

Electromagnetic Radiation and Autism: 2015

D.Klinghardt, London 2015

Electrosmog and it's Destructive Effect on the Brain,
our Genome, Proteome and Microbiome of the Unborn Child

Electrosmog refers to our exposure to the sum total of all man-made electric fields,
magnetic fields, radiowaves, TV broadcasting, home lighting and all other sources of
radiation

Mediators of Inflammation

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Clinical Study

Metabolic and Genetic Screening of Electromagnetic Hypersensitive Subjects as a Feasible Tool for Diagnostics and Intervention

[Chiara De Luca](#),^{1,2} [Jeffrey Chung Sheun Thai](#),³ [Desanka Raskovic](#),⁴ [Eleonora Cesareo](#),⁴
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Clinical Study

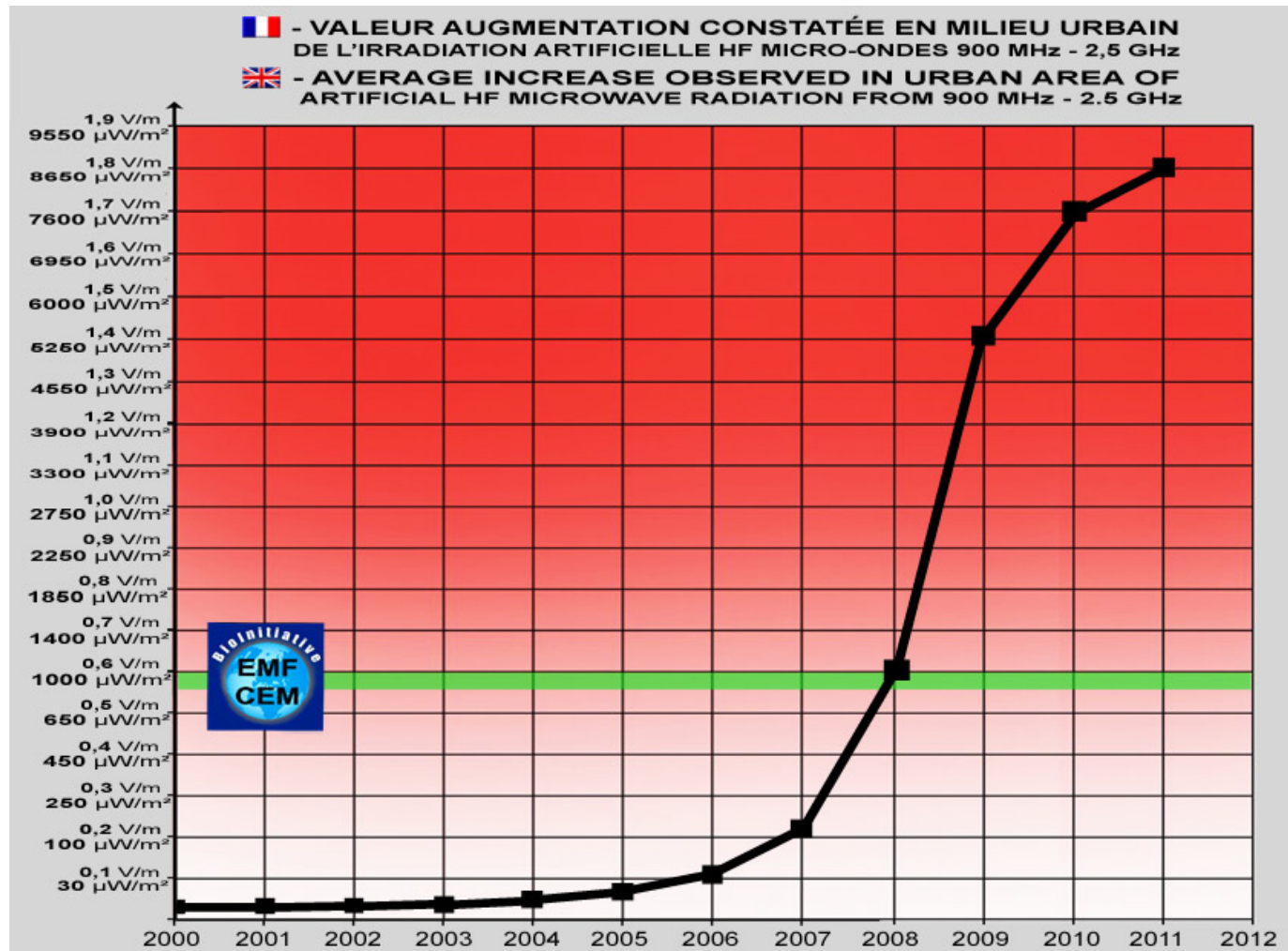
Metabolic and Genetic Screening of Electromagnetic Hypersensitive Subjects as a Feasible Tool for Diagnostics and Intervention

[Chiara De Luca](#),^{1,2} [Jeffrey Chung Sheun Thai](#),³ [Desanka Raskovic](#),⁴ [Eleonora Cesareo](#),⁴ [Daniela Caccamo](#),⁵ [Arseny Trukhanov](#),² and [Liudmila Korkina](#)^{1,2}

Abstract

Growing numbers of “electromagnetic hypersensitive” (EHS) people worldwide self-report severely disabling, multiorgan, non-specific symptoms when exposed to low-dose electromagnetic radiations, often associated with symptoms of multiple chemical sensitivity (MCS) and/or other environmental “sensitivity-related illnesses” (SRI). This cluster of chronic inflammatory disorders still lacks validated pathogenetic mechanism, diagnostic biomarkers, and management guidelines. We hypothesized that SRI, not being merely psychogenic, may share organic determinants of impaired detoxification of common physic-chemical stressors. Based on our previous MCS studies, we tested a panel of 12 metabolic blood redox-related parameters and of selected drug-metabolizing-enzyme gene polymorphisms, on 153 EHS, 147 MCS, and 132 control Italians, confirming MCS altered (p less than 0.05 – 0.0001) glutathione-(GSH), GSH-peroxidase/S-transferase, and catalase erythrocyte activities. We first described comparable—though milder—metabolic pro-oxidant/proinflammatory alterations in EHS with distinctively increased plasma coenzyme-Q₁₀ oxidation ratio. Severe depletion of erythrocyte membrane polyunsaturated fatty acids with increased ω 6/ ω 3 ratio was confirmed in MCS, but not in EHS. We also identified significantly ($p=0.003$) altered distribution-versus-control of the CYP2C19*1/*2 SNP variants in EHS, and a 9.7-fold increased risk (OR: 95% C.I=13–74.5) of developing EHS for the haplotype (null)GSTT1 + (null)GSTM1 variants. Altogether, results on MCS and EHS strengthen our proposal to adopt this blood metabolic/genetic biomarkers’ panel as suitable diagnostic tool for SRI.

Growth in Exposure to Microwave Radiation 2000-2010



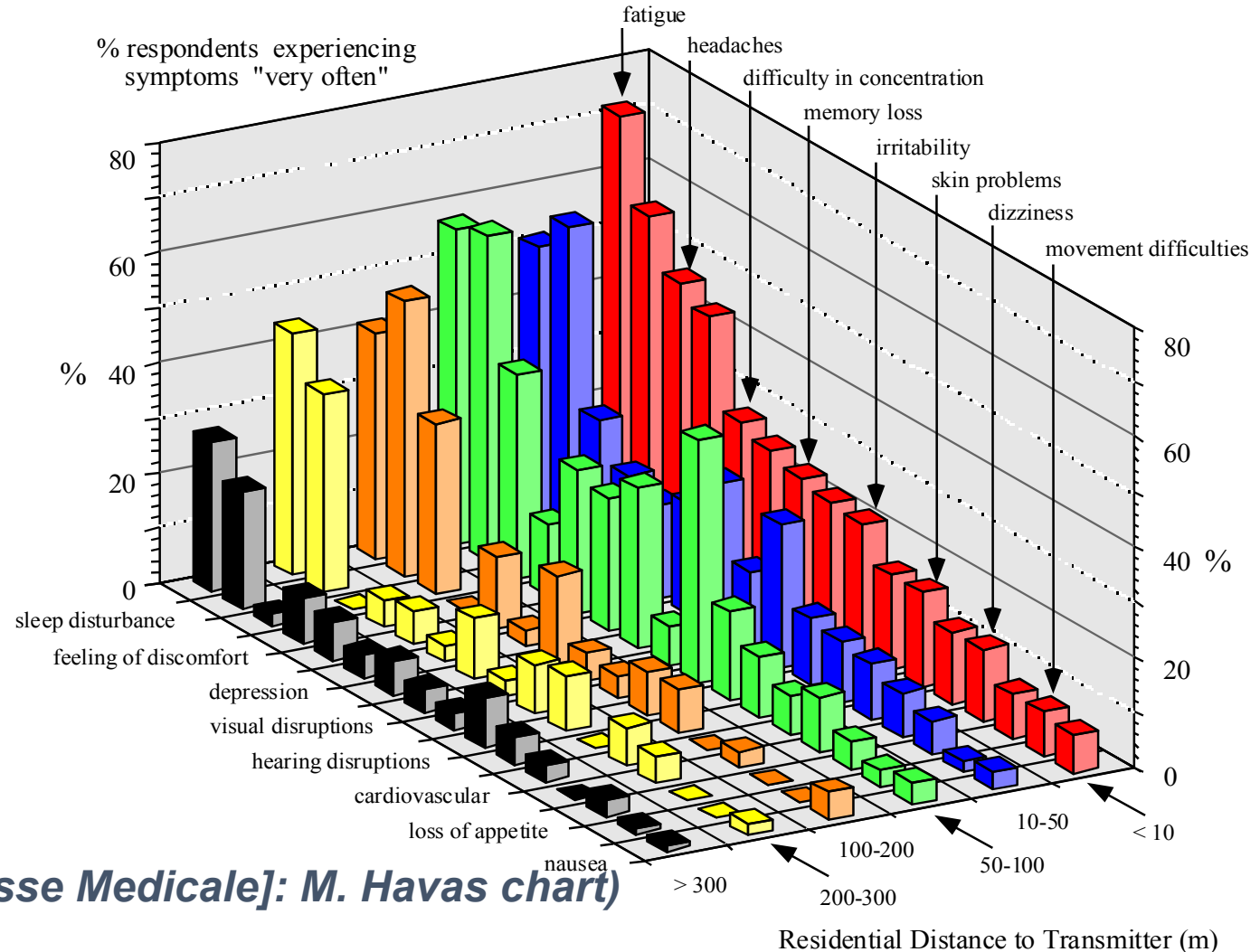
2001 Spain

Frequency of Electromagnetic Hypersensitivity Symptoms Based on Distance to Cell Phone Base Station

Electro-Hyper-Sensitivity (EHS)

1. Fatigue *
2. Sleep disturbance *
3. Headaches
4. Feeling of discomfort
5. Difficulty concentrating *
6. Depression *
7. Memory loss *
8. Visual disruptions *
9. Irritability *
10. Hearing disruptions *
11. Skin problems *
12. Cardiovascular *
13. Dizziness *
14. Loss of appetite *
15. Movement difficulties *
16. Nausea

* Associated with Aging:
"Rapid Aging Syndrome"



[Santini 2001, La Presse Medicale]: M. Havas chart)

“Brain proteome response following whole body exposure of mice to mobile phone or wireless DECT base radiation”

Electromagnetic Biology and Medicine; Posted online on January 20, 2012.

(doi:10.3109/15368378.2011.631068 (1–25) Adamantia F. Fragopoulou, Athina Samara, Marianna H. Antonelou, Anta Xanthopoulou, Aggeliki Papadopoulou, Konstantinos Vougas, Eugenia Koutsogiannopoulou, Ema Anastasiadou, Dimitrios J. Stravopodis, George Th. Tsangaris, Lukas H. Margaritis Department of Cell Biology and Biophysics, Athens University

Abstract:

The objective of this study was to investigate the effects of two sources of electromagnetic fields (EMFs) on the proteome of cerebellum, hippocampus, and frontal lobe in Balb/c mice following long-term whole body irradiation. Three equally divided groups of animals (6 animals/group) were used; the **first** group was exposed to a **typical mobile phone**, at a SAR level range of 0.17–0.37 W/kg for 3 h daily for 8 months, the **second** group was exposed to a **wireless DECT base** (Digital Enhanced Cordless Telecommunications/Telephone) at a SAR level range of 0.012–0.028 W/kg for 8 h/day also for 8 months and the **third** group comprised the **sham**-exposed animals. Comparative proteomics analysis revealed that long-term irradiation from **both EMF sources** altered significantly ($p < 0.05$) the **expression of 143 proteins** in total (as low as 0.003 fold downregulation **up to 114 fold overexpression**). Several neural function related proteins (i.e., Glial Fibrillary Acidic Protein (GFAP), Alpha synuclein, Glia Maturation Factor beta (GMF), and apolipoprotein E (apoE)), heat shock proteins, and cytoskeletal proteins (i.e., Neurofilaments and tropomodulin) are included in this list as well as proteins of the brain metabolism (i.e., Aspartate aminotransferase, Glutamate dehydrogenase) to nearly all brain regions studied. Western blot analysis on selected proteins confirmed the proteomics data. The observed **protein expression changes may be related to brain plasticity** alterations, indicative of **oxidative stress in the nervous system** or involved in **apoptosis** and might potentially explain human health hazards reported so far, such as **headaches, sleep disturbance, fatigue, memory deficits, and brain tumor long-term induction** under similar exposure conditions.

Wireless Radiation in the Etiology and Treatment of Autism: Clinical Observations and Mechanisms

J. Aust. Coll. Nutr. & Env. Med. Vol. 26 No.2 (August 2007) pages 3-7

Tamara J Mariea and George L Carlo

• Results

The sentinel subject's history suggested that the **efficiency of heavy metal detoxification was dramatically increased when EMR was eliminated**. For the larger groups, data indicated that heavy metals were cleared in a time and molecular weight-dependent manner after EMR was eliminated from the treatment environment.

• Conclusions

The findings suggest a significant **role of EMR in both the etiology of Autism and the efficacy of therapeutic interventions**. The mechanism of EMR impact could be direct by facilitating early clinical onset of symptoms or indirect, including **trapping heavy metals in cells** and both accelerating the onset of symptoms caused by heavy metal toxicity as well as impeding therapeutic clearance. These data also suggest that wireless device EMR is a synergen in the etiology of Autism, acting in conjunction with environmental and genetic factors, and offer a mechanistic explanation for the correlation between concurrent increases in the incidence of Autism and the use of wireless technology.

A Possible Association Between Fetal/Neonatal Exposure to Radiofrequency Electromagnetic Radiation and the Increased Incidence of Autism Spectrum Disorders (ASD).

Medical Hypotheses, Eden Press, New York. USA (2004); R.C. Kane

<http://linkinghub.elsevier.com.proxy.healwa.org/retrieve/pii/S0306987703003098?showall=true>

Abstract

Recently disclosed epidemiological data indicate a dramatic increase in the incidence of autism spectrum disorders. Previously, the incidence of autism has been reported as 4-5 per 10000 children. The most recent evidence indicates an increased incidence of about 1 per 500 children. However, the etiology of autism is yet to be determined. The recently disclosed data suggest a possible correlation between autism incidence and a previously unconsidered environmental toxin. It is generally accepted in the scientific community that radiofrequency (RF) radiation is a biologically active substance. It is also readily acknowledged that human exposures to RF radiation have become pervasive during the past 20 years, whereas such exposures were uncommon prior to that time.

It is suggested that fetal or neo-natal exposures to RF radiation may be associated with an increased incidence of autism

“Out of Time: A Possible Link Between Mirror Neurons, Autism and Electromagnetic Radiation”

Medical Hypotheses; I.M. Thornton, Eden Press, New York. USA (2006)

<http://linkinghub.elsevier.com.proxy.heal-wa.org/retrieve/pii/S0306987706000934?showall=true>

Abstract

Recent evidence suggests a link between autism and the human mirror neuron system. In this paper, I argue that **temporal disruption from the environment** may **play an important role in the observed mirror neuron dysfunction**, leading in turn to the pattern of deficits associated with autism. I suggest that the developing nervous system of an infant may be particularly prone to temporal noise that can interfere with the initial calibration of brain networks such as the mirror neuron system. **The most likely source of temporal noise in the environment is artificially generated electromagnetic radiation.** To date, there has been little evidence that electromagnetic radiation poses a direct biological hazard. It is clear, however, that time-varying electromagnetic waves have the potential to temporally modulate the nervous system, particularly when populations of neurons are required to act together. This modulation may be completely harmless for the fully developed nervous system of an adult. For an infant, this same temporal disruption might act to severely delay or disrupt vital calibration processes.

Extremely-Low Frequency (ELF) and Radiofrequency (RF) Electromagnetic Fields Have Very Similar Biological Effects

- **Genetic Effects**
- **Cancer**
- **Cellular/Molecular Effects**
- **Electrophysiology**
- **Behavior**
- **Nervous System**
- **Blood-brain barrier**
- **Calcium**
- **Cardiovascular**
- **Warm sensation**
- **Hormones**
- **Immunology**
- **Metabolic rate/ effects**
- **Reproduction/ growth**
- **Subjective symptoms**
- **Stress**

Source: Dr. Henry Lai, Research Professor, Department of Bioengineering, University of Washington. Presentation March 21, 2008 at Council on Wireless Technology Impacts EMF Panel, San Francisco, CA.

Melatonin clears the brain at night of toxins

It is the most potent brain anti-oxidant and detox agent

1. Melatonin induces sleep. We only heal and detoxify in deep non-rem sleep. Without melatonin no regeneration and no detoxification
2. Melatonin is the most effective and potent neuroprotective chemical in the CNS and prevents damage from mercury, lead, aluminum, chemicals, mycotoxins, viruses, cigarette smoke, bacterial and parasitic endo- and exotoxins (Lyme, clostridia, ascaris) outgassing of carpets and new car plastics, etc.
 - Sener, G. et al: **"Melatonin protects against mercury induced oxidative tissue damage"**. *Basic and Clinical Pharmacology & Toxicology* Vol 93, Dec 2003, pp 290-296
 - L. Xie, H. Kang, Q. Xu, M. J. Chen, Y. Liao, M. Thiyagarajan, J. O'Donnell, D. J. Christensen, C. Nicholson, J. J. Iliff, T. Takano, R. Deane, M. Nedergaard. **"Sleep Drives Metabolite Clearance from the Adult Brain"**. *Science*, 2013; 342 (6156): 373 DOI:[10.1126/science.1241224](https://doi.org/10.1126/science.1241224)

[Curr Neuropharmacol](#). 2008 Sep;6(3):203-14. doi: 10.2174/157015908785777201.

Cellular and biochemical actions of melatonin which protect against free radicals: role in neurodegenerative disorders.

[Ortiz GG1](#), [Benítez-King GA](#), [Rosales-Corral SAPacheco-Moisés FP](#), [Velázquez-Brizuela IE.](#),

Abstract

Molecular oxygen is toxic for anaerobic organisms but it is also obvious that oxygen is poisonous to aerobic organisms as well, since oxygen plays an essential role for inducing molecular damage. Molecular oxygen is a triplet radical in its ground-stage ($\cdot\text{O}-\text{O}\cdot$) and has two unpaired electrons that can undergoes consecutive reductions of one electron and generates other more reactive forms of oxygen known as free radicals and reactive oxygen species. These reactants (including superoxide radicals, hydroxyl radicals) possess variable degrees of toxicity. Nitric oxide (NO^{\cdot}) contains one unpaired electron and is, therefore, a radical. NO^{\cdot} is generated in biological tissues by specific nitric oxide synthases and acts as an important biological signal. Excessive nitric oxide production, under pathological conditions, leads to detrimental effects of this molecule on tissues, which can be attributed to its diffusion-limited reaction with superoxide to form the powerful and toxic oxidant, peroxynitrite. Reactive oxygen and nitrogen species are molecular "renegades"; these highly unstable products tend to react rapidly with adjacent molecules, donating, abstracting, or even sharing their outer orbital electron(s). This reaction not only changes the target molecule, but often passes the unpaired electron along to the target, generating a second free radical, which can then go on to react with a new target amplifying their effects. This review describes the mechanisms of oxidative damage and its relationship with the most highly studied neurodegenerative diseases and the roles of melatonin as free radical scavenger and neurocytoskeletal protector.

[Aging \(Milano\)](#). 1995 Oct;7(5):340-51.

Oxygen radical detoxification processes during aging: the functional importance of melatonin.

[Reiter RJ](#)

Abstract

That free radical destruction of macromolecules is a basis of aging and age-related diseases has considerable experimental support. Melatonin, a hormone produced in organisms as diverse as algae and humans, is believed to have evolved coincident with aerobic metabolism. In all organisms melatonin is produced primarily during the daily period of darkness, with only small amounts being synthesized during the day. In mammals including man, melatonin is produced by and secreted from the pineal gland during the night; however, the night-time production of melatonin falls markedly with aging such that in senescent animals a night-time melatonin rise is barely measurable. This may be significant in terms of aging in the light of recent observations which show that melatonin is a highly efficient free radical scavenger and antioxidant both in vitro and in vivo. In vitro, melatonin has been shown to directly scavenge both the hydroxyl and peroxy radical, and it does so more efficiently than other known antioxidants. Furthermore, melatonin greatly potentiates the efficiency of previously-discovered endogenous and exogenous antioxidants. In vivo, both physiological and pharmacological levels of melatonin reportedly counteract the devastatingly destructive actions of free radical generating chemicals. For example, **melatonin effectively combats DNA damage** in rats given massive doses of the chemical carcinogen safrole, and the indole overcomes much of the genomic damage inflicted by ionizing radiation. Also, **lipid peroxidation** induced by either paraquat, bacterial lipopolysaccharide or H₂O₂ is highly **significantly reduced by concurrent melatonin administration**. Finally, **cataracts** produced in newborn rats by the depletion of the endogenous antioxidant glutathione are **prevented by melatonin**. These findings provide evidence that **melatonin is operative in the cell nucleus**, in the aqueous cytosol and in lipid-rich cellular membranes **as an antioxidant**. Considering this, the loss of this potent antioxidant during aging may be consequential in terms of cellular and organismal aging as well as the onset of age-related diseases. These experimental results from a variety of sources suggest that a more determined approach to the study of melatonin as an anti-aging factor is warranted.

Mom's Cell phone use leads to decreased melatonin production and decreased protection of the fetus

Melatonin metabolite excretion among cellular telephone users

Int J Radiat Biol. 2002 Nov;78(11):1029-36 [Burch JB](#), [Reif JS](#), [Noonan CW](#), [Ichinose T](#), [Bachand AM](#), [Koleber TL](#), [Yost MG](#).

Department of Environmental and Radiological Health Sciences, Colorado State University, Fort Collins, CO

80523, USA. james.burch@colostate.edu

PURPOSE: The relationship between cellular telephone use and excretion of the melatonin metabolite 6-hydroxymelatonin sulfate (6-OHMS) was evaluated in two populations of male electric utility workers (Study 1, n=149; Study 2, n=77).

MATERIALS AND METHODS: Participants collected urine samples and recorded cellular telephone use over 3 consecutive workdays. Personal 60-Hz magnetic field (MF) and ambient light exposures were characterized on the same days using EMDEX II meters. A repeated measures analysis was used to assess the effects of cellular telephone use, alone and combined with MF exposures, after adjustment for age, participation month and light exposure. **RESULTS:** No change in 6-OHMS excretion was observed among those with daily cellular telephone use >25 min in Study 1 (5 worker-days). Study 2 workers with >25 min cellular telephone use per day (13 worker-days) had lower creatinine-adjusted mean nocturnal 6-OHMS concentrations (p=0.05) and overnight 6-OHMS excretion (p=0.03) compared with those without cellular telephone use. There was also a linear trend of decreasing mean nocturnal 6-OHMS/creatinine concentrations (p=0.02) and overnight 6-OHMS excretion (p=0.08) across categories of increasing cellular telephone use. A combined effect of cellular telephone use and occupational 60-Hz MF exposure in reducing 6-OHMS excretion was also observed in Study 2.

CONCLUSIONS: Exposure-related reductions in 6-OHMS excretion were observed in Study 2, where daily cellular telephone use of >25 min was more prevalent. **Prolonged use of cellular telephones may lead to reduced melatonin production, and elevated 60-Hz MF exposures may potentiate the effect.**

[Acta Trop.](#) 2014 Sep;137:31-8. doi: 10.1016/j.actatropica.2014.04.021. Epub 2014 May 6.

Effects of melatonin on oxidative stress, and resistance to bacterial, parasitic, and viral infections: a review.

[Vielma JR](#), [Bonilla E](#), [Chacín-Bonilla L](#), [Mora M](#), [Medina-Leendertz S](#), [Bravo Y](#)

Abstract

Melatonin, a hormone secreted by the pineal gland, works directly and indirectly as a free radical scavenger. Its other physiological or pharmacological activities could be dependent or independent of receptors located in different cells, organs, and tissues. In addition to its role in promoting sleep and circadian rhythms regulation, it has important immunomodulatory, antioxidant, and neuroprotective effects suggesting that this indole **must be considered as a therapeutic alternative against infections**. The aim of this review is to describe the effects of melatonin on oxidative stress and the resistance to bacterial (*Klebsiella pneumoniae*, *Helicobacter pylori*, *Mycobacterium tuberculosis*, and *Clostridium perfringens*), viral (Venezuelan equine encephalomyelitis virus and respiratory syncytial virus), and parasitic (*Plasmodium* spp., *Entamoeba histolytica*, *Trypanosoma cruzi*, *Toxoplasma gondii*, and *Opisthorchis viverrini*) infections.

[Int J Exp Pathol](#). 2015 Apr 19. doi: 10.1111/iep.12122. [Epub ahead of print]

The protective properties of melatonin against aluminium-induced neuronal injury.

[Al-Olayan EM1](#), [El-Khadragy MF](#), [Abdel Moneim AE](#)

Abstract

Aluminium (Al) toxicity is closely linked to the pathogenesis of Alzheimer's disease (AD). This experimental study investigated the neuroprotective effect of melatonin (Mel; 10 mg/kg bwt) on aluminium chloride (AlCl₃; 34 mg/kg bwt) induced neurotoxicity and oxidative stress in rats. Adult male albino Wistar rats were injected with AlCl₃ for 7 days. The effect on brain structure, lipid peroxidation (LPO), nitric oxide (NO) levels, glutathione (GSH) content, antioxidant enzymes (SOD, CAT, GPx and GR), apoptotic proteins (Bax and Bcl-2) and an apoptotic enzyme (caspase-3) was investigated. No apparent changes occurred following the injection of melatonin. **Melatonin** pre-treatment of the AlCl₃ -administered rats **reduced brain damage**, and the tissues appeared like those of the control rats. Compared to treatment with AlCl₃, pre-treatment with melatonin **decreased LPO and NO levels** and **increased the GSH content and antioxidant enzyme activity**. Moreover, melatonin increased the levels of the anti-apoptotic protein, Bcl-2, decreased the levels of the pro-apoptotic protein, Bax, and inhibited caspase-3 activity.

Therefore, our results indicate that melatonin may provide therapeutic value against aluminium-induced oxidative stress and histopathological alternations in the rat brain and that these effects may be related to anti-apoptotic and antioxidant activities.

[Int J Immunopharmacol.](#) 2000 Oct;22(10):821-32.

Influence of melatonin on immunotoxicity of lead.

[Kim YO](#)1, [Pyo MY](#), [Kim JH](#)

Abstract

The results suggested that immunotoxicity induced by lead [Pb, as Pb(NO₃)₂] was significantly restored or prevented by melatonin(MLT). MLT (10 or 50 mg/kg) was orally administered to ICR mice daily for 28 days, and Pb was also administered at 35 mg/kg in the same way 2 h after the administration of MLT, and the normal mice were given vehicle. Within the Pb plus MLT-treated group, the body weight gains and the relative thymus weights were significantly increased when compared with the treatment of Pb alone. The relative spleen and liver weights were increased by the treatment of Pb alone, and then restored to normal value by MLT treatment. Hemagglutination (HA) titer, plaque-forming cell response to sheep red blood cell (SRBC), and secondary IgG antibody response to BSA were significantly enhanced in the Pb plus MLT-treated mice, as opposed to when compared with the treatment of Pb alone. The mitogenic response of splenic T cell to concanavalin A and that of B cells to lipopolysaccharide was remarkably increased by MLT treatment when compared with treatment of Pb alone. Splenic CD4(+) cells were significantly increased by MLT treatment when compared with treatment of Pb alone. In case of CD8(+) cells, the slight enhancement was observed in MLT treatment. Splenic T and B cells were significantly increased by MLT treatment when compared with the treatment of Pb alone. The **natural killer cell, phagocytic activity** and the number of peripheral **leukocytes were significantly enhanced in Pb plus MLT-treated mice** when compared with the treatment of Pb alone.

[Int J Immunopharmacol.](#) 2000 Apr;22(4):275-84.

Influence of melatonin on immunotoxicity of cadmium.

[Kim YO](#), [Ahn YK](#), [Kim JH](#).

Abstract

- The results suggested that immunotoxicity induced by Cd was significantly restored or prevented by MLT. MLT (10 or 50 mg/kg) was orally administered to ICR mice daily for 28 consecutive days, and cadmium (Cd, as [Cd(AC)(2)]) was also administered at 25 mg/kg by the same route 2 h after the administration of MLT, and the normal mice were given vehicle. Within the Cd plus MLT-treated group, the body weight gains and relative thymus weights were significantly increased when compared with the treatment of Cd alone. The relative spleen and liver weights were increased by treatment of Cd alone, then restored to normal value by MLT treatment. Hemagglutination (HA) titer, primary IgM antibody response to SRBC, and secondary IgG antibody response to BSA was significantly increased with the Cd plus MLT-treated mice, as opposed to when compared with treatment of Cd alone. **The NK cell and phagocytic activity** used for evaluation of non-specific immunocompetence was **significantly increased** in Cd plus MLT-treated mice when compared with the treatment of Cd alone. The **number of peripheral leukocytes was significantly increased** in Cd plus MLT-treated mice when compared with treatment of Cd alone.

Body Voltage: the effect of ambient electricity at home on Melatonin

Melatonin and N-tert-butyl-alpha-phenylnitronone block 60-Hz magnetic field-induced DNA single and double strand breaks in rat brain cells

J Pineal Res. 1997 Apr; 22(3):152-62 [Lai H](#), [Singh NP](#).

Bioelectromagnetics Research Laboratory, Center for Bioengineering, University of Washington, Seattle 98195, USA. hlai@u.washington.edu

In previous research, we have found an increase in DNA single- and double-strand breaks in brain cells of rats after acute exposure (two hours) to a sinusoidal 60-Hz magnetic field. The present experiment was carried out to investigate whether treatment with melatonin and the spin-trap compound N-tert-butyl-alpha-phenylnitronone (PBN) could block the effect of magnetic fields on brain cell DNA. Rats were injected with melatonin (1 mg/kg, sc) or PBN (100 mg/kg, ip) immediately before and after two hours of exposure to a 60-Hz magnetic field at an intensity of 0.5 mT. We found that both drug treatments blocked the magnetic field-induced DNA single- and double-strand breaks in brain cells, as assayed by a microgel electrophoresis method.

Since melatonin and PBN are efficient free radical scavengers, these data suggest that free radicals may play a role in magnetic field-induced DNA damage

Parental exposure to 50Hz electromagnetic fields causes CNS damage of the fetus

Risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields: a population based study.

Occup Environ Med. 2002 Feb;59(2):92-7 [Blaasaas KG](#), [Tynes T](#), [Irgens A](#), [Lie RT](#).

National Institute of Occupational Health, Oslo, Norway. karl.g.blaasaas@nrpa.no

OBJECTIVES: To study the risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields.

METHODS: The Medical Birth Registry of Norway was linked with census data on parental occupation. An expert panel constructed a job exposure matrix of parental occupational exposure to 50 Hz magnetic fields. Exposure to magnetic fields was estimated by combining branch and occupation into one of three exposure levels: <4 hours, 4-24 hours, and >24 hours/week above approximately 0.1 mu T. Risks of 24 categories of birth defects were compared across exposure levels. Out of all 1.6 million births in Norway in the period 1967-95, 836,475 and 1,290,298 births had information on maternal and paternal exposure, respectively. Analyses were based on tests for trend and were adjusted for parents' educational level, place of birth, maternal age, and year of birth. **RESULTS:** The total risk of birth defects was not associated with parental exposure. Maternal exposure was associated with increased risks of spina bifida (p=0.04) and clubfoot (p=0.04). A negative association was found for isolated cleft palate (p=0.01). Paternal exposure was associated with increased risks of anencephaly (p=0.01) and a category of "other defects" (p=0.02).

CONCLUSION: The present study gives an indication of an association between selected disorders of the central nervous system and parental exposure to 50 Hz magnetic fields. Given the crude exposure assessment, lack of comparable studies, and the high number of outcomes considered, the results should be interpreted with caution.

“Extremely Low-Frequency Magnetic Field Decreased Calcium, Zinc and Magnesium Levels in Costa of Rat”

Abstract

Electromagnetic field (EMF) can affect cells due to biochemical change followed by a change in level of ions trafficking through membrane. We aimed to investigate possible changes in some elements in costa of rats exposed to long-term extremely low-frequency magnetic field (ELF-MF). Rats were exposed to 100 and 500 μ T ELF-MF, which are the safety standards of public and occupational exposure for 2 h/day during 10 months. At the end of the exposure period, the samples of costa were taken from the rats exposed to ELF-MF and sham. The levels of elements were measured by using atomic absorption spectrophotometry (AAS) and ultraviolet (UV) spectrophotometry. Ca levels decreased in the ELF-500 exposure group in comparison to sham group ($p < 0.05$). Statistically significant decrease was found in Mg levels in the ELF-500 exposure group in comparison to sham and ELF-100 exposure groups ($p < 0.05$). Zn levels were found to be lower in the ELF-500 exposure group than those in the sham and ELF-100 exposure groups ($p < 0.05$). No significant differences were determined between groups in terms of the levels of P, Cu and Fe.

In conclusion, it can be maintained that **long-term ELF-MF exposure can affect the chemical structure and metabolism of bone by changing the levels of some important elements such as Ca, Zn and Mg in rats.**

The damage caused by wireless devices

“Oxidative damage in the kidney induced by 900-MHz-emitted mobile phone: protection by melatonin”

Arch Med Res. 2005 Jul-Aug;36(4):350-5

[Oktem F](#), [Ozguner F](#), [Mollaoglu H](#), [Koyu A](#), [Uz E](#).

Department of Pediatric Nephrology, School of Medicine, Suleyman Demirel University, Isparta, Turkey.

BACKGROUND: The mobile phones emitting 900-MHz electromagnetic radiation (EMR) may be mainly absorbed by kidneys because they are often carried in belts. Melatonin, the chief secretory product of the pineal gland, was recently found to be a potent free radical scavenger and antioxidant. The aim of this study was to examine 900-MHz mobile phone-induced oxidative stress that promotes production of reactive oxygen species (ROS) on renal tubular damage and the role of melatonin on kidney tissue against possible oxidative damage in rats. **METHODS:** The animals were randomly grouped as follows: 1) sham-operated control group and 2) study groups: i) 900-MHz EMR exposed (30 min/day for 10 days) group and ii) 900-MHz EMR exposed+melatonin (100 microg kg⁻¹ s.c. before the daily EMR exposure) treated group. Malondialdehyde (MDA), an index of lipid peroxidation, and urine N-acetyl-beta-d-glucosaminidase (NAG), a marker of renal tubular damage were used as markers of oxidative stress-induced renal impairment. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status. **RESULTS:** In the EMR-exposed group, while tissue MDA and urine NAG levels increased, SOD, CAT, and GSH-Px activities were reduced. Melatonin treatment reversed these effects as well. In this study, the increase in MDA levels of renal tissue and in urine NAG and also the decrease in renal SOD, CAT, GSH-Px activities demonstrated the role of oxidative mechanism induced by 900-MHz mobile phone exposure, and melatonin, via its free radical scavenging and antioxidant properties, ameliorated oxidative tissue injury in rat kidney.

CONCLUSIONS: These results show that melatonin may exhibit a protective effect on mobile phone-induced renal impairment in rats.

Melatonin Reduces Oxidative Stress Induced by Chronic Exposure of Microwave Radiation from Mobile Phones in Rat Brain

[Dusan Sokolovic et al](#), Corresponding author: Phone: +381-642136478 Fax: +381-18238770 E-mail: soko@medfak.ni.ac.yu

Abstract

Purpose: The aim of the study was to evaluate the intensity of oxidative stress in the brain of animals chronically exposed to mobile phones and potential protective effects of melatonin in reducing oxidative stress and brain injury. **Materials and methods:** Experiments were performed on Wistar rats exposed to microwave radiation during 20, 40 and 60 days. Four groups were formed: I group (control)- animals treated by saline, intraperitoneally (i.p.) applied daily during follow up, II group (Mel)- rats treated daily with melatonin (2 mg kg⁻¹ body weight i.p.), III group (MWs)- microwave exposed rats, IV group (MWs + Mel)- MWs exposed rats treated with melatonin (2 mg kg⁻¹ body weight i.p.). The microwave radiation was produced by a mobile test phone (SAR = 0.043-0.135 W/kg). **Results:** A significant increase in the brain tissue malondialdehyde (MDA) and carbonyl group concentration was registered during exposure. Decreased activity of catalase (CAT) and increased activity of xanthine oxidase (XO) remained after 40 and 60 days of exposure to mobile phones. Melatonin treatment significantly prevented the increase in the MDA content and XO activity in the brain tissue after 40 days of exposure while it was unable to prevent the decrease of CAT activity and increase of carbonyl group contents.

Conclusion: We demonstrated two important findings; **that mobile phones caused oxidative damage** biochemically by increasing the levels of MDA, carbonyl groups, XO activity and decreasing CAT activity; and that treatment with the **melatonin significantly prevented oxidative damage in the brain.**

The damage to the CNS caused by wireless technologies: “Mobile phone radiation decreases pre-bedtime melatonin level”

Does evening exposure to mobile phone radiation affect subsequent melatonin production?

Int J Radiat Biol. 2006 Feb;82(2):69-76 [Wood AW](#), [Loughran SP](#), [Stough C](#)

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PURPOSE: To test whether exposure to the emissions from a digital mobile phone handset prior to sleep alters the secretion of melatonin. **MATERIALS AND METHODS:** In a double-blind cross-over design, 55 adult volunteers were both actively exposed or sham-exposed (in random order on successive Sunday nights) to mobile phone emissions for 30 min (0.25 W average power). Urine collection occurred immediately prior to retiring to bed and on rising the next morning. Melatonin output was estimated from principal metabolite concentrations (6-sulphatoxymelatonin (aMT6s) via radioimmunoassay), urine volumes and creatinine concentrations. **RESULTS:** Total melatonin metabolite output (concentration x urine volume) was unchanged between the two exposure conditions (active 14.1+/-1.1 microg; sham 14.6+/-1.3 microg). The pre- and post-bedtime outputs considered separately were also not significantly different, although the pre-bedtime value was less for active versus sham exposure.

When melatonin metabolite output was estimated from the ratio of aMT6s to creatinine concentrations, the ***pre-bedtime value was significantly less ($p = 0.037$) for active compared to sham.***

“Mobile phone-induced myocardial oxidative stress: protection by a novel antioxidant agent caffeic acid phenethyl ester”

Toxicol Ind Health. 2005 Oct;21(9):223-30

[Ozguner F](#), [Altinbas A](#), [Ozaydin M](#), [Dogan A](#), [Vural H](#), [Kisioglu AN](#), [Cesur G](#), [Yildirim NG](#)

Electromagnetic radiation (EMR) or radiofrequency fields of cellular mobile phones may affect biological systems by increasing free radicals, which appear mainly to enhance lipid peroxidation, and by changing the antioxidant defense systems of human tissues, thus leading to oxidative stress. Mobile phones are used in close proximity to the heart, therefore 900 MHz EMR emitting mobile phones may be absorbed by the heart. Caffeic acid phenethyl ester (CAPE), one of the major components of honeybee propolis, was recently found to be a potent free radical scavenger and antioxidant, and is used in folk medicine. The aim of this study was to examine 900 MHz mobile phone-induced oxidative stress that promotes production of reactive oxygen species (ROS) and the role of CAPE on myocardial tissue against possible oxidative damage in rats. Thirty rats were used in the study. Animals were randomly grouped as follows: sham-operated control group (N: 10) and experimental groups: (a) group II: 900 MHz EMR exposed group (N: 10); and (b) group III: 900 MHz EMR exposed+CAPE-treated group (N: 10). A 900 MHz EMR radiation was applied to groups II and III 30 min/day, for 10 days using an experimental exposure device. Malondialdehyde (MDA, an index of lipid peroxidation), and nitric oxide (NO, a marker of oxidative stress) were used as markers of oxidative stress-induced heart impairment. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status. In the EMR exposed group, while tissue MDA and NO levels increased, SOD, CAT and GSH-Px activities were reduced. CAPE treatment in group III reversed these effects. In this study, the increased levels of MDA and NO and the decreased levels of myocardial SOD, CAT and GSH-Px activities demonstrate the role of oxidative mechanisms in 900 MHz mobile phone-induced heart tissue damage, and CAPE, via its free radical scavenging and antioxidant properties, ameliorates oxidative heart injury.

These results show that CAPE from Propolis exhibits a protective effect on mobile phone-induced and free radical mediated oxidative heart impairment in rats.

How to make Liposomal melatonin

Ingredients: 1. LipoSorb Biopure.eu 2. Melatonin capsules with minimal fillers, we use BioTech or a compounding pharmacy 3. Organic Coconut oil 4. Organic Honey 5. Water, filtered 6. Blender; a Magic Bullet/NutriBullet works well 7. Ultrasonic jewelry cleaner (not the one that you use to clean your own jewelry with); we use [www. Biopure.eu](http://www.Biopure.eu) 8. Glass container like a custard cup or ramekin that fits inside the jewelry cleaner

Put into the blender, in order: 2 tsp water, 2 tablespoon room temperature coconut oil, 2 tsp Lipo-Health, 7-10 days worth of melatonin (open appropriate number of capsules into blender; see below for example calculation), and 1.5 tsphoney or to taste. Blend until well mixed. You want it to look a little gelatinous and thick enough that it still pours, but slowly. Add water as needed to make product blendable. Add coconut oil if too fluid. Put into a glass container in ultrasonic jewelry cleaner that is filled with water. Following instructions for cleaner, run for 20 minutes. Place in refrigerator, where it should thicken up to resemble butter. After it has thickened, cut into slices like a pie, so that each slice is your desired dose.

How to take: let it absorb through your mouth by moving it around to coat insides of cheeks, roof of mouth, etc. for several minutes. Avoid brushing teeth, eating, drinking, or taking other supplements for at least 15 minutes.

- This is more potent than non-liposomal melatonin. Start your dose low or as your doctor recommends. The sign of having taken too much is drowsiness the next day.
- Honey here also acts as an emulsifier, so it is preferred over other sweeteners
- Simple dosing calculation example: if your dose is 2mg/night, and you are going to cut the product into 8 slices (one slice/night), you need to add 16mg to the formula. If your melatonin is 5mg/capsule, open 3 capsules into the blender, for a total of 15mg/batch, or just under 2mg/dose.

Radioprotection for ASD kids

1. Daytime:

- No wireless in home, no cordless phones
- Child should wear radio protective clothing (BioPure.eu)
- **Rosemary tincture:** *“highly significant protective anti-mutagenic activity”. “Even the most powerful water-soluble antioxidants lack the capacity to protect against gamma ray induced damage”. (British Journal of Radiology, February 2 edition, 2015)*
- Using the **Stetzer filters** throughout home or school to decrease “dirty electricity”

2. Evening:

- **Liposomal Melatonin** (+ 50-100 mg DMSA for a few weeks)
- Trial with 5 HTP (adult dose: 200 mg)
- **Propolis tincture** 4-6 pipettes after dinner. A propolis compound (CAPE) protects lymphocytes against radiation (2008 **Journal of Biochemical and Molecular Toxicology**)
- TD-Magnesium, **Epsom salt baths** twice daily, oral Mag.glycinate. Magnesium act as calcium channel blocker. Voltage gate calcium channels are upregulated by EMR (M.Pall, 2013)

3. Nights:

- Sleep sanctuary, fuses off
- Consider Samina bed

Autism may be Linked to Electromagnetic Radiation Levels In Mother's Bedroom During Pregnancy

Pilot Study Finds Over 20x Higher Microwave Power Density Levels in Mothers' Sleeping Locations During Pregnancy

Dr. Dietrich Klinghardt, MD, PhD of the Sophia Health Institute in Woodinville, WA recently conducted a pilot study to assess the potential role of electromagnetic frequencies in the dramatic rise in autism and other neurological impairments over the past decade. Various measurements of electromagnetic radiation exposure were assessed in the case of 10 children with neurological impairment, 8 categorized with Autism Spectrum Disorder. Data was obtained for:

- 1) Mothers' Body Voltage in the mothers' sleeping location during pregnancy;**
 - 2) Child's Body Voltage in current sleeping location;**
 - 3) Microwave Power Density in mothers' sleeping location during pregnancy (microwatt/square meter);**
and
 - 4) Child's Microwave Exposure in current sleeping location.**
- Data for mothers with neurologically impaired children were contrasted with similar data for 5 healthy children and their mothers.

The results were as follows:

Body Voltage Levels:

Median Body Voltage Level in Mom's Bed During Pregnancy*

	Value	Range
Neurologically Impaired Children	1,872 millivolts	(380-6,040)
Healthy Group	224 millivolts	(12-480)

8.4x Higher body voltage levels in moms with neurologically impaired children

**Note research shows whatever the Body Voltage of the Mom, it is even higher in the fetus.*

Body Voltage of child in current bed location

	Value	Range
Neurologically Impaired Children	1,028 millivolts	(420-4,900)
Healthy Group	120 millivolts	(0-230)

Conclusion: 8.5x Higher Body Voltage in Neurologically Impaired Child's Sleeping Location

Microwave Exposure:

Microwave Power Density in Sleeping Location

	mw/sq. meter	Range
Neurologically Impaired Children-Mom's Bed Exposure In Pregnancy	290	(110-1,710)
Healthy Group	14	(0-67)

Conclusion: 20.7x higher microwave power density in moms sleeping location in cases where children were neurologically impaired

This pilot data strongly suggests that electromagnetic radiation in the sleeping environment of mothers during pregnancy, as well as electromagnetic radiation in the sleeping environment of children, may be the undiscovered key contributing - if not causative - factor in neurological impairments in children, including autism. Given increasing levels of ambient electromagnetic radiation in modern environments from society's use of electronic equipment, wireless technologies, such as cell phones and wireless networks, high frequency transients on electric lines, and broadband over power lines (BPL), this association needs immediate further exploration