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SCIENCE

Low Intensity Electromagnetic Fields Act *via* Voltage-Gated Calcium Channel (VGCC) Activation to Cause Very Early Onset Alzheimer's Disease: 18 Distinct Types of Evidence

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Abstract: Electronically generated electromagnetic fields (EMFs), including those used in wireless communication such as cell phones, Wi-Fi and smart meters, are coherent, producing very high electric and magnetic forces, which act on the voltage sensor of voltage-gated calcium channels to produce increases in intracellular calcium $[Ca^{2+}]_i$. The calcium hypothesis of Alzheimer's disease (AD) has shown that each of the important AD-specific and nonspecific causal elements is produced by excessive $[Ca^{2+}]_i$. $[Ca^{2+}]_i$ acts in AD *via* excessive calcium signaling and the peroxynitrite/oxidative stress/inflammation pathway, which are each elevated by EMFs. An apparent vicious cycle in AD involves amyloid-beta protein ($A\beta$) and $[Ca^{2+}]_i$. Three types of epidemiology suggest EMF causation of AD, including early onset AD. Extensive animal model studies show that low intensity EMFs cause neurodegeneration, including AD, with AD animals having elevated levels of $A\beta$, amyloid precursor protein and BACE1. Rats exposed to pulsed EMFs every day are reported to develop universal or near universal very early onset neurodegeneration, including AD; these findings are superficially similar to humans with digital dementia. EMFs producing modest increases in $[Ca^{2+}]_i$ can also produce protective, therapeutic effects. The therapeutic pathway and peroxynitrite pathway inhibit each other. A summary of 18 different findings is provided, which collectively provide powerful evidence for EMF causation of AD. The author is concerned that smarter, more highly pulsed "smart" wireless communication may cause widespread very, very early onset AD in human populations.

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REVIEW



Genetic effects of non-ionizing electromagnetic fields

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ABSTRACT

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

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Article

The Effect of Continuous Low-Intensity Exposure to Electromagnetic Fields from Radio Base Stations to Cancer Mortality in Brazil

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Abstract: Background: this study aims to estimate the rate of death by cancer as a result of Radio Base Station (RBS) radiofrequency exposure, especially for breast, cervix, lung, and esophagus cancers. Methods: we collected information on the number of deaths by cancer, gender, age group, gross domestic product per capita, death year, and the amount of exposure over a lifetime. We investigated all cancer types and some specific types (breast, cervix, lung, and esophagus cancers). Results: in capitals where RBS radiofrequency exposure was higher than 2000/antennas-year, the average mortality rate was 112/100,000 for all cancers. The adjusted analysis showed that, the higher the exposure to RBS radiofrequency, the higher cancer mortality was. The highest adjusted risk was observed for cervix cancer (rate ratio = 2.18). The spatial analysis showed that the highest RBS radiofrequency exposure was observed in a city in southern Brazil that also showed the highest mortality rate for all types of cancer and specifically for lung and breast cancer. Conclusion: the balance of our results indicates that exposure to radiofrequency electromagnetic fields from RBS increases the rate of death for all types of cancer.



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Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission

ABSTRACT

Background: In 2011, IARC classified radiofrequency radiation (RFR) as possible human carcinogen (Group 2B). According to IARC, animals studies, as well as epidemiological ones, showed limited evidence of carcinogenicity. In 2016, the NTP published the first results of its long-term bioassays on near field RFR, reporting increased incidence of malignant glial tumors of the brain and heart Schwannoma in rats exposed to GSM – and CDMA – modulated cell phone RFR. The tumors observed in the NTP study are of the type similar to the ones observed in some epidemiological studies of cell phone users.

Objectives: The Ramazzini Institute (RI) performed a life-span carcinogenic study on Sprague-Dawley rats to evaluate the carcinogenic effects of RFR in the situation of far field, reproducing the environmental exposure to RFR generated by 1.8 GHz GSM antenna of the radio base stations of mobile phone. This is the largest long-term study ever performed in rats on the health effects of RFR, including 2448 animals. In this article, we reported the final results regarding brain and heart tumors.

Methods: Male and female Sprague-Dawley rats were exposed from prenatal life until natural death to a 1.8 GHz GSM far field of 0, 5, 25, 50 V/m with a whole-body exposure for 19 h/day.

Results: A statistically significant increase in the incidence of heart Schwannomas was observed in treated male rats at the highest dose (50 V/m). Furthermore, an increase in the incidence of heart Schwann cells hyperplasia was observed in treated male and female rats at the highest dose (50 V/m), although this was not statistically significant. An increase in the incidence of malignant glial tumors was observed in treated female rats at the highest dose (50 V/m), although not statistically significant.

Conclusions: The RI findings on far field exposure to RFR are consistent with and reinforce the results of the NTP study on near field exposure, as both reported an increase in the incidence of tumors of the brain and heart in RFR-exposed Sprague-Dawley rats. These tumors are of the same histotype of those observed in some epidemiological studies on cell phone users. These experimental studies provide sufficient evidence to call for the re-evaluation of IARC conclusions regarding the carcinogenic potential of RFR in humans.