivo studies. Quality filtering had no significant impact on these results (25 % of 56 in vitro studies and 25 % of 12 in vivo studies). In contrast, 63 % of the 465 experimental studies were classified as independent research, including 130 in vitro studies (44.5 %) and 162 in vivo studies (55.5 %). Overall, study quality had little influence on independent research. Seventy-four percent of all independent studies (n = 292) showed statistically significant DNA damage, compared to 73 % of the 62 higher-quality studies.

Independently funded research conducted a much broader range of experiments and primarily reported statistically significant DNA damage. These findings suggest that the more conservative results of higher-quality studies, compared to all studies overall, are likely due to the high proportion of industry-funded researchers conducting higher-quality studies, the larger proportion of in vitro studies with short exposure times, and the exclusion of epidemiological studies.

Conclusions:

All of this demonstrates how methodological preferences and potential conflicts of interest can bias the results of studies and reviews, reducing the robustness of the overall evidence base and creating uncertainty. To confirm the U-shaped dose–response relationship and better understand its implications, future research should use standardized protocols, incorporate multiple assays, and examine a broader range of exposure intensities and time periods. These methods will enhance the consistency and reproducibility of results. The evidence map indicates that medium- to long-term exposure to radiofrequency electromagnetic fields (RF-EMF), especially at low intensities, may lead to genetic damage through non-thermal mechanisms, such as increased free radical production and oxidative stress. Genetic damage can have far-reaching, long-term, and potentially irreversible consequences. To prevent potential genotoxic risks, these findings support the introduction of precautionary measures in addition to existing thermal exposure guidelines.

Editor's note:

This Australian scoping review is characterized by its excellent methodology and thorough consideration of the studies' findings. The data is presented in an excellent graphical format, and the shortcomings of this controversial field of research are highlighted as well. (AT)

Prihoda TJ (2019). Comprehensive review of quality of publications and meta-analysis of genetic damage in mammalian cells exposed to non-ionizing radiofrequency fields. Radiation Research, 191(1): 20–30. https://doi.org/10.1667/RR15117.1

Lai H, Levitt BB (2022). The roles of intensity, exposure duration, and modulation on the biological effects of radiofrequency radiation and exposure guidelines. Electromagnetic Biology and Medicine, 41(2): 230–255. https://doi.org/10.1080/15368378.2022.2065683

Authors biased?

The WHO-commissioned systematic reviews on health effects of radiofrequency radiation provide no assurance of safety

Melnick RL, Moskowitz JM, Héroux P, Mallery-Blythe E, McCredden JE, Herbert M, Hardell L, Philips A, Belpoggi F, Frank JW, Scarato T (2025). The WHO-commissioned systematic reviews on health effects of radiofrequency radiation provide no assurance of safety. Environmental Health, 24: 70. https://doi.org/10.1186/s12940-025-01220-4

As part of its Electromagnetic Field (EMF) Project, the World Health Organization (WHO) commissioned 12 systematic reviews and meta-analyses to clarify the health effects of radiofrequency radiation (0–300 GHz). Modern wireless communication technologies, such as mobile phones, Wi-Fi routers, and base stations, operate within this frequency range. The reviews cover topics including cancer risks in humans and laboratory animals, fertility issues, cognitive deficits, electromagnetic hypersensitivity (EHS), and oxidative stress. The reviews were published between 2023 and 2025. This paper by Melnick et al. critically evaluates the methodological quality, selection criteria, risk and evidence assessment, and interpretation of the results by the WHO-commissioned teams of authors.

Study design and implementation:

Instead of conducting a primary study, Melnick et al. performed a systematic and methodological critique of twelve systematic reviews and meta-analyses that were commissioned by the WHO. The authors analyzed 6 areas: (1) possible bias on the part of the review authors, (2) inclusion and exclusion criteria for primary studies, (3) adequacy of the design of the included studies, (4) approach used in the reviews, (5) meta-analysis, and (6) risk and evidence assessment.

Results:

First, Melnick et al. criticize the overrepresentation of ICNIRP members among the authors of the WHO-commissioned studies. Each working group included at least one current or former ICNIRP member, and some included several. Some individuals appeared in different review teams multiple times. Because the ICNIRP has historically believed that thermal effects are the only health consequences of radiofrequency electromagnetic fields, Melnick et al. argue that the authors are at high risk for systematic bias ("groupthink bias"). Former ICNIRP member James C. Lin (Lin, 2021) came to the same conclusion. In addition, the connections between ICNIRP and industry, particularly in the telecommunications and military sectors, are concerning because these sectors have a major economic interest in exposure limits. Melnick et al. also criticize the glaring deficiencies in the inclusion and exclusion criteria of primary studies in various reviews. These deficiencies resulted in the exclusion of relevant, well-conducted studies while studies of insufficient quality received disproportionate consideration. This is particularly evident in the example of RF effects on oxidative stress markers. Of the 897 original articles that Meyer et al. deemed suitable for their review, only 52 were included in their meta-analysis. This corresponds to less than 6 %. Meyer et al. excluded 360 primary studies because the WHO reviewers deemed the biomarkers used in these studies unsuitable for evaluating oxidative stress. However, adequate sham-exposed controls invalidate this argument, demonstrating that the examined biomarkers reliably indicate oxidative stress. In addition, primary studies with low exposure levels (despite their relevance; editor's note) were excluded due to selected field strength thresholds (e.g. E > 1 V/m). Meyer et al. failed to mention the exclusion of protective antioxidant enzyme activity when describing the inclusion and exclusion criteria. These enzymes have long been used as biomarkers for oxidative stress in many studies. Therefore, the Meyer et al. review does not reflect the current state of research. Not only were relevant studies on oxidative stress markers omitted, but also methodologically inadequate studies were given significant weight. For example, the effects of RF radiation on cancer in humans relied heavily on the results of the Danish cohort study despite its serious methodological shortcomings and classification as inconclusive by the International Agency for Research on Cancer (IARC). A second review of "less well-researched" human cancers included only primary studies from 1988 to 2019, excluding the UK Biobank cohort study. With over 400,000 participants, the UK Biobank cohort study is one of the largest and best-conducted studies in this field. The study found statistically significant increases in the incidence of various types of cancer, including overall cancer incidence, in connection with mobile phone use. Another example of inadequacies is the evaluation of electromagnetic hypersensitivity (EHS) (Bosch-Capblanch et al., 2024). In assessing sleep disorders, not a single study involving participants with EHS was included. Furthermore, only power density and frequency were considered relevant exposure criteria. Variables closely related to EHS, such as magnetic fields, extremely low frequency (ELF) pulse modulation, and data transmission, were ignored. Melnick et al. identified weaknesses in nearly all methodologies of the meta-analyses, except for the review by Mevissen et al. (Mevissen et al., 2025). However, Mevissen et al. did not perform a meta-analysis due to the heterogeneity of the primary studies. Despite high heterogeneity, meta-analyses were performed on other topics. In addition, a series of meta-analyses were conducted with too few primary studies (often fewer than 5 and sometimes as few as 2) and with studies that had different designs (e.g. case-control and cohort) and/or endpoints. The resulting data suggest a precision of results that does not exist due to the aforementioned methodological shortcomings. Melnick et al. raise another point of criticism regarding the use of the risk-of-bias tool and the GRADE system. They found that downgrading the strength of evidence was inconsistent. In addition, documentation of assessment decisions and traceability of GRADE steps were often insufficient. Based on their data, 8 of the 12 WHO-funded studies concluded that RF-EMF exposure probably does not cause adverse health effects. However, these conclusions are based on data that the authors classified as having low or very low strength of evidence. This is a logical error because the evidence that RF-EMF exposure is safe is also low or very low.

Conclusions:

Melnick et al. identified recurring and systematic methodological flaws in nearly all WHO reviews. These flaws include overly restrictive inclusion criteria, flawed exposure definitions, inappropriate meta-analyses based on a small number of studies, selective weighting of results, inconsistent application of assessment tools, and potential conflicts of interest among authors. Rather than providing a thorough evaluation of the scientific evidence on the health risks of RF-EMF, these reviews create a false sense of security that endangers the health of the general population. In addition, consistent evidence of harmful effects has been found in reviews of cancer and reproduction in animal studies. Melnick's working group argues that the FCC and ICNIRP exposure limits are outdated and based on invalid assumptions (thermal dogma). New, scientifically sound guidelines that adequately protect human health and the environment are urgently needed. Due to their shortcomings, most reviews and meta-analyses commissioned by the WHO do not provide sufficient evidence that mobile phones and other mobile devices are safe.

Editor's note:

This unusually harsh criticism is based on documented methodological processes. The criticisms raised are all valid and understandable. In light of the methodological shortcomings and the presentation of the findings as robust despite this not being the case (keyword: meta-analysis), one must question whether the WHO-reviewers' intentions and methodology were designed to downplay the risks posed by wireless communication technologies. (RH)

Bosch-Capblanch X, Esu E, Oringanje CM, Dongus S, Jalilian H, Eyers J et al. (2024). The effects of radiofrequency electromagnetic fields exposure on human self-reported symptoms: A systematic review of human experimental studies. Environment International, 187: 108612. https://doi.org/10.1016/j.envint.2024.108612

Lin JC (2021). Science, politics, and groupthink [Health Matters]. IEEE Microwave Magazine, 22(5), 24–26. https://doi.org/10.1109/MMM.2021.3056975

Mevissen M, Ducray A, Ward JM, Kopp-Schneider A, McNamee JP, Wood AW et al. (2025). Effects of radiofrequency electromagnetic field exposure on cancer in laboratory animal studies, a systematic review. Environment International, 199: 109482. https://doi.org/10.1016/j.envint.2025.109482

Meyer F, Bitsch A, Forman HJ, Fragoulis A, Ghezzi P, Henschenmacher B et al. (2024). The effects of radiofrequency electromagnetic field exposure on biomarkers of oxidative stress *in vivo* and *in vitro*: A systematic review of experimental studies. Environment International, 194: 108940. https://doi.org/10.1016/j.envint.2024.108940

RF radiation has genetic effects

Radiofrequency radiation-induced gene expression

Lai H, Levitt BB (2025). Radiofrequency radiation-induced gene expression. Reviews on Environmental Health. Advance online publication. https://doi.org/10.1515/reveh-2025-0104

Gene expression (the process by which genetic information is converted into a functional product) changes in response to environmental changes to restore cellular homeostasis. This is an adaptive mechanism. The nature of these changes reflects how cells are affected by disruptive factors. In their narrative review, the authors summarize the extensive scientific literature on the effects of radiofrequency electromagnetic fields (RF-EMF) on gene expression.

Study design and implementation:

In their narrative review, the authors evaluated over 500 primary studies that examined the genetic effects of RF radiation. The authors focused on studies examining frequencies between 800 and 2500 MHz – a spectrum typical of mobile devices. The included studies used in vitro and in vivo models, as well as acute and chronic exposure. The model organisms included a wide variety, ranging from cell cultures of various origins to rodents, insects, and plants. The 6 categories of gene expression covered were: 1) repair and removal of damaged proteins, 2) DNA damage, 3) oxidative processes, 4) stress responses, 5) apoptosis, and 6) brain functions. Exposure parameters varied considerably, with specific absorption rates (SAR) ranging from a few μW/kg to 20 W/kg.

Results:

Given the wide variation in study design among the primary studies, it is not surprising that they present an inconsistent picture of altered gene expression. However, one consistent finding is that RF radiation alters the expression of genes involved in key cellular processes. For example, heat shock proteins (Hsp70 and Hsp90) are consistently overexpressed. (Despite their name, these proteins respond to various cellular stress factors helping, among others, to protect DNA and break down damaged cellular components; editor's note.) Changes have also been observed in genes associated with DNA damage and repair, such as p53 and p21. Other areas in which adaptive gene expression has been repeatedly observed include oxidative processes (e.g. antioxidant and protective enzymes, Hsp70), programmed cell death (both pro- and anti-apoptotic), and genes associated with brain function. These include neurotransmitter genes, NMDA receptor genes, and cancer-related microRNA (miRNA). At a SAR value of less than 0.4 W/kg, 40 different effects of RF radiation on gene expression were detected.

Conclusions:

The authors argue that changes in gene expression provide strong evidence that RF radiation affects cellular functions. This is a direct response to external stress, through which normal conditions are restored. The function or association of the altered genes reveals the effects of RF radiation on cells. For example, it would be illogical to assume that genes associated with DNA repair would be upregulated in the absence of DNA damage. Due to the complex interaction between RF radiation and cells, a non-linear dose–response relationship is to be expected, as are studies in which no biological effect of RF radiation on gene expression can be detected. The frequency of effects at low intensities (SAR < 0.4 W/kg) calls into question the appropriateness of current exposure guidelines. Lai and Levitt call for more in-depth research on altered gene expression due to RF radiation, focusing on two areas in particular: (1) the involvement of cellular stress responses in the hypothalamic–pituitary–adrenal (HPA) axis and its effects on the limbic system and (2) cellular oxidation processes, particularly the induction of free radicals.

Editor's note:

As a narrative review, this study does not claim to provide a complete statistical evaluation of all relevant primary studies. However, it identifies recurring patterns by using a wide range of primary studies with different designs, endpoints, and results. (RH)

Fundamentals of biological effects of EMF

The collision between wireless and biology

Héroux P (2025). The collision between wireless and biology. Heliyon. 11(10): e42267. https://doi.org/10.1016/j.heliyon.2025.e42267

The energy and telecommunications industries have presented two arguments in support of their position that human exposure to non-thermal, non-ionizing radiation is safe. First, the radiation is non-ionizing. Second, the energy quanta of the radiation are too weak to overcome the energy of thermal motion of water molecules. However, powerful financial interests have obscured the relevant scientific principles. Consequently, the controversy over the health effects of non-thermal electromagnetic radiation (NTER) has persisted much longer than necessary. Industry-affiliated bodies, such as IEEE and ICNIRP, have adopted the specific absorption rate (SAR) and the associated temperature increase as the sole measure of the biological effects of electromagnetic fields. Only tissue heating above 1°C is considered relevant. This illustrates their a priori belief that heat is the only possible damaging factor and that long-term effects are not significant enough to be considered.

Historical overview and extension of thermodynamic concepts:

In 1889, Arrhenius introduced the concept of activation energy, which is defined as the energy barrier that must be overcome (e.g. by heating) for two molecules to react. In 1935, Eyring proposed an improved equation for chemical reaction rates. Unlike the Arrhenius equation, which was limited to gases, Eyring's equation included condensed matter, such as liquids and solids. In 1977, Bennett and Chandler further refined the equation of reaction rates. This refinement can be interpreted as an expression of all reactions occurring in living systems. Maintaining life (homeostasis) requires continuously arranging atoms and molecules into assemblies. The development of these complex structures reduces entropy (negative ΔS), thus counteracting the universal tendency toward disorder and uniformity dictated by the second law of thermodynamics. Life requires and forms ordered structures, which require energy from the environment (e.g. sunlight or food). In this process, energy is also released back into the environment. This energy comes from redox reactions, which involve the transfer of electrons. These electron transfers inevitably result in electron losses. Oxygen eagerly captures these losses as reactive oxygen species (ROS), which increase entropy to compensate for the entropy lost through biosynthesis. The negative effects of reactive oxygen species (ROS) are controlled by enzymatic antioxidants, such as superoxide dismutase, catalase, and glutathione, as well as non-enzymatic antioxidants, such as vitamins C and E.

According to the second law of thermodynamics, living tissues actively generate ROS through metabolic activity. The fact that life provides its own metabolic energy, activating reactions independently of energy supplied by electromagnetic fields, removes objections to the health effects of NTER. The movement of electrons from food to oxygen within the mitochondria in our bodies is called the electron transport chain. The process by which oxygen combines with fatty acids from food in the mitochondria is called oxidative phosphorylation (OXPHOS). A single OXPHOS chain consists of many enzymes, some of which are large molecules called Complexes I through V, and electron and proton currents circulate continuously between them. These traveling electrons are inevitably attracted to external alternating electromagnetic fields, which causes their position and circulation to change. Complex I, the first enzyme in the OXPHOS chain, is approximately 1.7 billion times more massive than an electron. It forms an immovable reference to the NTER action on electrons. Although OXPHOS units are only a few hundred nanometers in size, countless numbers of them are scattered throughout the human body. The human body contains quintillions of conductors, each measuring a few hundred nanometers, that continuously transport the necessary electron and proton currents to maintain cellular energy.

The author re-writes the aforementioned Bennett-Chandler equation by incorporating the signal crest factor, which is often observed in radiofrequency electromagnetic fields (RF-EMF) and indicates the extent to which peak values within the signal deviate from the mean. A higher crest factor amplifies non-thermal biological effects. The crest factor has steadily increased from 1G to 5G. Furthermore, according to the Shannon-Hartley theorem on information transmission, the steadily increasing speed of modern communication signals implies a need for higher crest factors and stronger biological effects. Héroux's analysis reveals that the IEEE/ICNIRP's position on the health implications of NTER is rooted in the late 19th century due to its reliance on the Arrhenius equation and the concept of activation energy.

Conclusions:

Modeling living tissue as a mixture of sugar, water, and salt (the same mixture used to establish SAR values) ignores a fundamental aspect of life: the continuous transport of electrons and protons, which is maintained by metabolism. These currents are susceptible to NTER, especially with regard to OXPHOS. A more complete model (than the one used by the ICNIRP), which is based on the principle of superposition, shows that oscillating electric or magnetic fields penetrating the body from the environment alter the tissue's electron and proton currents. For NTER to be biologically active, it does not need to generate ionized targets or electron currents; it only needs to interfere with the currents produced by biology. The current environmental perspective is flawed because an extensive engineering toolbox is available to mitigate NTER exposure. This toolbox includes fiber-optic networks, wire twisting, grounding measures, and direct current.

Editor's note:

An interesting and novel approach is extending the thermodynamic equation to biological effects, including a historical timeline. Many studies have shown that oxidative stress is the primary effect of non-thermal electromagnetic radiation. Although experiments have proven Panagopoulos's theory of voltage-gated calcium channel activation (which also triggers oxidative stress), the precise biophysical mechanism has yet to be confirmed. The thermodynamic approach used here explains the direct formation of ROS in mitochondria by disrupting electron and proton flows in OXPHOS. However, this approach has only been partially confirmed by experiments and has yet to be confirmed by computer modeling. Nevertheless, it seems plausible that several biological processes are disrupted simultaneously by different mechanisms. (AT)

Addresses for additional reliable information

Diagnose-Funk e. V. - Environmental and consumer organization for protection from electromagnetic radiation (Germany):
Website: diagnose-funk.org
Email: info@diagnose-funk.de

Microwave News (USA):
Website: microwavenews.com
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Institute Website: publichealth.berkeley.edu/people/joel-moskowitz
EMF Website: www.saferemr.com

Prof. Devra Davis (USA):
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Prof. Igor Belyaev, Biomedical Research Center of the Slovak Academy of Science, Department of Radiobiology:
http://www.biomedcentrum.sav.sk/research-departments/department-of-radiobiology/?lang=en
Shortened Link: kurzlinks.de/belyaev

Blog by Prof. Darius Leszczynski (Finland):
Website: betweenrockandhardplace.wordpress.com

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www.emfdata.org
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