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ElektrosmogReport

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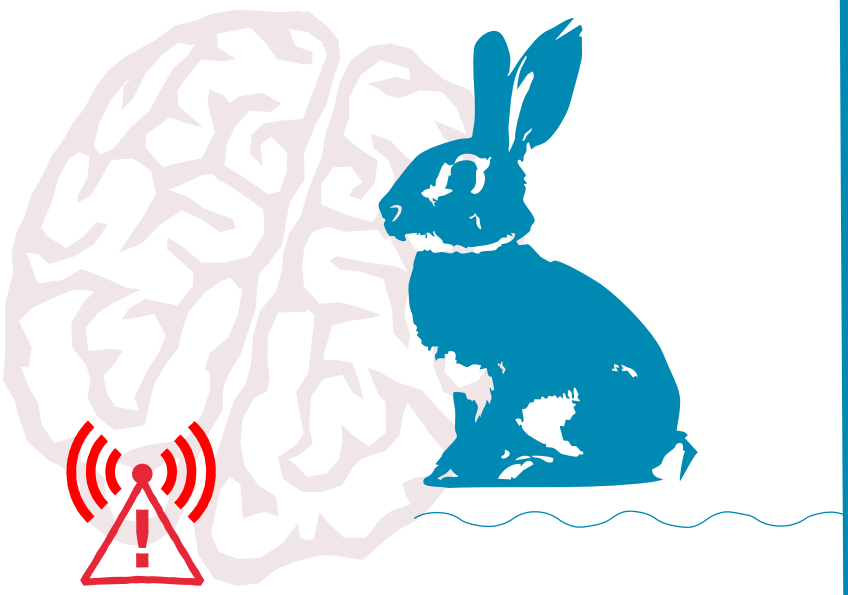

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2100 MHz GSM radiation compromises the blood-brain barrier

Effects of 1800 MHz and 2100 MHz mobile phone radiation on the blood-brain barrier of New Zealand rabbits

Kizilçay AO, Tütüncü B, Koçarslan M, Gözel MA (2024): Effects of 1800 MHz and 2100 MHz mobile phone radiation on the blood-brain barrier of New Zealand rabbits. Medical & Biological Engineering & Computing, November. https://doi.org/10.1007/s11517-024-03238-1

In the context of potential health risks associated with mobile phone radiation, its effect on the blood-brain barrier (BBB) is of particular relevance. Under normal conditions, this barrier functions so effectively that it significantly limits the absorption of most drugs, posing a major challenge to drug manufacturers. Impairment of the BBB is associated with serious neurological diseases such as Alzheimer's, stroke, and multiple sclerosis. Previous studies have shown that radiofrequency radiation can compromise the BBB, allowing substances to enter the brain that would be excluded by an intact barrier. Under-

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standing the interaction between mobile phone radiation and the BBB is critical because any disruption of BBB integrity could have serious implications for neurological health. The present study investigates the effects of 1800 MHz and 2100 MHz radiation on the BBB using New Zealand rabbits. These animals are commonly used in neurobiological research because of their well-characterized physiology and relatively large brains. The experimental design included non-thermal exposure within the typical range of mobile phone use and a thorough characterization of the exposure setup.

Study design and methods:

A total of 21 female rabbits were divided into three groups (n = 7 each): cage control, 1800 MHz GSM and 2100 MHz GSM. The animals were exposed once for 38 minutes at a power level of 15 dBm. Exposure was performed in a metal cage to minimize electromagnetic interference. The exposure system allowed real-time monitoring of the emitted radiofrequency power levels, ensuring that the animals were reliably and consistently exposed to the specified power levels. BBB permeability was assessed using Evans Blue dye, which binds to plasma proteins, primarily albumin. The integrity of the BBB is compromised if the dye enters the brain. (Albumin can induce a number of pathological responses in the brain, including disruption of potassium and neurotransmitter homeostasis, editor's note.) For each animal, two brain samples from each hemisphere were collected, homogenized and analyzed spectrometrically at 620 nm.

Results:

The researchers observed changes in Evans Blue levels in the brain tissue of the exposed rabbits compared to the unexposed controls. BBB permeability values were increased in both the 1800 MHz and 2100 MHz groups compared to controls, but only the 2100 MHz group showed statistically significant results. A statistically significant difference with a 95 % confidence interval was found in absorption at 620 nm for both the left and right hemispheres in the 2100 MHz group compared to the control group.

Conclusions:

Even a single exposure under non-thermal conditions with a radiofrequency intensity approximately ten times lower than the „normal value" resulted in increased BBB permeability at 2100 MHz. The exposure setup was designed by the researchers to minimize external interference and to allow for a valid assessment of the effects of radiofrequency radiation. The experimental animals were exposed only to the intended radiofrequency signals, with continuous environmental monitoring to detect any unusual variations. The observed permeability of the BBB may play a role in several neurological diseases such as Alzheimer's, stroke, and multiple sclerosis.

Editor's note:

In particular, the real-time monitoring of radiofrequency exposure and the measurement system are considered high-quality aspects of this study. A sham exposure for the control group would have been desirable in this context. The present study confirms the findings of Sırav & Seyhan, 2016, who also observed increased BBB permeability after a single RF exposure, albeit in rats. BBB impairment has also been documented with long-term RF exposure (Tang et al., 2015). (RH)

References:

Abu-Taweel GM (2019): Neurobehavioral protective properties of curcumin against the mercury chloride treated mice offspring. Saudi Journal of Biological Sciences, 26(4), 736-743. https://doi.org/10.1016/J.SJBS.2018.10.016

Cai H, Chen M, Gao Y, Ruan J, He C, Zuo Z (2023): Transgenerational Effects and Mechanisms of Tributyltin Exposure on Neurodevelopment in the Male Offspring of Rats. Environmental Science & Technology, 57(28), 10201-10210. https://doi.org/10.1021/acs.est.3c01546

Huang CF, Hsu CJ, Liu SH, Lin-Shiau SY (2008): Neurotoxicological mechanism of methylmercury induced by low-dose and long-term exposure in mice: Oxidative stress and down-regulated Na+/K+-ATPase involved. Toxicology Letters, 176(3), 188-197. https://doi.org/10.1016/J.TOXLET.2007.11.004

„Increased permeability of the BBB may contribute to Alzheimer's, stroke, and multiple sclerosis."

Worker exposure and biomarkers

The effect of electrical substations and cellular communication towers on oxidative stress and thyroid gland hormones

Hakeem Kadhim L, Taha Mohammed M, S Al-Fartusie F, Almohammadawi K (2023): The Effect of Electrical Substations and Cellular Communication Towers on Oxidative Stress and Thyroid Gland Hormones. Egyptian Journal of Chemistry. 2023 Apr 1;66(4):115-21. https://doi.org/10.21608/ejchem.2022.141247.6173

Reactive oxygen species (ROS) are normal byproducts of the human body under physiological conditions and serve a beneficial function as redox signaling molecules. However, ROS possess oxidative properties that can damage cellular macromolecules, such as lipids, proteins, DNA and RNA, potentially leading to cell apoptosis and pathological conditions. There are exogenous sources of ROS that can directly influence the development of oxidative stress, including radiation, heavy metals, smoke, and insecticides. The body contains many antioxidants that are designed to neutralize and detoxify the oxidative effects of ROS. Thus, oxidative stress results from either increased ROS concentrations, decreased antioxidant levels, or both.

In the present study, oxidative stress, antioxidant levels, and thyroid hormones were analyzed in the serum of workers in electrical substations (operating with ultra-high voltage) and cell tower technicians. Oxidative stress was assessed using malondialdehyde (MDA) and total oxidative status (TOS) as indicators, while antioxidant levels were measured using glutathione (GSH) and total antioxidant capacity (TAC). For thyroid function, thyroxine (T4), triiodothyronine (T3), and thyroid-stimulating hormone (TSH) were measured.

Study design and methods:

The study included 40 substation workers (aged 19-60 years), 40 cell tower technicians (aged 26-55 years), and 40 healthy controls (aged 21-55 years). All participants were male. Data were collected from November 2021 to January 2022. TSH, T3, and T4 levels were determined using a Cobas e411 analyzer (Roche, Germany). TAC, TOS, MDA, and GSH levels were measured with a PD-303 APEL spectrophotometer (Japan).

Results:

There were no significant differences ($p > 0.05$) in age, BMI, or smoking prevalence between the exposed groups and the control group. TSH levels were slightly but significantly elevated ($p < 0.05$) in substation workers compared to both the control group and cell tower technicians. The differences in T3 and T4 levels were not significant.

MDA levels were significantly elevated in the serum of substation workers (by 220 %) and cell tower technicians (by 120 %) compared to the control group. TOS concentrations were significantly elevated in the serum of substation workers (by 530 %) and cell tower technicians (by 240 %) compared to controls. In addition, MDA and TOS levels were significantly higher ($p < 0.05$) in substation workers than in cell tower technicians.

TAC levels were significantly reduced in the serum of substation workers (-61 %) and cell tower technicians (-42 %) compared to controls. GSH levels were significantly reduced in substation workers (-77 %) and cell tower technicians (28 %) compared to controls. Furthermore, TAC and GSH levels were significantly lower ($p < 0.05$) in substation workers than in cell tower technicians.

Conclusions:

The results indicate an increase in lipid peroxidation (MDA) and total oxidative stress (TOS), coupled with a decrease in reduced glutathione (GSH) and total antioxidant capacity (TAC) in the serum of substation workers and cell tower technicians. The most severe effects were observed in substation workers. The high electromagnetic field (EMF) exposure associated with these occupations leads to increased oxidative stress and may pose several long-term health risks. (AT)

Can cell phone radiation cause cancer?

Relationship between radiofrequency electromagnetic radiation from cellular phones and brain tumor: meta-analyses using various proxies for RF-EMF exposure outcome assessment

Moon J, Kwon J, Mun Y (2024): Relationship between radiofrequency-electromagnetic radiation from cellular phones and brain tumor: meta-analyses using various proxies for RF-EMR exposure-outcome assessment. Environmental Health, 23(1), 82. https://doi.org/10.1186/s12940-024-01117-8

The debate about whether there is an association between radiofrequency electromagnetic fields (RF-EMF) from mobile phones and the incidence of brain tumors has been ongoing since the early 2000s. Many researchers have conducted meta- and subgroup analyses to address this question. However, these studies have not found a clear positive association. More recently, with the introduction of 3G phones, user behavior has changed dramatically. The focus has shifted from voice calls to multime-

dia use, such as YouTube, TikTok, and other social media platforms. In addition, the use of WPAN technologies such as Bluetooth devices has become widespread. These changes increase the duration of mobile phone exposure and complicate the comparability of exposure based on individual use characteristics. In light of these developments, current metrics for assessing mobile phone exposure are imprecise and inadequate for a valid assessment of tumor incidence. In theory, an accurate assessment of mobile phone exposure should be based on i) site-specific, ii) time-integrated, and iii) specific absorption rate (SAR) parameters. Common metrics such as years of mobile phone use, cumulative call duration, and number of calls per week are rough indicators at best. The iii) SAR parameter is roughly made up of two components: the duration of use and the phone's output power. However, output power varies significantly depending on factors such as phone model, network used, and location of use. Regarding the ii) time-integrated parameter, an appropriate approximation would be the cumulative duration of use, weighted by the power at each time point, differentiated between sides of the head, and considering Bluetooth hands-free devices or headphones. This review addresses the potential association between mobile phone use and brain tumor incidence. Specifically, the authors conducted a series of meta-analyses and subgroup analyses using multiple exposure assessment categories, ranging from broad to more precise categorizations.

Study design and methods:

The authors analyzed original research articles on mobile phone use (1G, 2G, and 3G) and brain tumor risk from the PubMed, EMBASE, and Cochrane Library databases, published through July 2024. Review articles, conference abstracts, and similar publications were excluded. Cohort studies were included but analyzed separately. Data from 19 case-control studies and 5 cohort studies were extracted and included in the meta-analysis. Instead of the usual risk of bias (RoB) assessment for each study, the authors assessed selection and recall bias for total mobile phone use and misclassification and recall bias for ipsilateral/contralateral use. (Ipsilateral means on the same side of the body, contralateral means on the opposite side. For ipsilateral use, the mobile phone is held on the same side of the head where the tumor is located; for contralateral use, the phone is held on the opposite side, editor's note.) The primary meta- and subgroup analyses of the case-control studies were categorized into four groups: 1) regular vs. irregular mobile phone use; 2) laterality (ipsilateral and contralateral use vs. irregular use); 3) duration of use > 10 years or < 10 years; 4) analyses of the first three categories differentiated by tumor type (glioma, meningioma, acoustic neuroma, pituitary tumor, and malignant tumor). A separate meta-analysis was performed for participants with more than 896 hours of cumulative use. For cohort studies, the categories were: a) „ever use" vs. „never use" and b) „over 10 years use" vs. „never use"; c) within these categories, meningioma, acoustic neuroma, and glioma were distinguished. All meta-analyses were statistically evaluated with pooled point estimates (odds ratio, OR) and 95 % confidence intervals (CI).

Results:

First, the results of the case-control studies are summarized. Comparing regular and infrequent mobile phone users (category 1), no statistically significant differences in tumor incidence were observed. Considering laterality (category 2), a statistically significant 40 % increase in the overall risk of brain tumors was found for ipsilateral mobile phone use. In contrast, the risk was not increased for contralateral use. For use over 10 years (category 3), a statistically significant 27 % increase in risk was documented. Shorter periods of use of less than 10 years were not associated with an increased incidence of brain tumors. When differentiating by tumor type in category 1, a significant 16 % reduced risk of meningioma was observed for regular users. For ipsilateral use, the risk increased by 20 % for meningioma, 45 % for glioma, and 93 % for malignant tumors. Participants with more than 10 years of use had a 32 % increased risk of developing glioma. All other combinations of subcategories and tumor types did not yield statistically significant data. Cumulative use of more than 896 hours was associated with a 59 % increased risk of brain tumors, regardless of tumor type. The meta-analysis of cohort studies did not provide statistically significant evidence of an increased tumor risk from mobile phone use.

Conclusions:

In this meta-analysis, the pooled point estimates (ORs) showed increasing and statistically significant values with more precise subgrouping. (Double-digit percentage risk increases are considered significant, editor's note.) According to the authors, the number of cohort studies included was too small to draw valid conclusions. In addition, the assessment of exposure conditions in terms of i) site-specific, ii) time-integrated, and iii) specific absorption rate (SAR) parameters was too imprecise. The authors call for future cohort studies to refine exposure subcategories. It is also critical to account for changing use patterns, including WPAN technologies. Furthermore, there is evidence of potential underestimation in the risk assessments of previous studies, leading to adjustments in their risk of bias. The relatively short observation periods, considering the latency of tumor development, and the age at exposure onset are factors that, if not properly accounted for, could lead to risk underestimation. According to the authors, future studies should aim to address these biases in their study designs. (RH)

„For ipsilateral use, the risk increased by 20 % for meningioma, 45 % for glioma, and 93 % for malignant tumors."

Non-thermal RF-EMF effects

Potential non-thermal molecular effects of external radiofrequency electromagnetic fields on cancer

Dieper A, Scheidegger S, Füchslin RM, Veltsista PD, Stein U, Weyland M, Gerster D, Beck M, Bengtsson O, Zips D, Ghadjar P (2024): Literature review: potential non-thermal molecular effects of external radiofrequency electromagnetic fields on cancer. International Journal of Hyperthermia. 2024 Dec 31;41(1):2379992. https://doi.org/10.1080/02656736.2024.2379992

Hyperthermia therapy, the heating of tumors to temperatures of 39-44°C using radiofrequency electromagnetic fields (RF-EMF), has been proposed as an adjunct cancer treatment to established therapies such as radiation and chemotherapy. However, there are technical limitations in achieving the required temperatures deep within the body. In particular, patient thermoregulation, the heat transfer from target areas due to blood flow, presents a significant limitation for effective hyperthermia. The cancer-inhibiting effects of RF-EMF, which sensitize tumors to chemotherapy and radiotherapy, have so far been attributed to the induced temperature increases at the tumor site and the resulting effects. A growing body of evidence suggests that RF-EMF may also have tumor-damaging effects beyond local temperature increases. Numerous clinical data suggest that the method of Tumor Treating Fields (TTF) (Novocure, Switzerland), which uses low-intensity, intermediate frequency RF-EMF at temperatures below 38°C, results in significant cancer cell death. The same is true for RF-EMF in the GHz spectrum at intensities that cause only negligible temperature increases (+1.58°C).

While the exact mechanisms through which non-thermal RF-EMF might exert anti-cancer effects remain largely unclear, there is a scientific consensus that certain features occur exclusively in tumor cells and not in healthy tissue cells. In addition to the altered expression of oncogenes, tumor promoters, and suppressor genes that are ubiquitous in cancer cells, these specific characteristics of cancer cells are thought to lie in their pronounced bioelectric properties, such as the expression of aberrant ion channels and membrane potentials, and in their specific mechanical properties, such as altered cell membrane elasticity and cytoskeletal organization. This raises the question of whether these characteristics make cancer cells particularly susceptible to the effects of RF-EMF radiation. The exact nature of the molecular mechanisms activated during RF-EMF exposure remains to be investigated and identified.

Study design and methods:

The authors conducted a review according to PRISMA guidelines. All studies reporting effects induced by frequencies outside the RF spectrum were excluded. Studies without temperature control or those using RF-EMF that caused significant temperature increases within cells (> 40.5°C) were also excluded, as the aim was to only investigate the non-temperature-induced effects of RF-EMF. A literature search was conducted in several databases, particularly PubMed (MEDLINE) and Scopus (Elsevier). Additional literature was identified by reviewing the sources of previously collected and selected papers. A risk of bias assessment was not performed as it was considered unsuitable for a preclinical investigation.

Results:

This review included 32 preclinical studies reporting a variety of EMF-induced molecular effects in cancer cells. The studies were divided into three EMF exposure groups:

- Group 1 (20 studies): The „TTF" category, in which an alternating electric field is generated and applied to the patient's body via a pair of insulated wires. TTF is characterized by intermediate frequency and low intensity, and includes studies with a frequency range of 100-300 kHz. The field strength varied from 0.5 to 3 V/cm.
- Group 2 (4 studies): „Therabionics/AutEMDev" approaches fall into the high frequency, low-intensity category. The included studies used frequencies between 27.12 MHz and 147 MHz, with SAR values ranging from 0.01 to 0.4 W/kg. This group is mainly characterized by uniform amplitude modulation with a particularly high modulation index of 80-85 %.
- Group 3 (6 studies): Millimeter waves (MMW) and microwave (MW) RF fall into the category of extremely high frequencies with low intensity. The frequencies range from 900 MHz to 105 GHz, the incident power densities range from 0.001 to 0.2 mW/m² (0.01 to 2 W/m²), and the SAR values are low (0.0038 to 1 W/kg).

Of the 32 preclinical studies analyzed, 26 directly reported reduced cancer cell proliferation, viability, or migration in vitro and in vivo in animal tumors after RF-EMF treatment. Several studies in this review reported significantly reduced cell proliferation after RF-EMF treatment, particularly in the intermediate RF-EMF spectrum between 100-300 kHz, applied via TTF or TTF-like devices. Continuous TTF exposure at 1.5 V/cm for 24 hours significantly inhibited the proliferation rate of breast cancer cells, resulting in a marked decrease in cell number ($p < 0.0001$). Jimenez et al. (2019) observed significant proliferation inhibition after a 27.12 MHz amplitude-modulated EMF treatment with Therabionics. Beneduci et al. (2005) also reported growth inhibitory effects after RF-EMF application in the microwave spectrum at 53-78 GHz in human breast cancer cells (MCF-7), with a 60 % reduction in cell growth.

Conclusions:

The literature analysis provides a first overview of the diverse non-temperature-related molecular effects that can be

observed in cancer cells under RF-EMF exposure, suggesting that RF-EMF treatment specifically targets ion channels and cell mechanics. None of the reviewed studies observed stimulation of cancer cell proliferation or migration by RF-EMF treatment. This is noteworthy because the observed RF-EMF effects on cancer cells appear to contrast with the positive stimulatory effects of EMF on healthy tissue cells (potentially increasing cancer risk). Careful dosimetry is essential for safe and effective RF therapy. Many studies have reported the importance of optimal frequency selection for TTF application in different cancer cell lines. They indicate that each cancer cell line has a specific frequency optimum at which the anti-cancer effect is maximal. Frequencies outside of this optimum result in either a reduced or no significant anti-cancer effect.

The existing literature suggests an untapped therapeutic potential of RF-EMF treatment when appropriate and careful dosimetry is applied. Further research is essential to determine optimal cancer-specific RF-EMF frequencies, field intensities, and exposure intervals. (AT)

Radiofrequency radiation leads to cell death

Radiofrequency-induced time-dependent alterations in gene expression and apoptosis in a glioblastoma cell line

Tuysuz MZ, Kayhan H, Saglam ASY, Senturk F, Bagriacik EU, Yagci M, Canseven AG (2025): Radiofrequency Induced Time-Dependent Alterations in Gene Expression and Apoptosis in Glioblastoma Cell Line. Bioelectromagnetics, 46(1). https://doi.org/10.1002/bem.22543

Numerous experimental studies have shown that radiofrequency (RF) fields can induce various biological responses, including apoptosis, autophagy, DNA damage, inflammatory responses, oxidative stress, and altered gene expression. In the context of apoptosis, RF has been shown to specifically affect apoptotic markers, including pro-apoptotic proteins such as BAX and CASP, and anti-apoptotic markers such as BCL-2. While epidemiological studies suggest an increased risk of cancer, experimental research on the effects and mechanisms of RF fields remains controversial and limited. This underscores the urgent need for new, comprehensive experiments. The present study aims to investigate the effects of 2100 MHz RF exposure on cell viability, apoptosis, and gene expression in the human glioblastoma cell line U118-MG in vitro. The U118-MG cell line was chosen because it is derived from human glial cell cancer and epidemiological studies suggest an increased risk of glioma associated with mobile phone use.

Study design and methods:

The U118-MG cell line was exposed to 2100 MHz RF at 60 V/m, resulting in an average SAR (Specific Absorption Rate) of 1.12 ± 0.18 W/kg per 1 g. Cells were exposed for 1 h, 24 h, or 48 h, with sham-exposed cells used as controls. The SAR level used is consistent with current safety guidelines and is below recommended limits. Real-time temperature monitoring was performed to confirm no temperature variations. The exposure system was shielded from external electromagnetic fields. Exposure conditions and dosimetry were carefully characterized to ensure consistent and controlled exposure conditions in a stable and reproducible environment. Biologically, the study examined cell viability, cell apoptosis by Annexin-V flow cytometry, gene expression of apoptosis markers (CASP3, CASP8, CASP9, BCL-2, and BAX) by quantitative PCR, and expression of transcription factors (CYCD1, C-MYC, and c-FOS) associated with cell division and apoptosis by quantitative PCR.

Results:

Data showed that cell viability remained unchanged compared to sham-exposed controls after 1 hour and 24 hours of exposure. However, a statistically significant decrease in cell viability was observed after 48 hours of exposure. This trend was consistent with the proportion of apoptotic cells. No changes were observed after 1 hour and 24 hours, but after 48 hours, the rate of apoptotic cells was significantly increased compared to sham-exposed controls. The relative gene expression of CASP3, CASP8, and CASP9 was significantly increased after 24 hours and 48 hours of exposure, whereas the BAX/BCL-2 ratio showed a statistically significant increase only after 48 hours of exposure. In contrast, the relative gene expression of the transcription factors was increased only after 1 hour of exposure and showed no significant changes after 24 or 48 hours.

Conclusions:

The data presented are consistent. Exposure to 2100 MHz RF at intensities below the recommended limits results in decreased cell viability and increased apoptosis after 48 hours. The BAX/BCL-2 mRNA ratio – considered a „cell death switch" and a critical regulator of cellular apoptosis – showed a 4.5-fold increase compared to sham-exposed controls. The authors hypothesize that during shorter exposure durations, cells activate stress response pathways (increased CASP3, CASP8, CASP9), but these changes in gene expression do not immediately lead to apoptosis. However, prolonged exposure exhausts the cell's ability to cope with RF-induced stress, resulting in significant apoptotic changes. The authors speculate that this may be related to exceeding the doubling time of U118-MG cells.

The transient increase in transcription factor expression suggests a temporary RF effect on the cells, which warrants further investigation as it may provide insight into the cells' compensatory mechanisms. (RH)

Prenatal mobile phone use delays development

Delayed growth in immature male rats exposed to 900 MHz radiofrequency

Bodin R, Robidel F, Rodrigues S, Lecomte A, Villégier A-S (2024): Delayed Growth in Immature Male Rats Exposed to 900 MHz Radiofrequency. Applied Sciences, 14(16), 6978. https://doi.org/10.3390/app14166978

The second generation of mobile communications (2G) was introduced in the 1990s with the GSM network (900 MHz). To date, exposure to 900 MHz mobile phone radiation is ubiquitous in both public and occupational settings. The 2G network has been shown to remain the primary source of brain penetration by radiofrequency electromagnetic fields during phone calls. The exposure limits are set at 0.08 W/kg for the general public (Pu) and 0.4 W/kg for occupational exposure (Oc). In both cases, there are particularly vulnerable groups, such as pregnant women, immature individuals (fetuses, newborns, children), and sick or elderly people. It is known that the skulls of fetuses and newborns provide less protection against the penetration of radiofrequency radiation than those of adults. Therefore, it can be assumed that the physiological and developmental protection provided by ICNIRP limits is less effective in vulnerable populations. This study aims to investigate how radiation exposure at public and occupational limits affects growth and development and to what extent these effects differ, using a rat model.

Study design and methods:

A total of 25 pregnant rats were divided into three groups: sham-exposed (n = 9), PuM (n = 8; whole-body SAR 0.08 W/kg), and OcM (n = 8; whole-body SAR 0.4 W/kg). The animals and their offspring were exposed to 900 MHz radiation from day 8 post-fertilization (prenatal) until day 17 after birth (postnatal) between 11:00 a.m. and 7:00 p.m. The field strengths were 30.2 V/m (PuM) and 67.5 V/m (OcM). At birth, the litters were reduced to three male and three female pups per litter. One pup of each sex was sacrificed on days 8, 17, and 43 after birth. General litter parameters (litter size, sex ratio, and number of stillbirths) were first analyzed. In addition, pup weight and size were documented from birth until day 43 postpartum. Each pup was examined daily to determine the timing of eye opening, incisor eruption, and ear unfolding. These events are considered developmental markers.

Results:

The study found no evidence of adverse effects of perinatal radiation exposure on general litter parameters (litter size, sex ratio, number of stillbirths) or on pup weight and size at birth. However, a statistically significant reduction in body weight was observed in OcM-exposed male pups (0.4 W/kg) from day 6, which persisted until the end of the experiment (day 43). Male pups in both exposed groups were also smaller, although this result did not reach statistical significance (p = 0.0557). However, the effects of 900 MHz exposure on early developmental markers were statistically significant. Eye opening and ear unfolding were observed earlier in both exposure groups and both sexes. Incisor eruption was significantly earlier only in the male pups of the PuM group.

Conclusions:

This publication is the first to compare the effects of public and occupational exposure limits on the physical development of rats. The results suggest physical developmental defects (premature ear unfolding, early eye opening, reduced body weight), with occupational exposure limits causing a more pronounced phenotype. The observed phenotype may indicate a metabolic disorder with increased energy demand. The authors note that comparable developmental defects have been observed in newborn rats and mice with chemically disrupted serotonin and dopamine metabolism (Abu-Taweel, 2019; Cai et al., 2023). The neurotoxic effects of these chemicals are partly due to oxidative stress (Huang et al., 2008). (RH)

References:

Abu-Taweel GM (2019): Neurobehavioral protective properties of curcumin against the mercury chloride treated mice offspring. Saudi Journal of Biological Sciences, 26(4), 736-743. https://doi.org/10.1016/J.SJBS.2018.10.016

Cai H, Chen M, Gao Y, Ruan J, He C, Zuo Z (2023): Transgenerational Effects and Mechanisms of Tributyltin Exposure on Neurodevelopment in the Male Offspring of Rats. Environmental Science & Technology, 57(28), 10201-10210. https://doi.org/10.1021/acs.est.3c01546

Huang CF, Hsu CJ, Liu SH, Lin-Shiau SY (2008): Neurotoxicological mechanism of methylmercury induced by low-dose and long-term exposure in mice: Oxidative stress and down-regulated Na+/K+-ATPase involved. Toxicology Letters, 176(3), 188-197. https://doi.org/10.1016/J.TOXLET.2007.11.004

„Premature eye opening and ear unfolding occurred at both field strengths and in both sexes."

5G mobile networks can cause testicular damage

Effects of 5G mobile phone network electromagnetic field exposure on testicular endoplasmic reticulum stress and the protective role of coenzyme Q10

Yilmaz H, Tümkaya L, Mercantepe T, Yılmaz A, Gül F, Suzan ZT (2025): Effects of 5 G mobile phone network electromagnetic field exposure on testicular endoplasmic reticulum stress and the protective role of coenzyme Q10. Archives of Medical Research, 56(4), 103157. https://doi.org/10.1016/j.arcmed.2024.103157

The fact that the 5G wireless standard was introduced without a comprehensive review of its potential health effects has raised concerns among both scientists and the general public. The lack of a standardized methodology for assessing radiofrequency exposure from 5G mobile phone handsets and base stations exacerbates these concerns. Furthermore, 5G networks will require an exponentially higher number of base stations (10 to 100 times more), which underscores the need to investigate potential health risks associated with 5G mobile networks. Coenzyme Q10, a powerful antioxidant, has been shown in the literature to play an active role in mitigating apoptosis, which is closely associated with endoplasmic reticulum (ER) stress. ER stress, which results from oxidative stress, apoptosis, and mitochondrial dysfunction, can lead to testicular dysfunction. During ER stress, the levels of the chaperone glucose-regulated protein (GRP78) and C/EBP homologous protein (CHOP) increase. The aim of the present study was to biochemically and histopathologically investigate the effects of 5G mobile phone radiation on ER stress in testicular tissue and to analyze the potential protective role of Q10.

Study design and methods:

The study was conducted on 3- to 4-month-old Sprague-Dawley rats. They were divided into three groups (n = 8): cage control, 5G exposure, and 5G exposure + 10 mg/kg Q10. The animals were exposed to 5.9 GHz for 2 hours per day at an electric field strength of 4 V/m (0.42 W/m^2), resulting in a whole-body SAR of 0.0213 W/kg. The field strength was measured at different locations in the exposure cage. The experimental duration was 30 days. The researchers performed biochemical analysis of oxidative stress markers (TBARS, total thiol), immunohistochemical analysis of ER stress markers (GRP78, CHOP), and histopathological analysis of testicular tissue. The immunohistochemical and histopathological examinations were evaluated semi-quantitatively. The degree of histopathological damage was determined using the Testicular Histopathologic Damage Scoring (THDS), which includes loss of spermatogenic cells, edema areas, inflammatory processes, and vascular occlusion. Semi-quantitative analysis of immunohistochemical samples was conducted double-blind, and an IHC score for immunopositive cells was determined.

Results:

The authors' results showed a very consistent pattern. All studied endpoints (see above) indicated adverse effects of 5.9 GHz exposure. Compared to controls, statistically significant harmful effects of 5G radiation were observed in oxidative stress markers, as well as in histopathological and immunohistochemical scores. Specifically, exposure resulted in histopathological damage (increased edema areas, vascular occlusions, and neutrophilic inflammation of seminiferous tubules), increased TBARS, decreased total thiol, and increased number of GRP78- and CHOP-positive cells. In the experimental group receiving the antioxidant Q10, the harmful effects of 5G mobile phone radiation were statistically significantly reduced compared to the group exposed to radiation only.

Conclusions:

Male infertility, a global health issue, may be associated with homeostatic ROS imbalance in the reproductive system. Excessive production of reactive oxygen species can lead to ER stress, which in turn may cause oxidative stress. This condition can adversely affect sperm quality. The authors highlight the potential of powerful antioxidants such as Q10 to break the vicious cycle of ROS-ER stress induced by mobile phone radiation.

Editor's note:

This study adds to the growing number of studies linking non-thermal mobile phone radiation of various frequencies to reduced male fertility. This has been extensively discussed in several reviews (Yadav et al., 2021; Yu et al., 2021). Even proponents of the „purely thermal effects" hypothesis have had to acknowledge potential harmful effects of radiofrequency radiation on male fertility (Cordelli et al., 2024). Currently, the effects of 5G mobile communication are not sufficiently characterized. The present study contributes to filling this knowledge gap. (RH)

References:

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Digital devices and migraine

The role of digital device use on the risk of migraine

He Z, Qiu F, Yang J, Zhao M (2024): The role of digital device use on the risk of migraine: a univariable and multivariable Mendelian randomization study. Frontiers in Neurology. 2024 Oct 30;15:1462414. https://doi.org/10.3389/fneur.2024.1462414

Migraine is a common and debilitating neurological disorder characterized by recurrent headaches, often accompanied by nausea, vomiting, and sensitivity to light and sound. It affects over 100 million people worldwide, predominantly under the age of 50, and is the second leading cause of years lived with disability across all age groups. The one-year prevalence rates of migraine in Western countries vary, ranging from 4 % to 9 % in men and from 11 % to 25 % in women. Given the severe physical and psychological impact on patients, preventing migraine attacks is crucial. Previous studies have identified several risk factors that contribute to migraines, including sleep patterns, dietary habits, physical activity, and medication use. Digital dependence, which is closely linked to genetic predisposition, significantly affects brain function and structure. Prolonged exposure to blue light and electromagnetic radiation can lead to neurological disorders such as headaches, sleep disturbances, negative emotions, memory loss, and attention deficits. Observational studies have shown that frequent use of electronic devices is associated with an increased risk of migraines, especially among students. However, traditional observational studies are susceptible to confounding factors that limit the reliability of causal inferences, making it difficult to establish a definitive causal link between digital device use and migraine risk.

Mendelian randomization (MR) is a method that uses measured genetic variation to investigate the causal effect of an exposure on an outcome. Data from genome-wide association studies (GWAS) are often used in the analysis of summary data. A GWAS is an observational study of a genome-wide set of genetic variants in different individuals to determine whether a variant is associated with a trait. In this case, the association between genetic variants and the exposure is derived from the summary results of a GWAS for the exposure. The association between the same genetic variants and the outcome is then derived from the summary results of a GWAS for the outcome. These two sets of pooled results are then used to derive the MR estimate. Genetic variants, such as single nucleotide polymorphisms (SNPs), are used as proxies for the exposure (or outcome) of interest. These genetic variants are assumed to be randomly distributed and unaffected by confounding factors. By using genetic variants as instruments, MR can help infer causal relationships between exposures and outcomes without the need for traditional randomized controlled trials. The present study investigates the causal relationship between digital device use and migraine risk using Mendelian randomization.

Study design and methods:

GWAS data on four types of digital device use – mobile phone use, television viewing, computer use, and video gaming – were obtained from the UK Biobank, a large-scale database of genetic and health data from half a million UK participants. Digital device use was self-reported. Mobile phone use was defined as the number of calls made or received per week in the previous three months (n = 386,626 participants). Television use was measured by daily viewing time (n = 437,887), computer use by daily use time (n = 360,895), and video game use by gaming practices (n = 462,433). GWAS data for migraines were obtained from two large datasets. The primary discovery cohort was FinnGen, a public-private partnership project in Finland that combines genetic data with digital health records from national health registries. It includes 20,908 cases of total migraine, 8,970 cases of migraine with aura (MA), and 7,593 cases of migraine without aura (MO). The replication cohort from the International Headache Genetics Consortium (IHGC) includes 48,975 European migraine cases overall, with 6,332 MA cases and 8,348 MO cases. To select robust instrumental variables, only SNPs with a p-value < 5e-8 and minor allele frequencies > 0.01 were chosen. SNP independence was ensured using the 1000 Genomes Project European reference panel with a linkage disequilibrium threshold of $r^2 < 0.001$ within a 10 megabyte window. Effect alleles were harmonized between the digital device use and migraine GWAS datasets. The univariable Mendelian randomization (UVMR) approach was used to examine potential causality between digital device use and migraine risk using various statistical methods. A meta-analysis was conducted to combine the causal estimates derived from the discovery and replication datasets, subsequently validating the causal relationship between digital device use and migraines. A multivariable MR analysis was then performed, adjusting for relevant confounders (stroke, physical activity, hypertension, insomnia, clinical depression, alcohol consumption, smoking, and body mass index).

Results:

Nine SNPs were uniquely associated with mobile phone use (either positively or negatively). Most of these SNPs have currently unknown functions. However, for three SNPs, the genes and their functions are known. PHLPP2 is a phosphatase involved in the regulation of Akt and PKC signaling pathways. ARPP21 regulates the effect of dopamine on the basal ganglia. FOXP2 appears to be involved in a variety of biological signaling pathways and cascades that may ultimately influence language development. Eighty-nine SNPs were associated with television viewing. UVMR estimates based on the FinnGen cohort showed that genetically predicted mobile phone use was associated with an increased risk of total migraines (OR = 2.39) and MO (OR = 2.25). Similarly, television viewing was positively associated with an increased risk of

total migraines (OR = 1.63) and MO (OR = 2.10), but neither activity was significantly associated with MA. Negative associations were observed between computer use (OR = 0.67) and video gaming (OR = 0.41) with MO, although neither was significantly associated with total migraines or MA.

Meta-analysis estimates from two separate datasets (FinnGen and IHGC) confirmed a significant causal relationship between mobile phone use and total migraines (OR = 1.58) and a suggestive association for MO (OR = 1.73). Television viewing was significantly associated with total migraines (OR = 1.63) and MO (OR = 1.92). No causal relationships were found between computer use, video gaming, and any migraine subtype. The multivariable MR analysis, adjusted for relevant confounders, confirmed that mobile phone use was associated with an increased risk of migraines (OR = 1.40) and MO (OR = 1.88). Similarly, television viewing increased the risk of migraines (OR = 2.01) and MO (OR = 3.56).

Conclusions:

Previous observational studies have highlighted the adverse effects of excessive use of electronic devices on migraines. Regarding mobile phone use, a meta-analysis combining data from 30 cohorts of different ethnicities and populations aged 9-63 years found a positive association between weekly mobile phone use and migraine risk, suggesting that mobile phone radiation may be a risk factor for migraine (Farashi 2022).

The association between electronic device use and migraines may be explained by prolonged exposure to blue light (television) and electromagnetic radiation (mobile phones). A clinical study found that migraine patients had significantly and persistently lower pain perception thresholds after exposure to intense light than healthy individuals (Kowacs 2001). This phenomenon can be attributed to the stimulation of retinal ganglion cells by blue light, which subsequently affects the conduction of the trigeminal nociceptive pathway. This suggests that visual stimuli can trigger migraines and also disrupt sleep. In addition, the nervous system is highly sensitive to electromagnetic radiation (EMR). Prolonged exposure to radiofrequency EMR can lead to disruptions in neurotransmitter metabolism and oxidative stress in brain cells, both of which have been linked to the development of migraines. Overall, the mechanisms underlying the effects of electronic devices on migraines are complex and multifaceted and require further research.

Editor's note:

In this study, significantly stronger effects of mobile phone use (OR = 2.39) on migraine occurrence were observed in the Finnish cohort compared to the pan-European dataset (OR = 1.32). This higher sensitivity of northern populations has also been reported in the literature on electromagnetic hypersensitivity and other conditions. Conversely, the association between migraines and television viewing was identical in both datasets (OR = 1.63). (AT)

Mobile phones and headaches

Mobile phone electromagnetic radiation and the risk of headache

Farashi S, Bashirian S, Khazaei S, Khazaei M, Farhadinasab A (2022): Mobile phone electromagnetic radiation and the risk of headache: A systematic review and meta-analysis. International Archives of Occupational and Environmental Health. 2022 Sep;95(7):1587-601. https://doi.org/10.1007/s00420-022-01835-x

Headaches, a common problem for many people (10-20 % of adolescents), are a symptom that can be triggered by mobile phone use. Mobile phone use may be responsible for certain types of headaches, dizziness, neck and shoulder pain, stress and organ strain, and sleep disturbances. There is some evidence suggesting a causal relationship between headaches and mobile phone use. However, there are conflicting reports in the literature. Some studies have identified a statistically significant association, while others with controlled exposure duration have reported non-significant associations. In this context, a systematic review could help clarify the inconsistencies regarding the effects of mobile phone electromagnetic fields (EMFs) on headaches.

A previous systematic review (Wong et al., 2017) focused on the relationship between mobile phone call duration and headaches and found a causal link between call duration and headaches. The current systematic review included more studies and considered the impact of age as another factor in addition to the duration of EMF exposure from mobile phones (primarily call duration).

Study design and methods:

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed for this systematic review. The Web of Science, PubMed, and Scopus databases were searched for studies. The search was limited to the period from 1990 to February 2021, as mobile phone use was virtually non-existent before 1990. The PICO model (Population, Intervention, Comparison, and Outcome) was used to identify relevant studies. Only studies involving digital (as opposed to analog) mobile phones were considered. All subtypes of headaches were included in the current study. There were no age restrictions for participants and no limitations on the duration of EMF exposure, as exposure duration and age were also considered as potential moderator variables for possible effects.

Study quality was assessed using the National Institutes of Health (NIH) quality assessment tool for observational cohort and cross-sectional studies. The adjusted odds ratio (OR) was extracted or calculated based on the reported results (per study).

Results:

Thirty-three eligible studies were selected for further analysis. Some of these studies found a statistically significant effect of EMF exposure from mobile phones or base stations on headaches (n = 25). The total number of participants in the included studies was 109,385 (approximately 73.24 % female). Some studies reported more than one effect size for different exposure conditions. In this context, a total of 58 effect sizes were identified for the effects of EMF emitted by mobile phones or base stations on different types of headaches. Thirty studies provided sufficient information to calculate the OR and were included in the meta-analysis.

Of the 33 included studies, 21 % were of low quality (quality score < 7), 70 % were of medium quality (7 ≤ quality score < 10), and 9 % were of high quality (quality score ≥ 10). When all studies with different age ranges, exposure durations, and EMF-generating devices (i.e., mobile phones and base stations) were considered, the pooled effect was OR = 1.30 (95 % CI 1.21-1.39), indicating a significant effect of EMF on headaches. However, the heterogeneity between the studies was relatively high (I^2 = 72.2 %, p < 0.01).

To investigate the source of heterogeneity, age range and exposure duration were examined as potential influencing factors. For the subgroup analysis using participant age as a moderating variable, studies were divided into three categories: studies with participants aged ≤ 18 years, studies with participants older than 18 years, and studies without age-specific results. For the subgroup analysis considering exposure duration as a moderator variable, studies were divided into two groups: EMF exposure from mobile phones for more than 100 minutes per week, EMF exposure for 100 minutes per week or less. Based on the effect sizes and analysis of variance for these groups, no effect was found for age or the interaction between exposure duration and age. However, a significant effect was observed for exposure duration. When the age of the participants was limited to ≤ 18 years, the pooled effect size was OR = 1.29 (95 % CI 1.20-1.37), with moderate heterogeneity between studies (I^2 = 45.2 %, p = 0.004). For adults (> 18 years), a larger effect was observed (OR = 1.33, 95 % CI 1.14-1.53), with statistically non-significant heterogeneity (I^2 = 2.30 %, p = 0.408). For individuals exposed to mobile phone EMF: ≤ 100 minutes/week: OR = 1.23 (95 % CI 1.12-1.34), moderate heterogeneity (I^2 = 63.7 %, p < 0.001); > 100 minutes/week: OR = 1.41 (95 % CI 1.22-1.61), significant heterogeneity (I^2 = 77.4 %, p = 0.00).

For individuals exposed to EMF from mobile phone base stations, the pooled effect on headaches was OR = 1.14 (95 % CI 0.75-1.52), indicating a smaller, non-significant effect compared to actual mobile phone use.

Conclusions:

The pooled effect of mobile phone use on headaches was OR = 1.30 (95 % CI 1.21-1.39), indicating a significant effect of mobile phone use on headache prevalence. This is consistent with recent findings from a Mendelian randomization study (He, 2024, also reviewed in this ElektrosmogReport), which reported an OR of 1.40 (95 % CI 1.03-1.90) for migraine prevalence. For both age groups (≤ 18 and > 18 years), the pooled effect of EMF exposure on headache risk was lower for shorter EMF exposure durations. The headache risk for the higher exposure group (OR 1.41 for > 100 min/week) was about twice that of the lower exposure group (OR 1.21 for ≤ 100 min/week). An increased risk of headaches with longer exposure duration was observed in both younger and older participants.

These findings are consistent with a previous meta-analysis of seven studies (Wong et al. 2017), which also reported an increased risk of headaches with longer exposure duration. The results of the current study further suggest that mobile phone users have a higher risk of headaches compared to individuals living near mobile phone base stations.

Editor's note:

Since obtaining accurate exposure data near base stations is even more challenging than exposure from mobile phones, this issue should be re-evaluated in future studies with improved dosimetry. (AT)

Baby monitor disrupts sleep

Does radiofrequency radiation impact sleep? A double-blind, randomized, placebo-controlled, crossover pilot study

Bijlsma N, Conduit R, Kennedy G, Cohen M (2024): Does radiofrequency radiation impact sleep? A double-blind, randomised, placebo-controlled, crossover pilot study. Frontiers in Public Health, 12(October), 1-11. https://doi.org/10.3389/fpubh.2024.1481537

Sleep is a vital biological function, and sleep disorders are a major risk factor for cardiovascular disease, metabolic disorders, and premature mortality. Chronic sleep disorders impair neurological functions such as memory, concentration, and higher cognitive processes. They are also closely linked to the development of Alzheimer's. In children and young adults, sleep disorders are associated with mental health issues, depression, and poor academic performance. Currently, 4 out of 10 Australians suffer from sleep disorders, resulting in a significant social, financial and health burden. The rise in sleep disorders correlates with the global spread of mobile phones, which now number in the bil-

lions. However, studying the relationship between radiofrequency electromagnetic fields (RF-EMFs) and sleep disturbances is challenging. Epidemiological surveys are prone to respondent bias and rarely assess clinically relevant endpoints. Experimental studies should ideally be conducted outside of sleep laboratories, as unfamiliar environments may affect sleep, especially over a single night. In addition, EMFs should be generated by real sources rather than artificial ones, as real-world signals vary greatly in intensity and waveform, making them more biologically active. The aim of this study was to investigate the effects of radiofrequency EMFs in a real-world setting (using a commercially available baby monitor in the participants' own bedrooms) on clinically relevant sleep parameters in healthy adults.

Study design and methods:

This pilot study was a four-week, randomized, double-blind, crossover trial involving 12 healthy adults (3 males, 9 females). After a one-week familiarization period, participants were randomly assigned to be exposed to either an active baby monitor or a placebo (turned-off) device for seven consecutive nights. After a one-week washout period, the conditions were reversed: participants previously exposed to the placebo were now exposed to the active baby monitor, and vice versa. The baby monitor operated at a power level of 15 dBm, using a frequency range of 2.4 to 2.4835 GHz with frequency modulation to avoid interference. The monitor and camera units were placed within 2 meters of the participants' headboards, depending on the bedroom layout. Dosimetry measurements showed field strengths ranging from 2.2 to 7 mW/m^2, well below the ICNIRP limits for far-field exposure (10 W/m^2). Background electromagnetic noise was kept below 0.1 µT for magnetic fields and below 0.02 mW/m^2 for radiofrequency EMF. Subjective sleep quality was assessed using the Pittsburgh Insomnia Rating Scale (PIRS-20). Objective sleep measures were obtained by portable polysomnography (PSG), wrist-worn actigraphy, and sleep diaries. Heart rate variability (HRV) was also measured.

Results:

The PIRS-20 results showed a statistically significant deterioration in sleep quality for participants exposed to the active baby monitor compared to the placebo condition. Three participants (27.3 %) scored above the threshold of 20, indicating a risk of clinical sleep disorders. Electroencephalography (EEG) also revealed statistically significant differences between the active exposure and placebo conditions. Specifically, the EEG power density of higher frequency bands (theta, beta, and gamma) increased significantly during non-rapid eye movement (NREM) sleep. No statistically significant changes were observed during rapid eye movement (REM) sleep. No differences were found between the groups for HRV and actigraphy measurements. Due to technical issues, actigraphy data were missing for 4 participants (n = 8) and PSG data were missing for 2 participants (n = 10). One participant exhibited cold symptoms during the fourth week, and their PIRS-20 data were excluded from the analysis (n = 11).

Conclusions:

Despite the small sample size, statistically significant results were obtained, indicating both subjective and objective deterioration in sleep quality, potentially reaching the threshold for clinical sleep disorders. The results suggest a large effect size (d = 0.75) compared to background EMF exposure. While the basic design was highlighted as a strength, the small sample size and technical issues leading to reduced usable data (n = 8-12) were acknowledged as major limitations. A follow-up study with a larger sample might detect potential effects with smaller effect sizes.

Editor's note:

Interpretation of these results in the context of the existing scientific literature is challenging because most studies of mobile phone effects on sleep are based on short-term exposures, simulated laboratory conditions, or epidemiological surveys. However, several reviews (Hamblin & Wood, 2002; Rubin et al., 2011; Zhang et al., 2017) have concluded that mobile phone EMFs can affect selective EEG power bands, especially when exposure occurs just before or during sleep. (RH)

References:

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Zhang J, Sumich A, Wang GY (2017): Acute effects of radiofrequency electromagnetic field emitted by mobile phone on brain function. Bioelectromagnetics, 38(5), 329-338. https://doi.org/https://doi.org/10.1002/bem.22052

5G and sleep EEG

5G radio-frequency-electromagnetic-field effects on the human sleep electroencephalogram

Sousouri G, Eicher C, D'Angelo RM, Billecocq M, Fussinger T, Studler M, Capstick M, Kuster N, Achermann P, Huber R, Landolt HP (2024): 5G Radio-Frequency-Electromagnetic-Field Effects on the Human Sleep Electroencephalogram: A Randomized Controlled Study in CACNA1C Genotyped Healthy Volunteers. MedRxiv. 2024:2024-12. https://doi.org/10.1101/2024.12.16.24319082

The introduction of 5G technology as the latest standard in mobile telecommunications has raised concerns about potential health effects. In particular, individuals who self-identify as electrohypersensitive are concerned about sleep disturbances, headaches, and related brain impairments due to exposure to radiofrequency electromagnetic fields (RF-EMF). While the long-term health effects of EMF exposure remain unclear, several independent studies have shown that 2G to 4G EMFs acutely alter electroencephalographic (EEG) oscillations in the ca. 9-16 Hz range during wakefulness and sleep.

Notably, repeated spectral increases in the EEG spindle frequency range (11-16 Hz) have been observed during non-rapid eye movement (NREM) sleep. Sleep spindles modulate interactions between the brain and the external environment, essentially dampening responsiveness to sensory stimuli—effectively isolating the brain from external disturbances during sleep. The pulse modulation of 2G to 4G EMF signals appears to be critical for inducing EEG changes during sleep. The high inter-individual variability and strong intra-individual stability of EMF-induced sleep EEG changes suggest a possible genetic predisposition.

Based on other research, the likely mechanism of EMF exposure effects on the brain involves the depolarization of neuronal membrane potentials, which activates voltage-gated calcium ion channels (Ca^{2+}), and leads to increased intracellular Ca^{2+} concentration. The influx of Ca^{2+} regulates processes such as hormone secretion, neurotransmitter release, gene transcription, and neuronal activity. RF-EMFs can activate L-type voltage-gated calcium channels (LTCC), which are associated with sleep quality and oscillatory EEG activity.

Different allelic variants of the CACNA1C gene, which encodes the α1C subunit of the L-type calcium channel, have been linked to prolonged sleep latency and reduced sleep quality. This subunit determines the voltage sensitivity and conductivity of LTCCs. LTCCs are expressed in nearly all types of neurons in the brain and regulate neuronal firing, learning, memory, addictive behaviors, and neural development.

The aim of this study was to investigate whether pre-sleep exposure to realistic, standardized 5G EMF signals affects the spectral properties of spindles in the NREM sleep EEG. In addition, the study aimed to determine whether EMF-induced changes are modulated by the rs7304986 variant of the CACNA1C gene (T/C or T/T alleles). A novel methodological approach, the Fitting Oscillations & One Over f (FOOOF) analysis, was used. This analysis provides a validated, intuitive method for reliable and meaningful extraction of individual spectral EEG features.

Study design and methods:

Thirty-four healthy, right-handed volunteers, predominantly women, were enrolled in this study and genotyped for rs7304986 (15 T/C carriers and 19 T/T carriers). Participants completed questionnaires regarding mobile phone use, medication use, sleep behavior, and general and neurological health status.

All participants underwent three experimental nights with different standardized exposure conditions in a randomized, double-blind crossover design:

- 30-minute exposure before bedtime to active 5G EMF at a 700 MHz carrier frequency, 20 MHz bandwidth, and 12.5 Hz power control;
- 30-minute exposure before bedtime to active 5G EMF at a 3.6 GHz carrier frequency, 100 MHz bandwidth, and 12.5 Hz power control;
- 30-minute sham exposure with no active field.

The exposure system (sXh5G, IT'IS Foundation, Zurich, Switzerland) was calibrated to ensure that the specific absorption rate (SAR) for the head did not exceed 2 W/kg. The output power was 4.28 W at 700 MHz and 1.63 W for the 3.6 GHz signal. Identical power control was applied to both signals, introducing low frequency amplitude modulation at 12.5 Hz. Peak exposures were concentrated in the cortical tissue closest to the antenna, with a much steeper SAR decay at the higher frequency.

For EEG recordings, the researchers used 128-channel Electrical Geodesics sensor nets for overnight high-density EEG (hd-EEG) monitoring (Electrical Geodesics Inc., EGI, Eugene, OR).

Results:

T/C carriers reported longer sleep latency compared to T/T carriers. Statistical analysis of nighttime sleep variables revealed an interaction between "exposure" and "genotype" for later sleep stages, although post hoc t-tests showed no significant differences. Further analysis identified distinct negative and positive peaks in specific power ratios, suggesting a shift in spindle peak frequency rather than a general increase in spectral power density. When analyzing the periodic components of oscillatory spindle activity in NREM sleep EEG, a significant interaction between "exposure" and "genotype" was found for the center frequency of sleep spindle activity.

Finally, a topographical comparison (i.e., high-density EEG analysis) showed a widespread shift toward higher spindle frequencies in T/C allele carriers after exposure to the 3.6 GHz field. This effect covered a large cluster of central, parietal, and occip-

ital cortical areas in 50 out of 109 EEG channels. The percentage increase in spindle center frequency was 1.43 ± 6.5 × 10^{-4} %, corresponding to an average shift from 13.62 ± 0.1 Hz in the sham condition to 13.82 ± 0.1 Hz after 3.6 GHz exposure. The acceleration of spindle center frequency in T/C genotype participants after 3.6 GHz exposure was consistent, with a large effect size (Cohen's d mean ± SD = 0.78 ± 0.18; Cohen's d [min, max] = [0.28, 1.28]; Cohen's d > 0.57 in 48 out of 50 channels).

Conclusions:

Using the recently developed FOOOF algorithm, this study identified 5G-induced changes in spindle peak components in the NREM sleep EEG. A significant interaction was found between exposure and the genetic variant in the center frequency of sleep spindles.

The researchers observed a widespread shift in the center frequency of sleep spindles toward faster oscillatory activity in T/C allele carriers after exposure to a 5G RF-EMF with a carrier frequency of 3.6 GHz, implicating L-type voltage-gated calcium channels in the physiological response to RF-EMF. A smaller shift was also observed after exposure to the 700 MHz signal. If present, the effect of the lower frequency 700 MHz field is only marginally detectable with the current methodology. The discrepancy between the deeper penetration of the 700 MHz signal (as indicated by the simulated SAR distribution in the brain) and the more pronounced effects on sleep spindles observed after 3.6 GHz exposure remains unclear.

These results highlight the need for a comprehensive investigation into the complex properties of the new 5G signals. Knoblauch et al. (2005) demonstrated the circadian regulation of spindle center frequency, showing a decrease (from about 13.85 to 13.7 Hz) coinciding with melatonin secretion. Since an increase in spindle center frequency was observed after 5G exposure in this study, a circadian effect of RF-EMFs, such as reduced melatonin production, cannot be ruled out (although melatonin levels were not measured in this study).

These findings provide preliminary evidence that the LTCC type Cav1.2 may play a mechanistic role in the interaction between EMF exposure and the human brain. This hypothesis could be further tested by examining the effects of RF-EMF on sleep EEG after selective pharmacological modulation of these channels. (Furthermore, only individuals with the higher-sensitivity LTCC variant were susceptible to EMF exposure, suggesting that genetic factors play a role in electrosensitivity, editor's note.) (AT)

Mobile phones and hearing loss

High-frequency hearing loss amongst smart mobile phone users

Jha I, Alam MK, Kumar C, Sinha N, Kumar T (2024): High-Frequency Hearing Loss Amongst Smart Mobile Phone Users: A Case-Control Study. Annals of African Medicine. 2024 Oct 1;23(4):684-7. https://doi.org/10.4103/aam.aam_93_24

Over the past 20 years, the number of mobile phone users has surged from 12.4 million to approximately 5.6 billion, representing about 70 % of the world's population. Mobile phones can cause headaches, altered sensory perception, sleep disturbances, and a warming sensation around the ear, and devices held close to the ear can potentially damage hearing.

Brainstem Evoked Response Audiometry (BERA) is a non-invasive test that records brainstem potentials in response to click stimuli delivered through headphones. Surface electrodes are placed on the mastoid and the scalp vertex to measure these responses. BERA is used to diagnose defects along the auditory pathway, from the eighth cranial nerve to the auditory cortex.

The purpose of this study was to investigate the effects of smartphone exposure on the central auditory pathway using BERA.

Study design and methods:

Sixty individuals between the ages of 18 and 30 years were included in the study. Exclusion criteria: Individuals with epilepsy, smokers, those with a history of ear trauma or noise-induced hearing loss, users of neuroleptics, antidepressants, or furosemide, COVID-positive individuals, drug users, and those with chronic conditions such as diabetes, hypertension, arrhythmias, or a family history of hearing disorders were excluded. The participants were divided into two groups: Group I (n = 30): Individuals who had used smartphones for 1-5 years, averaging more than 2 hours per day. Group II (n = 30): Individuals who had used smartphones for more than 5 years, averaging more than 2 hours per day. The hypothesis was that BERA waves and interpeak latency (IPL) would be delayed in individuals with prolonged smartphone use. (IPL refers to the delay between peaks I and III or peaks III and V in the BERA test, typically around 2 ms. Peak I corresponds to the auditory nerve, while peaks III and V represent subsequent brainstem structures, editor's note.) Disk electrodes were placed on the mastoid or earlobe as the active electrode, the reference electrode on the vertex of the skull, and the ground electrode on the midline of the forehead. The right ear was tested, while the opposite ear was masked with 40 dB white noise. Acoustic click stimuli were delivered through headphones at 80 dB at frequencies of 4, 6, and 8 kHz. Two recordings were made to reproduce BERA waves I-VII and the interpeak latencies.

Results:

BERA waves III and V were significantly delayed in Group II (> 5 years of smartphone use), indicating potential damage to brainstem structures involved in auditory processing due to electromagnetic exposure. At 4 kHz, significant delays were observed in waves III and V, as well as in the interpeak latencies I-III and I-V. At 6 kHz, significant delays ($p < 0.05$) were noted in waves III and V, along with highly significant interpeak latency prolongations for I-III and I-V. At 8 kHz, all parameters showed highly significant delays compared to 4 kHz in Group II. The percentage of participants in Group II with delayed BERA waves III and V was: 60 % at 4 kHz, 70 % at 6 kHz, 83.33 % at 8 kHz.

Conclusions:

The results suggest that BERA can detect not only the harmful effects of electromagnetic radiation on the hearing of mobile phone users, but also an early involvement of the central nervous system. This effect is particularly pronounced when higher frequencies, such as 8 kHz, are used as auditory stimuli.

These findings are supported by previous research. Philip (2017) reported high-frequency hearing loss in mobile phone users using otoacoustic emissions testing, indicating damage to outer hair cells, especially in the basal turn of the cochlea. Similar trends have been observed by Kothari (2020) and Kellényi (1999). The authors recommend limiting the use of mobile phones for extended periods of time, particularly avoiding holding the phone close to the ear. This may significantly improve physiological and psychological health outcomes in the long term. (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
Email: louis@microwavenews.com

Prof. Joel Moskowitz, Director of the Center for Family and Community Health, School of Public Health, Berkeley (USA):
Institute Website: publichealth.berkeley.edu/people/joel-moskowitz
EMF Website: www.saferemr.com

Prof. Devra Davis (USA):
Website: ehtrust.org
Email: info@ehtrust.org

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

Databases:

www.emfdata.org
www.emf-portal.de
www.orsaa.org

Thank you, Isabel Wilke

The graduate biologist Isabel Wilke (Dipl.-Biol.) led the editorial team of the ElektrosmogReport for nearly 20 years, from 2006 to 2024, and authored hundreds of study reviews. It is largely thanks to her that the body of research on the risks of non-ionizing radiation has been documented and made widely known.

In an interview with diagnose:funk Magazin kompakt, Isabel Wilke stated, "After thirty years of evaluating the research, I can say that non-ionizing radiation is harmful to health – even in the non-thermal range," and added, "The topic of radiation is like a red thread through my life. Even as a child, I knew from my father – a high-ranking officer in the Air Force – that radioactive radiation is harmful and that high-energy, ionizing radiation can cause tissue damage. During my training as a medical technician, I learned about the harmful effects of X-rays on living organisms, and later, in graduate school, I learned more about what ionizing radiation can do to DNA – for example, forming thymine dimers in the DNA strand, which prevents it from being read correctly. In genetics, ionizing radiation is deliberately used to select the appropriate mutations for certain research questions. A quote from medicine that has always stuck with me is, 'In principle, a single ray is enough to cause a mutation that can lead to the development of cancer.'"

Wilke, who had been working at the Katalyse Institut in Cologne since 1994, began researching non-ionizing radiation in response to an increasing number of inquiries received through the consumer hotline about electrosmog. In 2002, she wrote the book Das große Strahlen - Handy & Co, published by the Katalyse Institut – a fundamental work that already analyzed all the risks. For the ElektrosmogReport, she reviewed hundreds of studies and became one of the most knowledgeable experts on both the state of EMF research and on the intense debate over the interpretation of study results. Time and again, she condemned the influence of industry on research in pointed comments in the ElektrosmogReport, fearlessly naming institutes and scientists affiliated with industry.

Because her work is so valuable, diagnose:funk has scanned every issue of ElektrosmogReport from 1995 to the present and archived them on the EMF:data website. Since about 2014, Isabel Wilke has also been a scientific advisor for diagnose:funk, and in 2018 she produced for us the most comprehensive WLAN review to date. Isabel Wilke is now taking a well-deserved retirement. We warmly thank her for her dedication and outstanding work – she will continue to advise us. The biologist Roman Heeren and the environmental scientist Alain Thill now form the editorial team of the ElektrosmogReport.

To learn more about the history of the ElektrosmogReport, read the interview with Isabel Wilke at www.diagnose-funk.org/2003.

The ElektrosmogReport:

An indispensable contribution to raising awareness about electromagnetic radiation

The ElektrosmogReport has a long history as an independent specialized information service on the significance of electric and magnetic fields for the environment and health. Its origins go back to Strahlentelex, a German information service on radioactivity, radiation, and health that was published from January 1987 to 2019. The section ElektrosmogReport developed into an important source of information and scientific discussion on the effects of non-ionizing radiation. In 2019, diagnose:funk took over the publication of the ElektrosmogReport. The diagnose:-funk database, www.emfdata.org, is based on the evaluations of the ElektrosmogReport and currently documents more than 700 studies and 520 reviews. With the English edition of the ElektrosmogReport starting with issue 4/2024, diagnose:funk expands the access to current research findings for an international audience.

This is a significant step to stimulate a global debate on the risks of electromagnetic fields. For more information and to download the latest issues, please visit the diagnose:funk website at kurzlinks.de/t3kf. The ElektrosmogReport archive is available at emfdata.org.