**November 21, 2017**

# ELF-EMF Oxidative Effects

Of 186 total studies: **(E= 162** (**87**%**); NE= 24** (**13**%)

(E = reported effect; NE = reported no significant effect)

**ELF-EMF and Cellular Oxidative Processes**

Reactive oxygen species (ROS) are produced as a result of cellular metabolism. Presence of ROS in cells can lead to macromolecular damages (in DNA, proteins, and lipids), disturbance in cell functions, and cell death. Damage in DNA is a cause of cancer. Under normal conditions, ROS level is kept in check by various cellular anti-oxidative processes. In instances when there is an increase in ROS production or a deficit in anti-oxidative capacity, oxidative stress occurs leading to cell damage and functional deficits. However, free radicals are also involved in so called cellular signaling cascades that keep cells functioning properly and in immune defense against bacteria. Thus, it is essential to keep free radicals at a critical physiological homeostatic level. Any disturbance could lead to detrimental biological effects. Non-ionizing electromagnetic field is a disturber of cellular oxidative processes. A brief description of cellular oxidative processes is included in Appendix A at the end of this file.

Effects of extremely-low frequency electromagnetic field (0-300Hz) (ELF-EMF) on oxidative status in biological systems have been extensively studied in the past decades. There are rather strong indications that exposure to ELF-EMF affects oxidative status in cells and animals. Many of the oxidative and anti-oxidative components described in Appendix A have been shown to be affected by ELF-EMF. This is a summary and review of the literature on the effects of ELF-EMF exposure on oxidative processes in living organisms. Findings of papers related to the topic published up to October, 2017 are summarized in Table I below followed by the literature list with abstracts.

Effect on cellular oxidative status is probably the most consistent biological effect of non-ionizing electromagnetic fields (EMF). It has been reported after exposure to static to radiofrequency (see the RFR section on oxidative effects in the BioInitiative Report) EMF, and in many different animal and plant species.

Cellular oxidative-process is a complex physiological mechanism. It involves feedbacks and compensatory responses of the components to maintain homeostasis. EMF could disturb several components of the process described above leading to a cascade of changes. Thus, it is not surprising that the changes described in Table 1 show a complex pattern, i.e., changes are not always in the same direction. This could be caused by the cell type and organ studied, time when the changes were measured, and EMF exposure conditions (such as intensity and duration of exposure and characteristics of the field).

Several papers reported changes in biochemistry, physiology, and functions as a consequence of changes in cellular oxidative status resulting from exposure to ELF-EMF. These include: DNA damage (Lai and Singh, 1997, 2004); immune response (Akan et al., 2012); inflammatory response (Kim et al, 2017); apoptosis (a form of cell death) (Koh et al., 2008); cell proliferation (Lee et al., 2010); rhythmic slow activity in hippocampal slices of the brain (Bawin et al., 1996); visual evoked potentials (Akpinar et al., 2012); auditory event-related potentials (Akpinar et al., 2016); visual and somatosensory evoked potentials (Gok et al., 2014); heart rate (Ciejka and Goraca, 2009); wound healing (Glinka et al., 2013); hyperalgesia (i.e., excessive sensitivity to pain) (Jeong et al., 2006); opioid-induced antinociception (Kavaliers et al., 1998); spatial memory and learning (Cui et al., 2012; Deng et al., 2013); cognitive impairment (Duan et al., 2013); mismatch-negativity response (Kantar-Gok et al., 2014); depressive disorder (Ansari et al., 2016); anxiety-like behavior (Djordjevic et al., 2017); and obsessive compulsive disorder-like behavior (Salunke et al., 2014). However, in most of these studies, the cause-effect relationship was not well established. Do ELF-EMF-induced changes in oxidative status cause these effects? Or, are they effects of ELF-EMF caused by different mechanisms unrelated to oxidative changes? One powerful proof is to establish whether an effect, e.g., memory deficit, can be blocked by antioxidants or pro-oxidants. An effect caused by a change in free radicals should be able to be blocked by antioxidants or pro-oxidants. This is why a colume labelled “Effect of antioxidants” is included in Table 1.

In most of the ELF-oxidative effects studies, the intensities used were relatively high (i.e., in the mT range) which are much higher than ambient levels of ELF-EMF in the human environment. However, the exposure durations in most of these studies are short-term (from hours to several days), whereas environmental exposure is chronic. Can the results apply to real-life exposure situation? Do oxidative changes occur after exposure to ambient levels of ELF-EMF? But, the effects could possibly occur in occupational exposure conditions and below the guideline limits set by most international regulation agencies. There are several studies that showed oxidative effects at low ELF-EMF intensities: on activated mouse peritoneal neutrophils at 74.7 T (Belova et al., 2010); human keratinocytes at 50 T (Cilejka et al., 2014); maize seedlings at 22 T (Hajnorouzi et al., 2011); SH-SY5Y human neuroblastoma cells pretreated with menadione at 10 T (Kesari et al., 2016); K562 human leukemia cell at or below 25 T (Mannerling et al., 2010); rat brain at 50 T (Manikonda et al., 2014); human umbilical vein endothelial cells at 30 T (Martino, 2011); rat glioma C6 cells at 30 T (Naarala et al., 2017); human neutrophils at 10 T (Poniedzialek et al., 2013a); snail digestive gland at 2.88 T (Regoli et al. 2005); human serum and red blood cells at 8.8 - 84 T (Sharifian et al., 2009); and rat lymphocytes stimulated by FeCl2 at 40 T (Zmylony et al., 2004a). Related to this is a recent paper by Kapri-Pardes et al. ([Kapri-Pardes E](https://www.ncbi.nlm.nih.gov/pubmed/?term=Kapri-Pardes%20E%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Hanoch T](https://www.ncbi.nlm.nih.gov/pubmed/?term=Hanoch%20T%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Maik-Rachline G](https://www.ncbi.nlm.nih.gov/pubmed/?term=Maik-Rachline%20G%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Murbach M](https://www.ncbi.nlm.nih.gov/pubmed/?term=Murbach%20M%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Bounds PL](https://www.ncbi.nlm.nih.gov/pubmed/?term=Bounds%20PL%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Kuster N](https://www.ncbi.nlm.nih.gov/pubmed/?term=Kuster%20N%5BAuthor%5D&cauthor=true&cauthor_uid=29035881), [Seger R](https://www.ncbi.nlm.nih.gov/pubmed/?term=Seger%20R%5BAuthor%5D&cauthor=true&cauthor_uid=29035881). *Activation of Signaling Cascades by Weak Extremely Low Frequency Electromagnetic Fields.* [Cell Physiol Biochem.](https://www.ncbi.nlm.nih.gov/pubmed/29035881) 2017 Oct 16;43(4):1. doi: 10.1159/000481977. [Epub ahead of print]) showing effects on cellular signal cascades in eight cell lines exposed to ELF-EMF at 0.15 T (i.e., 1.5 mG) at a similar level that has been suspected to cause childhood leukemia. Though the authors did not investigate oxidative status of their cells, they concluded that the effects were mediated by NADP oxidase, an enzyme that can generate superoxide free radicals. In addition, similar to the finding of Kesari et al. (2016), Maes et al., (Maes A, Anthonissen R, Verschaeve L. *On the allerged association between extremely low frequency magnetic field exposures and increase risk of Alzheimer’s disease.* Rad. Applic. 1(2) 151-154, 2016.) also reported an increase in micronucleus formation in SH-SY5Y human neuroblastoma cells after exposure to a 50-Hz magnetic field at 10 T, but without pretreatment with menadione as in the Kesari et al. (2016) study. Thus, disturbance of oxidative processes can occur at ambient levels of ELF-EMF.

Cellular oxidative processes serve important functions. Free radicals are involved in cellular signaling cascades that govern normal cell functions. They are also involved in cell chemistry that triggers apoptosis. Harnessing cellular oxidative status using ELF-EMF could be beneficial in the treatment of diseases. Several papers in the literature list suggested such possibilities including: improve immune responses (Akan et al., 2010; Belova et al., 2010; Frahm et al., 2006; Kim et al., 2017); treatment of osteoarthritis (De Mattei et al., 2003); attenuation of ischemic brain injury (Duong et al., 2016; Raus Balind et al., 2014); increase antioxidant properties in cells and tissues (Falone et al., 2016); treatment of myopathies (Vignola et al., 2012); wound healing and tissue regeneration (Glinka et al., 2013; Patruno et al., 2010, 2011); cytoprotection (Osera et al., 2011, 2015); induce differentiation of stem cells (Park et al., 2013); and protective effect on Huntington’s disease (Tasset et al., 2012; Tunez et al., 2006). One interesting prospect is the use of ELF-EMF in the treatment of cancer. EMF can selectively kill cancer cells (Lai H, Singh NP. *Medical applications of electromagnetic fields.* Institute of Physics Conference Series: Earth and Environmental Science 10 (2010) 012006 (<http://dx.doi.org/10.1088/1755-1315/10/1/012006>; and see also the introduction section in Lai H, Chan HW, Singh NP. *Effects of radiation from a radiofrequency identification (RFID) microchip on human cancer cells.* Inter J Radiat Biol. 92:156-161, 2016.) Many years ago, we (Lai and Singh, 2004) speculated that cancer cells are more vulnerable to EMF than normal cells and that EMF kills cancer cells by free radical formation. Since it is much easier to produce ELF-EMF than RFR, and ELF-EMF gives a more uniform distribution and better tissue penetration that RFR, it is more advantageous to use ELF-EMF for cancer treatment. Let us look at the studies of ELF-EMF exposure on cancer cells that are included in the ELF-free radical literature list summarized in the table below.

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|  | **Cancer cell type** | **Exposure conditions** | **Effects** |
| Ayşe et al. (2010) | K562 human leukemia cells | 50 Hz MF, 5 mT, 1 h or 1 h/day for 4 days | Single exposure decreased differentiation, repeated exposure increased differentiation |
| Benassi et al. (2016) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 1 mT, 6-72 h | Enhanced neurotoxin-induced apoptosis |
| Buldak et al. (2012) | AT478 murine squamous carcinoma cells | 50-Hz MF, 1 mT, 16 min | MF lessen oxidative stress and DNA damage induced by cisplatin |
| Calabro et al. (2013) | SH-SY5Y human neuroblastoma cells | Static MF, 2.2 mT, 24 h | Increased ROS, decreased mitochondrial membrane potential |
| Consoles et al. (2017) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 1 mT, 24-72 h | Increased microRNA activity, increased oxidative stress |
| De Nicola et al. (2006) | U937 human lymphoma cells | Static MF, 0.6 mT, 2 h; 50-Hz MF 0.07-0.1 mT, 2 h | Reduced apoptosis |
| Ding et al. (2004) | HL-60 human leukemia cells | 60-Hz MF, 5 mT, 24 h | Enhanced apoptotic effect of H2O2 |
| Falone et al. (2016) | Human drug-resistant SK-N-BE(2) neuroblastoma cells | 72-Hz pulsed MF, 2 mT, 15 min 3 times over 5 days | Increased anti-oxidation activity, decreased ROS production |
| Falone et al. (2017) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 0.1 or 1 mT, 5 and 10 days | Increased proliferation and survival advantage of cells |
| Garip and Akan (2010) | K562 human leukemia cells | 50-Hz MF, 1 mT, 3 h | Decreased and increased apoptosis in untreated and H2O2-treated cells, respectively |
| Giorgi et al. (2014) | Human SK-N-BE(2) neuroblastoma cells | Bipolar pulsed square-wave MF, 50-Hz, 1 mT, up to 72 h | No significant effect on H2O2-induced DNA double strand breaks |
| Höytö et al. (2017) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 0.1 mT, 24 h | No significant change in micronucleus formation |
| Kesari et al. (2015) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 0.1 mT, 24 h | Increased micronucleus formation observed days after exposure |
| Kesari et al. (2016) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 10 or 30 T, 24 h | Increased micronucleus formation when combined with menadione |
| Koh et al. (2008) | Human prostate cancer cells (DU145, PC3, and LNCaP) | 60-Hz MF, 1 mT, 6, 24, 48, or 72 h | Apoptosis and cell cycle arrest |
| Koyama et al. (2006) | Human A172 glioblastoma cells | 60-Hz MF, 5 mT, 2, 4, 8, 16, or 24 h | Potentiated H2O2-induced DNA lesion |
| Luukkonen et al. (2014) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 0.1 mT, 24 h | Increased micronucleus formation at 8- 15 days after exposure |
| Mahmoudinasab et al. (2016) | Human MCF-7 breast adenocarcinoma cells | 50-Hz EMF, 0.25 and 0.5 mT; 5-min on/5-min off; 15-min on/15-min-off, or 30 min continuously; total exposure time 30 min | Changes in mRNA level of 7 antioxidant genes |
| Mannerling et al. (2010) | K562 human leukemia cells | 50-Hz MF 0.025-0.1 mT, 1 h | Accumulation of cells in the G2 phase |
| Martinez et al. (2016) | Human NB69 neuroblastoma cells | 50-Hz MF, 0.1 mT, 3-h on/3-h off for 24, 42, or 63 h, or continuously for 15-120 min | MF activated MAPK-p38 and ERK ½, increase in cell proliferation |
| Martino and Castello (2011) | Human HT1080 fibrosarcoma and AsPC-1 pancreatic cancer cells | Static MF, geomagnetic field (45-60 T) or shielded field (0.2-2 T), 24 h | Decreased H2O2 in shielded samples compared to geomagnetic field |
| Morabito et al. (2010a) | Rat PC-12 pheochromocytoma cells | 50-Hz MF, 0.1 or 1 mT, 30 min or 7 days | No significant effect on cell proliferation |
| Naarala et al. (2017) | Rat C6 glioma cells | Nearly vertical 33 T static MF plus a horizontal or vertical 50-Hz 30 T MF, 2 h | Cell proliferation suppressed by a horizontal ELF field |
| Osera et al. (2011) | SH-SY5Y human neuroblastoma cells | 72-Hz pulsed MF, 2 mT, 24 h | Decreased cell proliferation with higher quiescence |
| Osera et al. (2015) | SH-SY5Y human neuroblastoma cells | 72-Hz pulsed MF, 2 mT, 10 min for 4 times over 7 days or 72 h | Increased protection against oxidative stress |
| Patruno et al. (2011) | Human THP-1 acute myeloid leukemia cells | 50-Hz MF, 1 mT, 24 h | Increased nitric oxide and catalase activities |
| Patruno et al. (2012) | Human THP-1 acute myeloid leukemia cells | 50-Hz MF, 1 mT, 24 h | Increased nitric oxide and superoxide, decreased SOD and catalase |
| Patruno et al. (2015) | Human K562 leukemia cells | 50-Hz MF, 1 mT, 24 h | Decreased nitric oxide activity and increased catalase activity |
| Reale et al. (2014) | SH-SY5Y human neuroblastoma cells | 50-Hz MF 1 mT, 1, 3, 6, or 24 h | Increased ROS and antioxidation activity |
| Sadeghipour et al. (2012) | Human T47D breast carcinoma cells | 100 and 217 Hz pulsed EMF, 0.1 mT, 24-72 h | No significant change in apoptosis |
| Villarini et al. (2017) | SH-SY5Y and SK-N-BE-2 human neuroblastoma cells | 50-Hz MF, 0.01, 0.1 or 1 mT, 1 h continuously or 5 h intermittently | No significant effect on GSH/GSSG ratio |
| Wartenberg et al. (2008) | Human UM-SCC-14-C oral mucosa cancer cells | DC EF, 4 V/m, 24 h | Increased apoptosis |
| Wolf et al. (2005) | Human HL-60 leukemia cells | 50-Hz MF, 0.5-1 mT, 24-72 h | Dose-dependent increase in cell proliferation |
| Zwirska-Korczala et al. (2004) | Murine AT478 squamous carcinoma cells | Mixture of frequencies up to 400 Hz, MF, 0.11 mT, 16 min, assayed 24 and 72 h after exposure, cells also treated with melatonin | ELF-MF attenuated antioxidative effects of melatonin |

Several studies f the above list suggested a possible beneficial effect on cancer treatment under ELF-EMF exposure (Benassi et al., 2016; Calabro et al. 2013; Consles e al., 2017; Ding et a., 2004; Kesari et al., 2015, 2016; Koh et al. 2008; Koyama et al., 2006;Luukkonen et al., 2014; Mannerling et al., 2010; Naarala et al. 2017; Osera et al., 2011; Wartenberg et al. 2008), others suggested a protective effect that would allow cancer cells to proliferate (Buldak et al. 2012; De Nicola et al., 2006; Falone et al., 2016, 2017; Martinez et al., 2016; Osera et al., 2015; Wolf et al., 2005), whereas no effect was reported by some (Giorgi et al. 2014; Höytö et al., 2017; Morabito et al. 2010a; Reale et al., 2014; Sadeghipour et al., 2012; Villarini et al., 2017). Interestingly, two studies (Ayse et al., 2010; Garip and Akan, 2010) showed opposite effects depending on the duration of exposure. This reflects the discussion above on the dynamic of cellular oxidative processes and its ability to compensate. Cell type probably plays a significant role. Cell-type specific responses to ELF-EMF have been reported by Sullivan et al. (2011). The conditions of exposure probably played a role in the diversity of the responses, but the conditions of exposure described in the table above do not reveal any consistent pattern on how exposure parameters affect cellular oxidative processes. Thus, it is imperative to understand the conditions under which ELF-EMF could lead to a consistent increase in free radicals in cells.

Finally, a few words have to be said on ELF-electric fields. There are 14 electric field studies: Akpinar et al. (2012; 2016); Calota et al. (2006): Fitzsimmons et al. (2008); Gok et al. (2014); Guler et al. (2008; 2009a, b); Harkaw et al. (2005); Kantar-Gok et al. (2014); Milisa et al. (2017) Turkozer et al. (2008); Wartenberg et al. (2008); and Wu et al. (2016). In most studies, 50-Hz electric field at kV/m intensity (2- 21.8 kV/m) and exposure time from hours to days were studied. Most studies reported effects indicative of increase in free radicals, e.g., increases in lipid peroxidation and protein carboxylation (see Table 1). A study by Fitzsimmons et al. (2008) using a pulsed electric field at 0.2 mV/cm (1 mV/cm = 0.0001 kV/m) reported an increase in nitric oxide after 30 minutes of exposure. Wartenberg et al. (2008) used a 4 V/m (1 V/m = 0.001 kV/m) DC-electric field and reported changes in the antioxidative enzymes SOD and GSH activities. This is actually quite interesting. Since electric fields do not penetrate into cells, do electric and magnetic fields act on different mechanisms leading to changes in cellular oxidative processes?

Table I. Summary of papers on the effects of ELF-EMF on oxidative processes in cells and animals. (\* Study reported no significant effect on oxidative processes; ↑ increase; ↓ decrease; Ø no significant effect; MF= magnetic field; EF = electric field; CAT= catalase; GSH= glutathione; GST = glutathione S-transferase; GPx = glutathione peroxidase; NOS= nitric oxide synthase; MPO= myeloperoxidase; ROS = reactive oxygen species; SOD= superoxide dismutase) In some studies, the term EMF (electromagnetic field) was used. The authors may mean magnetic field or a combination of magnetic and electric fields, since most exposure systems emits both fields when not properly shielded and grounded. On the other hand, fields labelled as magnetic field in some studies may contain electric component.,

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|  |  |  | *Oxidative damages (DNA, protein, lipid)* | *ROS (O2****.-****, OH, H2O2, NO)* | *NOS* | *Antioxidative processes (SOD, CAT/ peroxidase, GSH, GPx)* | *Effect of antioxidants* | *Remarks* |
| Akan et al. (2010) | Activated THP-1 cells (human monocytic leukemia cells) | 50-Hz EMF, 1 mT, 4-6 h |  | ↑NO | ↓ iNOS |  |  | ↑cGMP |
| Akdag et al. (2007) | Sprague-Dawley rat serum in vivo | 50-Hz MF,  0.1 and 0.5 mT, 2 h/day, 10 months |  | ↓NO |  |  |  |  |
| Akdag et al. (2010) | Sprague-Dawley rat brain in vivo | 50-Hz MF,  0.1 and 0.5 mT, 2 h/day, 10 months | ↑ lipid peroxidation |  |  | ↓ CAT |  | ↑ total oxidant status, ↓ total anti- oxidative capacity |
| Akdag et al. (2013a) | Sprague-Dawley rat brain in vivo | 50-Hz MF,  0.1 and 0.5 mT, 2 h/day, 10 months | ↑ protein carboxylation  ↑ lipid peroxidation |  |  |  |  |  |
| \*Akdag et al. (2013b) | Sprague-Dawley rat testes in vivo | 50-Hz MF,  0.1 and 0.5 mT, 2 h/day, 10 months | Ø lipid peroxidation |  |  | Ø CAT |  | No change in  total oxidant status and total anti- oxidative capacity |
| Akpinar et al. (2012) | Wister rat brain and retina in vivo | 50-Hz EF,  12 and 18 kV/m, 1 h/day, 14 days | ↑ lipid peroxidation |  |  |  |  | ↑ total oxidant status, ↓ total anti- oxidative capacity |
| Akpinar et al. (2016) | Wister rat brain in vivo | 50-Hz EF,  12kV/m, 1 h/day, prenatal (Pr), postnatal (Po, 30 days), and prenatal + postnatal (PP) | ↑lipid peroxidation in Po, ↓in PP (cf. Pr and Po)  ↓ protein carboxylation in PP |  |  |  |  |  |
| Aksen et al. (2006) | Wister rat uterus and ovary in vivo | 50-Hz EMF, 1 mT, 3 h/day, 50 or 100 days | ↑ lipid peroxidation |  |  |  |  |  |
| \*Alcaraz et al. (2014) | Micronucleated cells induced by EMF in bone morrow of mouse | 50-Hz EMF, 0.2 mT, for 7, 14, 21, or 28 days |  |  |  |  | Effect not blocked by 4 types of antioxidant |  |
| Ansari et al. (2016) | NMRI mice | 50-Hz MF, 0.5 mT, 2 h |  | ↑ NO |  |  |  | Blocked effect of L-NAME, a NOS inhibitor |
| Asghar et al. (2016) | Soybean seeds and seedlings | 50-Hz MF, 50, 75, or 100 mT for 3 or 5 min | ↑ lipid peroxidation at 50 mT for 3 min (not at other exposure conditions) | ↑H2O2 at 50 and 100 mT for 3 min |  | ↑ SOD at 75 mT for 3 and 5 min; ↑CAT/peroxidase at 50, 75 and 100 mT for 3 min; |  | ↑ ascorbic acid |
| Ayşe et al. (2010) | K562 cells, in vitro | 50-Hz EMF, 5 mT, 1 h or 1 h/day for 4 days |  | ↑ O2**.-** |  |  |  | Effect disappeared at 2 h post-exposure, no interaction with hemin |
| Bawin et al. (1996) | Electrical activity of rat hippocampal slices | 1-Hz MF, 0.56 and 0.056 mT, 10 min |  |  | Effect blocked by NOS inhibitor |  |  | 60-Hz MF has no significant effect |
| Bediz et al. (2006) | Sprague-Dawley rat blood and brain in vivo | 50-Hz EMF, 0.005 mT, 5 min every other day for 6 months | ↑ lipid peroxidation |  |  | ↓GSH |  | Effect attenuated by zinc |
| Belova et al. (2010) | Activated mouse peritoneal neutrophils | Combined magnetic field (CMF) tuned to calcium ion (DC 40.6 T, AC 74.7 T at 31 Hz): pulsed MF (225 s, 20 pulses packet at 15 Hz, 1500 T); up to 30 min exposure |  | CMF ↓ ROS, pulsed MF ↑ ROS |  |  |  |  |
| Benassi et al. (2016) | SH-SY5Y cells (human used to study Parkinson’s disease) | 50-Hz MF, 1 mT, 6-72 h | ↑ protein carboxylation |  |  |  |  |  |
| Buczyński et al. (2005) | Human blood platelets | 1 kHz MF, 0.5 mT, 30, 60 or 90 min | ↑ lipid peroxidation |  |  |  |  | Effect observed only after 30 and 90 min exposure, not at 60 min |
| Buldak et al. (2012) | AT478 murine squamous carcinoma cells | EMF 50-Hz, 1 mT, 16 min | ↓ lipid peroxidation |  |  | ↑SOD  ↑GPx |  | MF lessens oxidative effects of  cisplatin |
| Calabro et al. (2013) | SH-SY5Y cells | Static MF, 2.2 mT, 24 h |  | ↑ ROS production |  |  |  |  |
| Calota et al. (2006) | Human blood serum | 50-Hz EF, 5, 7.5 10, 15, 20 kV/m, 1-2 h |  | ↓ ROS production |  |  |  |  |
| Calota et al. (2007) | Human blood serum | 50-Hz MF, 0.357, 0.596, 1.788, 2.384 mT, 1-2 h |  | ↑ ROS production, enhanced by FeCl2 and H2O2 |  |  |  |  |
| Canseven et al. (2008) | Guinea pig, liver and heart tissues | 50-Hz MF, 1, 2, or 3 mT, 4 or 8 h/day for 5 days | ↑ and ↓ in lipid peroxidation | ↑ and ↓ in NO |  | MPO (↑ or ↓) depending on exposure condition (duration and intensity) and tissue studied;  ↑ and ↓ in GSH |  |  |
| Chen et al. (2014) | Mouse embryonic fibroblast | 50-Hz MF, 2 mT, 0.5, 2, 6, 12, 24 h |  | ↑ ROS |  |  |  |  |
| Cheun et al. (2007) | Canine kidney MDCK cells | 60-Hz MF, 1.4 mT, seconds |  | MF affected ROS kinetics when H2O2 was added to cells. |  |  |  |  |
| Chu et al. (2011) | Mouse cerebellum in vitro | 60-Hz MF, 2.3 mT, 3 h | ↑ lipid peroxidation | ↑OH |  | ↑SOD  Ø GPx |  |  |
| Chung et al. (2015) | Rat brain in vivo | 60-Hz MF, 2.0 mT, 2 or 5 days |  | ↑NO in striatum, thalamus and hippocampus |  |  |  |  |
| Cichon et al. (2017) | Post-stroke patients | 40-Hz, 7 mT for 15 min/day for 4weeks (5 days a week) |  |  |  | ↑ SOD and CAT in hemolysates |  | Ø total antioxidant status in plasma; exposed patients showed better improvement in functional and mental status |
| Ciejka et al. (2009) | Sprague-Dawley rats in vivo  (plasma) | 40-Hz MF, 7 mT, 30 or 60 min per day for 14 days |  |  |  |  |  | Repeated 30-min and 60-min exposure increased and decreased plasma antioxidant activity, respectively. |
| Ciejka et al. (2010) | Sprague-Dawley rats in vivo  (muscle) | 40-Hz MF, 7 mT, 30 or 60 min per day for 14 days |  |  |  |  |  | Both exposures caused an increase in  -SH and decrease in proteins in muscle |
| Ciejka et al. (2011) | Sprague-Dawley rats in vivo  (brain) | 40-Hz MF, 7 mT, 30 or 60 min per day for 14 days | ↑ lipid peroxidation  in brain of 30-min per day exposed rats |  |  |  |  | Rats exposed for 60 min per day, 14 days showed increases in  -SH and proteins in brain (adaptation). |
| Ciejka et al. (2014) | Sprague-Dawley rats in vivo  (muscle) | 40-Hz MF, 7 mT, 30 or 60 min per day for 14 days |  |  |  | ↑GSH |  |  |
| Cinzia et al. (2016) | Human keratinocyte (NCTC 2544) | 50 Hz MF, 0.025 – 0.2 mT, 1 h | ↑ lipid peroxidation at 0.05 and 0.1 mT | ↑ ROS at 0.05 and 0.1 mT |  | ↓ SOD and ↑GSH at 0.05 and 0.1 mT |  | ↑ ROS blocked by the iron chelator o-phenanthroline |
| Consales et al. (2017) | SH-SY5Y human neuroblastoma cells and mouse primary cortical neurons | 50-Hz MF. 1 mT, 24, 48 or 72 h | ↑ O2**.-**, H2O2, |  |  |  |  | Some ROS produced by mitochondria; affected by microRNA (miR-34) |
| Coskun et al. (2009) | Guinea pig in vivo- plasma, brain, and liver | 50-Hz MF, 1.5 mT, continuous (C) (4h/day) or intermittent (I) (2 h on/2 h off/2h on) for 4 days | Plasma: I ↑ lipid peroxidation  Brain: C, ↓ lipid peroxidation  Liver: C, I ↑lipid peroxidation | Plasma: C, I ↑ NO |  | MPO: Plasma C ↑, Brain C, I ↑,  Liver C, I ↓  GSH: C ↑ I ↓ in brain |  |  |
| Cui et al. (2012) | C57BL/6 mice in vivo, striatum and hippocampus | 50-Hz MF, 1 or 0.1 mT, 4h/day, 12 days | ↑ lipid peroxidation in 1 mT group |  |  | ↓ CAT and ↓GSH in 1 mT group |  | ↓Total antioxidant capability in 1 mT group |
| \*de Groot et al. (2014) | Normal and chemically-stressed PC12 cells | 50-Hz EMF, 30 min or 48 h, up to 1 mT |  | No effect on ROS production as measured by H2-DCFDA |  |  |  |  |
| \* De Mattei et al. (2003) | Bovine articular cartilage explants | 75-Hz EMF 1.3 ms pulses, 2.3 mT peak, 24 h |  | Ø NO |  |  |  | Pulses enhanced Interleukin-1induced NO production |
| De Nicola et al. (2006) | U937 cells | Static MF, 6 mT, 2 h; 50-Hz MF, 0.07-0.1 mT, 2 h |  | ↑ ROS |  | ↓GSH |  | Decreased apoptosis |
| Deng B. et al. (2014) | Rat primary cerebral cortical neurons | Electromagnetic pulses (peak 400 KV/m, width 350 ns, 0.5 pps, I Hz) | ↑ lipid peroxidation |  |  | ↓SOD |  | Decreased cell viability observed, effects antagonized by sevoflurane |
| Deng Y. et al. (2013) | SPF Kunming mouse in vivo, serum and brain | 50-Hz MF, 2 mT, 4h/day, 8 weeks | ↑ lipid peroxidation |  |  | ↓SOD |  | No interaction with aluminum |
| Di et al. (2012) | Human preosteoclast FLG29.1 cells | Large gradient high magnetic fields (12 T,  -1370 T2/m; 12 T, 1370 T2/m), 72 h |  | ↓ NO |  |  |  |  |
| \*Di Loreto et al. (2009) | Rat cortical neurons | 50-Hz MF, 0.1 or 1 mT, 7 days | Ø lipid peroxidation | Ø total ROS |  | Ø GSH |  | ↑ cell viability, ↓ apoptosis |
| Ding et al. (2004) | Human leukemia HL-60 cells | 60-Hz MF, 5 mT, 24 h |  |  |  |  |  | Enhanced apoptotic effect of H2O2 |
| Djordjevvic et al. (2017) | Wistar male rats | 50-Hz MF, 10 mT, 7 days, 24 h/day |  | ↑ *O2****.-*** *and* NO; Ø peroxynitrite (ONOO-) in hypothalamus |  |  |  |  |
| Duan Y. et al. (2013) | ICR mouse,  Serum and hippocampus | 50-Hz MF, 8 mT, 4 h/day, 28 days | ↑ lipid peroxidation | ↑NO | ↑NOS | ↓SOD  ↓ CAT  ↓GPx |  | Effects reversed by lotus seedpot procyanidins |
| \*Duan W. et al. (2015) | Mouse spermatocyte-devrived GC-2 cells | 50-Hz EMF, 1, 2, or 3 mT, 5-min on 10-min off, 24 h | Ø oxidative DNA base damage |  |  |  |  |  |
| Duong and Kim (2016) | Human microglial HMO6 | 50-Hz EMF, 1 mT, 4 h |  |  |  |  |  | EMF exposure decreased ROS induced by oxygen-glucose deprivation. |
| Emre et al. (2011) | Wistar rat in vivo, liver | Pulsed EMF (0.5 ms rise time, 9.5 ms fall time) EF 0.6 V/m, MF 1.5 mT, each [frequency](javascript:openWindow('/gl_detail.php?l=e&id=5','fad6c43b628858e0b472d0c164557fcf','width=480,height=480,scrollbars=yes')) train of 1 [Hz](javascript:openWindow('/gl_detail.php?l=e&id=27','d1e2634cb743590b235fe6b1dfa763ba','width=480,height=480,scrollbars=yes')), 10 [Hz](javascript:openWindow('/gl_detail.php?l=e&id=27','d1e2634cb743590b235fe6b1dfa763ba','width=480,height=480,scrollbars=yes')), 20 [Hz](javascript:openWindow('/gl_detail.php?l=e&id=27','d1e2634cb743590b235fe6b1dfa763ba','width=480,height=480,scrollbars=yes')) and 40 [Hz](javascript:openWindow('/gl_detail.php?l=e&id=27','d1e2634cb743590b235fe6b1dfa763ba','width=480,height=480,scrollbars=yes')) was given for 4-min and with 1-min interval between each [frequency](javascript:openWindow('/gl_detail.php?l=e&id=5','fad6c43b628858e0b472d0c164557fcf','width=480,height=480,scrollbars=yes')) (together 20 min.); on each day, three exposure cycles performed (1 h), 1h per day for 30 days | ↑ lipid peroxidation |  |  | ↑SOD |  | No effect on apotosis, decreased necrosis. |
| Erdal et al. (2008) | Male and female Wistar rat in vivo, liver | 50-Hz MF, 1 mT, 4/day, 45 days | Ø lipid peroxidation |  |  |  |  | Increased 3-nitrotyrosine (oxidative/ nitrosative stress) in liver of female rats. |
| Falone et al. (2008) | Female Sprague-Dawley rat in vivo, 3- and 19-month old, brain cortex | 50-Hz MF, 0.1 mT, 10 days |  |  |  | ↑SOD2 in young rats; ↓ catalase and GPx in old rats |  | ↓Glutathione reductase in old and young rats, ↓ glutathione-s-transferase in old rats: old and young rats responded differently. |
| Falone et al. (2016) | Human drug- resistant neuroblastoma SK-N-BE(2) cells | 72-Hz pulsed EMF, 1.3 ms pulse duration, 2 mT, 15 min, 3 times over 5 days |  |  |  |  |  | Pulsed EMF increased MnSOD-based antioxidant protection and reduced ROS production induced by H2O2 . |
| Falone et al. (2017) | SH-SY5Y human neuroblastoma cells | 50-Hz MF, 0.1 or 1 mT, 5 and 10 days | ↓ protein carboxylation and DNA oxidation |  |  | ↑ GPx/SOD and catalase/SOD ratios, i.e., increase antioxidating defense; ↑ GPx activity |  | Protects cell death by H2O2, ↑ Nrf2 activity |
| Feng et al. (2016a) | Human amniotic epithelial cells | 50-HZ MF, 0.4 mT, 5, 15, 30 or 60 min |  | ↑ ROS |  |  | MF-induced mitochondrial permeability transition blocked by NAC |  |
| Feng et al. (2016b) | Human amniotic epithelial cells | 50-HZ MF, 0.1, 0.2, or 0.4 mT, 5, 15, 30, or 60 min |  | ↑ ROS |  |  |  | ↑ total ROS at 0.2 mT and higher, ↑NADPH oxidase-produced superoxide |
| Feng et al. (2016c) | Human amniotic epithelial cells | 50-Hz MF, 0.2-2 mT, 30, 60, 120 min |  | ↑ mitochondrial ROS |  |  |  | ↑ ROS led to activation of Akt and anti-apoptotic effect |
| Fernie & Bird (2001) | American kestrel | 60-Hz EMF, 30T,10 kV/m, 91 days, 23.5 h/day |  |  |  |  |  | Decreased plasma carotenoids |
| Fiorani et al. (1997) | Rabbit red blood cells | 50-Hz MF, 0.2-0.5 mT, 90 min in the presence of an oxygen-generating system (Fe(II)/ascorbate) |  |  |  |  |  | Enhanced GSH reduction and hemoglobin oxidation caused by Fe(II)/ascorbate at 0.5 mT |
| Fitzsimmons et al. (2008) | Human chondrocyte | Pulsed electric field, EF in culture medium 0.2 mV/cm, 30 min |  | ↑NO |  |  |  | ↑cGMP,  calcium involved |
| Frahm et al. (2006) | Mouse bone-marrow derived macrophage | 50-Hz MF, 0.05, 0.1, 0.5, 1.0 mT, 45 min |  | ↑ROS |  |  |  |  |
| Frahm et al. (2010) | Mouse bone-marrow derived macrophage | 50-Hz MF, 1.0 mT, 45 min |  | ↑ROS |  |  |  | Activated enzymes  (NAD(P)H oxidases) and proteins involved in redox homeostasis |
| Garip and Akan (2010) | K562 human leukemia cells, normal or treated with H2O2 | 50-Hz EMF 1 mT, 3 h |  | ↑ROS |  |  |  | Decreased and increased apoptosis in untreated and H2O2-treatedcells, respectively. |
| Ghodbane et al. (2011a) | Wistar male  rat in vivo,  plasma | Static MF, 128 mT, 1 h/day, 5 days | Ø lipid peroxidation |  |  | ↑ GPx |  | Decreased vitamin A and E levels, effects blocked by selenium |
| Ghodbane et al. (2011b) | Wistar male  rat in vivo, liver, kidney, muscle , brain | Static MF, 128 mT, 1 h/day, 5 days |  |  |  | ↑ SOD in liver,  ↓ GPx in kidney and muscle,  ↑ GSH in liver |  | Selenium reversed GPx effect in kidney and muscle |
| Ghodbane et al. (2014) | Wistar male rat in vivo, plasma | Static MF, 128 mT, 1 h/day, 5 days |  |  |  |  | Vitamin E blocked static MF effects on blood glucose and liver glycogen |  |
| Ghodbane et al. (2015a) | Wistar male rat in vivo, brain and liver | Static MF, 128 mT, 1 h/day, 5 days | Ø lipid peroxidation in brain and liver |  |  | ↑ CAT in liver | selenium and vitamin E reversed liver catalase effect. | ↑ apoptosis in liver |
| Ghodbane et al. (2015b) | Wistar male rat in vivo, kidney and muscle | Static MF, 128 mT, 1 h/day, 5 days | ↑ lipid peroxidation in kidney |  |  | ↑ CAT in kidney | vitamin E reversed lipid peroxidation effect. | Selenium reversed MDA and calatlase effects. |
| \*Giorgi et al. (2014) | Human neuroblastoma BE(2) cells | Bipolar pulsed square wave MF, 50 Hz, 1 mT, up to 72 h | MF did not affect H2O2-induced DNA double strand break. |  |  |  |  |  |
| Glinka et al. (2013) | Male Sprague-Dawley rat in vivo, blood serum and liver | 40-Hz MF, 10 mT, 30 min /day for 6, 10, or 14 days | ↓ lipid peroxidation in liver of 6-day exposure |  |  | ↑SOD-Mn in serum only in 6 day exposure, ↓SOD-Mn in liver in 14-day exposure. No effect on SOD-ZnCu  ↑ GPx in serum in 10- and 14-day exposure |  | ↑glutathione s-transferase in liver of 6-day exposure |
| Gok et al (2014) | Wistar rat in vivo, brain and retina | 50-Hz EF, 12 kV/m, I h/day during prenatal, postnatal, and prenatal + postnatal period | ↑ lipid peroxidation in brain and retina of exposed animals |  |  |  |  | Prolonged visual evoked potentials were observed in exposed animals. |
| Goraca et al. (2010) | Male Wistar rat in vivo, heart and plasma | 40-Hz MF,  7 mT, 30 or 60 min/day, 14 days | ↑ lipid peroxidation in heart in 30 and 60 min/day exposure | ↑ H2O2 in heart in 30 and 60 min/day exposure |  | ↓ GSH in heart 60min/day |  | Total free –SH decreased in heart of 60 min/day,  decreased reducing capability in plasma of 60 min/day |
| Güler et al (2008) | Male guinea pig in vivo, liver | 50-Hz EF, 12 kV/m, 8 h/day, 7 days | ↑ lipid peroxidation | ↑NO |  | ↓SOD  ↓ GPx  ↓ MPO | Blocked by NAC |  |
| \*Güler et al (2009a) | Male guinea pig in vivo, plasma | 50-Hz EF, 12 kV/m, 8 h/day, 7 days | Ø oxidative protein damage |  |  |  |  |  |
| Güler et al (2009b) | Male guinea pig in vivo, lung | 50-Hz EF, 12 kV/m, 8 h/day, 7 days | ↑ protein carboxylation  Ø lipid peroxidation | Ø NO |  |  |  |  |
| Hajnorouzi et al. (2011) | Maize seedling | Combination of geomagnetic field (47 T) and perpendicular 10-kHz MF (22 T), 5 h/day for 4 days |  |  |  | ↓SOD |  | ↑Total antioxidant capacity, faster growth of seedlings, decrease iron increased growth |
| Hanini et al. (2017) | Mutant Pseudomonas aeruginosa without Mn- and Fe-SOD | Static magnetic field, 200 mT | ↑ lipid peroxidation |  |  | ↑ SOD, CAT, peroxidases |  | Wide type bacteria less responsive to the field |
| \*Harakawa et al. (2005) | Sprague-Dawley rat in vivo, plasma | 50-Hz EF, 17.5 kV/m, 15 min/day, 7 days | Ø lipid peroxidation (↓ in oxidatively stressed rats) |  |  |  |  | No effect on total antioxidant activity |
| Hashish et al., (2008) | Male Swiss (BALB/c) mouse in vivo, liver | Static MF (+/- 2.9 T), or 50-Hz MF 1.4 mT, 30 days | ↑ lipid peroxidation |  |  | ↓GSH in ELF-MF exposure only |  | ↑glutathione s-transferase |
| Henrykowska et al. (2009) | Human blood platelet | 50-Hz MF, 10 mT, 15 min (sinusoidal, triangular, or rectangular) | ↑ lipid peroxidation | ↑ ROS |  | ↓SOD-1  ↑catalase |  | Effects not wave-shape dependent |
| \*Hong et al. (2012) | Human breast epithelial cells (MCF10A) | 60-Hz MF, 1 mT, 4 h |  | Ø ROS level |  | Ø SOD  Ø GSH |  |  |
| Höytö et al. (2017) | Human SH-SY5Y neuroblastoma cells | 50-Hz MF, 0.1 mT, 24 h |  | ↑ cytosolic O2**.-**production;  ↓ mitochondrial O2**.-**production |  |  |  |  |
| Jajte et al. (2001) | Rat lymphocyte | 50-Hz MF, 7 mT, 3 h |  |  |  |  | DNA strand breaks induced by MF and FeCl2 blocked by melatonin. |  |
| Jajte et al. (2002) | Rat lymphocyte | Static MF, 7 mT, 3 h | ↑ lipid peroxidation with MF + FeCl2 |  |  |  |  |  |
| Jajte et al. (2003) | Rat lymphocyte | Static MF, 7 mT, 3 h | ↑ lipid peroxidation with MF + FeCl2 |  |  |  | Effect blocked by melatonin and vitamin E |  |
| Jelenković et al. (2006) | Male Wistar rat in vivo, different brain regions | 50-Hz MF, 0.5 mT, 7 days | ↑ lipid peroxidation in basal forebrain only | ↑ O2**.-**  ↑ NO |  | ↑ SOD in basal forebrain only |  | Different brain regions responded differently. |
| Jeong et al. (2006) | Male ICR mouse in vivo, brain and spinal cord | 60-Hz MF, 2 mT, 48 h |  | ↑NO | Ø  nNOS, eNOS, iNOS |  |  | Hyperalgesia observed, blocked by Ca2+ channel blocker |
| \*Jin et al. (2015) | Human lung epithelial L132 cell | 60-Hz MF, 1 or 2 mT, 9 h |  |  |  |  |  | MF did not affect H2O2-induced G2/M-arrested or aneuploid cells. |
| \*Jin et al. (2012) | Mouse embryonic fibroblast NIH3T3 and human lung fibroblast WI-38 cells | 60-Hz MF, I mT, 4 h |  |  |  |  |  | MF did not affect H2O2-induced micronucleus formation. |
| \*Jin et al. (2014) | Mouse embryonic fibroblast NIH3T3, human lung fibroblast WI-38, human lung epithelial L132, and human mammary epithelial MCF10A cells | 60-Hz MF,1 mT, 4 or 16 h |  |  |  |  |  | MF did not affect H2O2-induced DNA strand breaks. |
| Jouni et al. (2012) | Broad bean (Vicia faba L.) | Static MF, 15 mT, 8 h/day, 8 days | ↑ lipid peroxidation |  |  | ↑SOD  ↓ CAT and peroxidase |  |  |
| Kantar Gok et al. (2014) | Male Wistar rat in vivo, brain | 50-Hz EF, 12 or 18 kV/m for 2 or 4 weeks, 1 h/day | ↑ protein carboxylation in 18 kV/m 2 wk and 12 and 18 kV/m 4 wk  ↑ lipid peroxidation in all exposed groups |  |  |  |  |  |
| Kavaliers et al. (1998) | Land snail (Cepaea nemoralis) in vivo | 60-Hz MF, 0.141 mT, 15 min |  | ↑NO  (possible) |  |  |  | MF attenuated opioid-induced analgesia by increasing NO activity |
| \*Kesari et al. (2015) | Human neuroblastoma SH-SY5Y cells | 50-Hz MF, 0.1 mT, 24 h | Ø lipid peroxidation | Ø ROS change at 15, 30, and 45 days after exposure |  |  |  |  |
| Kesari et al. (2016) | Human neuroblastoma SH-SY5Y cells and rat C6 glioma cells, cells treated with menadione | 50-Hz MF, 10 or 30 T, 24 h |  | ↑ O2**.-** cytosolic and mitochondrial in C6 cells |  |  |  |  |
| Khadir et al. (1999) | Human neutrophils simulated by phorbol 12-myristate13-acetate | 60-Hz MF, 22 mT, 10 min |  | ↑ O2**.-** |  |  |  |  |
| Kim et al. (2017) | RAW 264.7 macrophage | 60-Hz MF, 0.8 mT, up to 20 h |  | ↑NO |  |  |  | Decreased effectiveness of antioxidant; increased macrophage activation |
| Koh et al. (2008) | Human prostate cancer cells (DU145, PC3, and LNCaP) | 60-Hz MF, 1 mT, 6, 24, 48, 72 h |  | ↑ H2O2 |  |  | Blocked by NAC | Apoptosis and cell cycle arrest observed. |
| Koyama et al. (2004) | pTN89 plasmids | 60-Hz MF, 5 mT, 4 h |  |  |  |  |  | MF potentiated H2O2-induced mutation |
| Koyama et al. (2008) | Human glioblastoma A172 cell | 60-Hz MF, 5 mT, 2, 4, 8, 16, or 24 h |  |  |  |  |  | MF potentiated H2O2-induced increase in apurinic/apyrimidinic sites (DNA lesion) |
| Kunt et al. (2016) | 47 electrical workers in power transmission facility, serum | Mean working period 15.9 + 6.72 yrs |  |  |  |  |  | ↑ oxidative stress index (increased total oxidant status, decreased antioxidant status) |
| Kurzeja et al. (2013) | Mouse fibroblast | Static MF, 0.4, 0.6, and 0.7 T, 4 days |  |  |  |  |  | Static MF reduced oxidative stress induced by fluoride ion by normalizing antioxidant enzymes. |
| Kuzay et al. (2017) | Healthy and diabetic male Wistar rats, testis tissue | 50-Hz MF, 8.2 mT, 20 min/day. 5 days/week. 1 month | ↑ lipid peroxidation | ↑NO |  | ↓GSH |  |  |
| Lai and Singh (1998) | Sprague-Dawley rat in vivo, brain | 60-Hz MF, 0.5 mT, 2 h |  |  |  |  | DNA strand breaks blocked by melatonin and a spin-trap compound. |  |
| Lai and Singh (2004) | Sprague-Dawley rat in vivo, brain | 60-Hz MF, 0.01 mT, 24 or 48 h |  |  |  |  | DNA strand breaks blocked by Trolox and a nitric oxide synthase inhibitor. |  |
| Lee et al. (2004) | Balb/c mouse in vivo, brain | 60-Hz MF, 1.2 mT, 3 h | ↑ lipid peroxidation | Ø O2**.-** |  | ↑SOD |  |  |
| \*Lee et al. (2012) | Mouse fibroblast NIH3T3 | 60-Hz MF, 1 mT, 4 h |  |  |  |  |  | MF did not affect H2O2-induced cellular transformation |
| Lee et al. (2010) | Human intervertebral disc cells | 60-Hz EMF, 1.8 mT, 72 h |  |  |  |  |  | EMF induced DNA synthesis blocked by NMDA, a NO blocker |
| Lewicka et al. (2015) | Human blood platelet | EMF (1 kHz, 0.5 mT; 50 Hz, 10 mT, 1 kHz, 220 V/m), 30 min | ↑ lipid peroxidation |  |  | ↑ CAT |  |  |
| \*Li et al. (2015) | Human workers performed inspection near transformers and power lines, plasma | 8-h time weighed average magnetic flux intensity 7.3 T (1.56-26.33 T), controls 0.07-0.72 T | Ø lipid peroxidation |  |  | ØSOD  Ø GPx |  | Ø Total antioxidant capacity,  no change in micronucleus frequency |
| Li et al. (2013) | Male Drosophila melanogaster in vivo | 50-Hz EMF, 72 h or long term (312 h), 3 mT |  |  |  |  |  | Short term exposure down-regulated CAT gene (endogenous antioxidant enzymes), trend of recovery with long term exposure |
| Liu et al. (2014) | Sprague-Dawley rat cerebellum neurons | 50-Hz MF, 1 mT, 1 h |  |  |  |  | Melatonin (MT) blocked MF-induced Nav current, MT2 receptor involved |  |
| Liu et al. (2002) | Mouse in vivo, brain and liver | 50-Hz EMF, 0.2 or 6 mT, 2 weeks | ↑ lipid peroxidation,  brain and liver |  |  | ↓ GSH in liver |  | ↓ decreased total antioxidant capacity in brain and liver, decreased cell membrane fluidity, synergism with lead |
| Luo et al. (2016) | ICR mouse  Blood and cerebral cortex | 50-Hz, 2-10 mT, 4 h/days. 28 days | ↑ lipid peroxidation in serum and cerebral cortex |  |  | ↓ SOD, ↓ CAT, ↓ glutathione reductase, ↓GSH-Px, and glutathione-s-transferase in serum and cerebral cortex |  |  |
| Lupke et al. (2004) | Human umbilical cord blood derived monocyte and human mono Mac 6 cells | 50-Hz MF, 1 mT, 45 min |  | ↑ total ROS, ↑ O2**.-** |  |  |  | Mono Mac 6 cells more sensitive, activation of NADPH oxidase not NADH oxidase. |
| Luukkonen et al. (2014) | Human SH-SY5Y neuroblastoma cell | 50-Hz MF, 0.1 mT, 24 h |  | ↑ ROS, ↑ H2O2 in mitochondria |  |  |  | interacts with  menadione; effects observed days after exposure |
| Malhmoudinasab et al. (2016) | Human MCF-7 cells | 50-Hz EMF, 0.25 and 0.5 mT; 5-min on/5-min off; 15-min on/15-min-off, or 30 min continuously; total exposure time 30 min |  |  |  |  |  | Changes in mRNA levels of 7 antioxidant genes |
| Manikonda et al. (2014) | Male Wistar rat in vivo, brain (hippocampus, cerebellum and cortex) | 50-Hz MF, 0.05 and 0.1 mT, 90 days | ↑ lipid peroxidation | ↑ ROS |  | ↑SOD  ↓ GSH/GSSG ratio |  | Larger response at 0.1 mT |
| Mannerling et al. (2010) | Human leukemia cell K562 | 50-Hz MF, 0.025-0.1 mT, 1 h |  | ↑ O2**.-** |  |  | Melatonin blocked MF-induced HSP70 |  |
| \*Markkanen et al. (2010) | Murine L929 fibroblast | 50-Hz MF, 0.1-0.3 mT, 1 h |  |  |  |  |  | Did not affect ROS production induced by UV. |
| Martinez et al. (2016) | Human neuroblastoma NB69 cells | 50-Hz MF, 0.1 mT, 3-h on/3-h off for 24, 42, or 63 h, or continuously for 15-120 min |  |  |  |  | MF-induced MAPK-p38 and ERK1/2 activation blocked by NAC |  |
| Martine-Samano et al. (2010) | Male Wistar rat in vivo, plasma , liver, kidney and heart | 60-Hz EMF, 2.4 mT, 2 h | Ø lipid peoxidation |  |  | ↓ SOD in plasma of MF and restrained rats  Ø CAT  ↓ GSH in heart |  | Interacts with restraint stress |
| Martine-Samano et al. (2012) | Male Wistar rat in vivo, brain | 60-Hz EMF, 2.4 mT, 2 h |  |  |  | ↓SOD  ↓ CAT  Ø GSH |  | Interacts with restraint stress |
| Martino (2011) | Human umbilical vein endothelial cell | Static MF, 0.12 and 0.03 mT (compared to 0.2-1 T), 2 days |  |  |  |  | Increased cell proliferation attenuated by SOD |  |
| Martino and Castello (2011) | Human fibrosarcoma HT1080, pancreatic AsPC-1 cancer cells, and bovine pulmonary artery endothelial cells | Static MF, geomagnetic field (45-60 T) or shielded field (0.2-2 T), 24 h |  | ↓ H2O2 in shielded samples compared to geomagnetic field |  |  |  | MnTBAP (a ROS scavenger) inhibited MF effect. |
| Miliša et al. (2017) | Euglena viridis and Paramecium caudatum | 50-Hz EF, 2.5, 5.0, 9.3 and 13.6 kV/m, 24 h |  | ↑ O2**.-** and H2O2, |  | ↑SOD |  |  |
| \*Missiha et al. (2015) | Flavin-dependent redox enzymes | Static MF, 10-160 mT, seconds |  |  |  |  |  | MF did not change enzyme kinetics. Radical pair not a mechanism of redox reaction with static MF. |
| Morabito et al. (2010a) | Rat pheochromocytoma PC-12 cell | 50-Hz MF. 0.1 or 1 mT, 30 min or 7days |  | ↑ ROS in 30 min exposure at 1 mT. |  | ↓ CAT in 0.1 and 1 mT 30-min exposure, ↑ catalase in 1 mT 7- day exposure |  | All effects were observed in undifferentiated and not in differentiated cells.  Calcium probably involved. |
| Morabito et al. (2010b) | Undifferentiated C2C12 myoblast | 50-Hz MF. 0.1 or 1 mT, 30 min |  | Ø O2**.-**  ↑ H2O2 in 1 mT exposure |  | ↑ CAT and GPx | NAC attenuated free radical increase by MF | Calcium probably involved. |
| Naarala et al. (2017) | Rat glioma C6 cells | Nearly vertical 33 T static MF plus a horizontal or a vertical 50-Hz 30 T MF, 2 h |  | ↑ cytosolic O2**.-** in vertical static field plus horizontal 50-Hz MF (but not vertical 50-Hz MF) |  |  |  |  |
| \*Nakayama et al. (2016) | Mouse macrophage (RAW 264) with or without LPS stimulation | 50-Hz MF, 0.5 mT, 24 h |  | Ø NO |  |  |  |  |
| Noda et al. (2000) | Rat brain cerebellum tissues | Pulsed DC MF, 0.1 mT, 1 h |  |  | ↑NOS |  |  | No effect from pulsed DC at 0.3 and 0.6 mT, 60 Hz (0.1 mT), and DC (3 or 20 mT) MF, no effect in hippocampus, cortex, medulla oblongagta, hypothalamus, striatum, and midbrain. |
| Osera et al. (2011) | Human neuroblastoma SH-SY5Y cells | 72-Hz pulsed EMF, 1.3 ms pulse duration, 2 mT, 72 h |  |  |  | ↑SOD-1 |  | Increased quiescent cells |
| Osera et al. (2015) | Human neuroblastoma SH-SY5Y cells | 72-Hz pulsed EMF, 1.3 ms pulse duration, 2 mT, 10, 15, or 30 min for 4 times over 7 days, or 72 h |  |  |  | ↑Mn-SOD |  | Interacts with H2O2.  Pulsed EMF prevented H2O2 –induced decrease in cell number and protein expression (HSP70). |
| Pandir and Sahingoz (2014) | Moth Ephesta kuehniella larvae | Static MF, 1.4 T; 3, 6, 12, 24, 48, or 72 h | ↑ lipid peroxidation |  |  | Exposure-time dependent  ↓ SOD, CAT, GPx and GST |  |  |
| Park et al. (2013) | Human bone marrow mesenchymal stem cells | 50-Hz EMF, 1 mT, 90 min |  | ↑ROS |  |  | Blocked by NAC |  |
| Patruno et al. (2010) | Human epidermal keratinocyte cell HaCaT | 50-Hz MF, 1 mT, 3, 18, 48 h |  | ↓ O2**.-**  ↑NO | ↑iNOS and eNOS | ↓ CAT |  | Increased cell proliferation. |
| Patruno et al. (2011) | Human epidermal keratinocyte cell HaCaT and acute myeloid leukemia THP-1 cell | 50-Hz MF, 1 mT, 24 h |  |  | ↑iNOS activity | ↑ CAT activity |  |  |
| Patruno et al. (2012) | Human acute myeloid leukemia THP-1 cell | 50-Hz MF, 1 mT, 24 h |  | ↑ O2**.-** | ↑iNOS | ↓SOD  ↓ CAT |  |  |
| Patruno et al. (2015) | Human erythro-leukemic K562 cell | 50-Hz MF, 1 mT, 24 h |  |  | ↓ iNOS reaction velocity | ↑ CAT activity |  |  |
| Politanski et al. (2010) | C57BL/g mouse in vivo, cochlear | Static MF, 5 mT, 2 h, repeated over 14 days  (also exposed to noise once) | ↑ lipid peroxidation in ‘MF + noise’ |  |  | ↑ SOD in MF, noise, and ‘MF + noise’,  ↑ CAT activity in MF, noise, and ‘MF + noise’ |  | MF interacted with noise |
| Poniedzialek et al. (2013a) | Human neutrophil | EMF tuned to calcium ion cyclotron resonance frequency (up to 60 T) |  | ↓ ROS in unstimulated cells, ↑ in phorbol 12-myristate 13-acetate stimulated cells |  |  |  |  |
| Poniedzialek et al. (2013b) | Human neutrophil | Gradient static MF, maximum value 60 mT, 15, 30 or 45 min |  | ↓ ROS in 15-min exposure, ↑ in 45-min exposure in both unstimulated and phorbol 12-myristate 13-acetate stimulated cells, effect depended on whether samples were placed close to south or north pole of magnet. |  |  |  |  |
| Pooam et al. (2017) | Human macrophage RAW264 | 50 Hz MF, 0.1 or 0.5 mT, 1, 17 or 24 h |  | ↑ O2**.-** |  |  |  |  |
| Potenza et al. (2010) | Human umbilical vein endothelial cells | Static MF, 300 mT, 4, 24, 48,and 72 h |  | ↑ROS only at 4-h exposure coincided with DNA damage |  |  |  |  |
| Rageh et al. (2012) | 10 -day old rat in vivo, brain | 50-Hz MF. 0.5 mT, 30 days (24 h/day) | ↑ lipid peroxidation |  |  | ↑ SOD  Ø GSH |  |  |
| Raggi et al. (2008) | Human blood sample | Magnetic therapy device based on ion cyclotron resonance | ↓ lipid peroxidation immediately and one month after exposure |  |  |  |  |  |
| Rajabbeigi et al. (2013) | Parsley cell | Static MF, 30 mT, 6 or 12 h |  |  |  | ↑ CAT with MF  ↓ CAT with ‘MF + iron’ |  | ↓ ascorbate peroxidase |
| Rauš Balind et al. (2014) | Gerbil subjected to 10-min global cerebral Ischemia in vivo, brain(forebrain, striatum and hippocampus) | 50-Hz MF, 0.5 mT, 7 days |  |  |  |  |  | MF decreased oxidative stress induced by ischemia (NO, SOD, MDA, O2**.-)** |
| Reale et al. (2006) | Human blood monocytes | 50-Hz EMF, 1 mT, overnight |  |  | ↓ iNOS |  |  |  |
| Reale et al. (2014) | Human neuroblastoma cell SH-SY5Y | 50-Hz MF, 1 mT, 1, 3, 6 or 24 h |  | ↑ O2**.-** | ↑ NOS, peaked at 1 h | ↑ CAT |  | MF enhanced oxidative effects of H2O2 (↓catalase, ↑ O2**.-).** |
| Regoli et al. (2005) | Snail Helix aspersa in vivo, digestive gland | 50-Hz MF, 0.5, 2.5,10 and 50 T, 10 days in lab; 2.88 and 0.75 T for 10, 20, 40, 60 days in field | Lab: Ø lipid peroxidation  Field: ↑ in 2.88 T more than 10 days and 0.75 T more than 20 days |  |  | Lab:↓ CAT in 50 T 10 days  Field: ↓ CAT in 2.88 T more than 10 days and 0.75 T more than 40 days  Lab: Ø GSH,  ↓ Glutathione reductase  Field:  ↓ glutathione reductase |  | Total oxyradical scavenger capacity:  Lab: ↓OH and ROO;  Field: ↑OH and ↓ROO |
| Rollwitz et al. (2004) | Mouse bone marrow-derived promonocytes and macrophage | 50-Hz MF, 1 mT, 45 min-24 h |  | ↑ ROS, ↑ O2**.-** |  |  |  | NADH-oxidase (not NADPH pathway) involved. |
| Roy et al. (1995) | Phorbol 12-myristate 13- acetate-stimulated rat neutrophil | 60-Hz MF, 0.1 mT |  | ↑ ROS |  |  |  |  |
| Sadeghipour et al. (2012) | Human breast carcinoma cell (T47D) | 100 and 217 Hz pulsed EMF, 0.1 mT, 24-72 h |  | ↑ ROS in 217 Hz 72-h, not in 100 Hz exposure |  |  |  |  |
| Sahebjamei et al. (2007) | Cultured tobacco cell | Static MF, 10 and 30 mT, 5 h/day, 5 days | ↑ lipid peroxidation |  |  | ↑SOD  ↓ CAT and ascorbate peroxidase |  |  |
| Salunke et al. (2014) | Swiss albino mouse in vivo, brain | 50-Hz MF, 1 mT, 8 h/day for 7, 30, 60, 90 and 120 days |  | ↑ NO in cortex, hippocampus, and hypothalamus |  |  |  |  |
| Seifirad et al. (2014) | Male Wistar rat in vivo, serum | 60-Hz MF, 0.5 mT, 4 h or 4 h/day 14 days | ↑lipid peroxidation immediately after and at 72 h after chronic exposure, Ø acute exposure |  |  |  |  | Total antioxidant activity: ↑ immediately after acute exposure (not at 3 days post-exposure), ↓immediately and 3 days after chronic exposure. |
| Selaković et al. (2013) | Male gerbils 3- and 10- month old in vivo, Forebrain cortex, striatum hippocampus, and cerebellum | 50-Hz MF, 0.5, 0.25 and 0.1 mT, 7 days | ↑ lipid peroxidation | ↑ O2**.-**  ↑NO |  | ↑SOD |  | Dose-response observed, effects smaller and recovered faster in 3-month than in 10-month old animals. |
| Sharifian et al. (2009) | Human welders occupational exposure, serum and red blood cells | 50-Hz EMF, 8.8-84T, 20-133 V/m, 40 h/week (6 days/ week) |  |  |  | ↓SOD  ↓GPX |  | Ø Total serum antioxidant status, a significant negative correlation between SOD/GPX and MF intensity was observed. |
| Shine et al (2012) | Soybean seeds | Static MF 150 and 200 mT, 1 h |  | ↑ O2**.-,** OH, H2O2, |  | ↓SOD & ascorbate peroxidase |  |  |
| Simko et al. (2001) | Mouse bone marrow-derived macrophage | 50-Hz MF, 0.5-1.5 mT, 45 min |  | ↑ O2**.-** |  |  |  | Increased phagocytic activity. |
| Sirmatel et al. (2007a) | Male human  blood | 1,5 T static MF from a MRI machne, 30 min |  |  |  |  |  | ↑ total antioxidant capacity; ↓ total oxidant ststus and oxidative stress index |
| Sirmatel et al. (2007b) | Male human  blood | 1,5 T static MF from a MRI machne, 30 min |  | ↑ NO (based on nitrite and nitrate leveks) |  |  |  |  |
| Solek et al. (2017) | Mouse spermatogenic cell lines | 2, 50, 120 Hz pulsed (1 sec on/1 sec off) and continuous-wave EMF, 2.5-8 mT, 2 h |  | ↑ O2**.-**  ↑ NO |  |  |  | Cell cycle arrest and apoptosis observed |
| Sullivan et al. (2011) | Various human cell lines | Static MF, 35-120 mT |  | Static MF ↑ ROS at 18 h (not at 5 days) of exposure in fetal lung (WI38) cells. |  |  |  |  |
| Sun et al. (2015) | Preosteoclast cell line RAW264.7 | Large gradient high magnetic fields (12 T,  -1370 T2/m; 12 T, 1370 T2/m), 48 h |  | ↓ NO |  |  |  |  |
| Tang et al. (2016) | Human Jurket cell and stimulated mouse primary T cell | 7.5 Hz MF, 0.4 T, 2 h |  | ↑ ROS |  |  |  |  |
| Tasset et al. (2012) | Male Wistar rat in vivo, brain | 60-Hz MF, 0.7 mT, 2 h in the morning and 2 h in the afternoon for 21 days (applied to the head) | Ø DNA oxidative damage  Ø lipid peroxidation |  |  | Ø GSH  ↓ GSSG |  | MF reversed 3-nitropropionic acid induced oxidative stress. |
| Tayefi et al. (2010) | Wistar rat pup in vivo, myocardium | 50-Hz MF, 3 mT, 4/h per day during gestation and to 20 day postnatal | ↑ lipid peroxidation |  |  | ↓ SOD |  |  |
| Todorovic et al. (2012) | Eggs of Baculum extradentatum (insert also known as Vietnamese walking stick) | Stattic MF, 50 mT; 50-Hz MF, 6 mT; exposed until completion og embryonic development |  |  |  | ↑SOD and CAT  Ø GSH |  |  |
| Túnez et al. (2006) | Male Wistar rat in vivo, striatum | 60-Hz MF, 0.7 mT, 2 h in the morning and 2 h in the afternoon for 4 days (applied to the head) |  |  |  |  |  | MF itself had no effect on different oxidative parameters, but reduced 3-nitropropionic acid induced oxidative and nitrosative stress. |
| \*Türközer et al. (2008) | Guinea pig in vivo, brain | 50-Hz EF, 2, 2.5, 3, 3.5, 4, 4.5, 5 V/m, 8 h/day, 3 days | Ø lipid peroxidation |  |  | Ø SOD  Ø CAT and GPx |  |  |
| \*Vannoni et al. (2012) | Human osteoarthritic chondrocyte | 100-Hz EMF and a field containing various frequencies |  | Ø ROS |  | Ø GSH |  |  |
| Vignola et al. (2012) | Female Wistar rat with drug-induced myopathy, in vivo, muscle | Pulsed EMF, 50-Hz carrier frequency, 20 mT, 30 min/day, 8 days, assayed 8 days after exposure |  | ↓NO |  | ↓SOD |  | Pulsed EMF caused muscle recovery. |
| \*Villarini et al. (2017) | SH-SY5Y5 and SK-N-BE-2 human neuroblastoma cells | 50-Hz MF; 0.01, 0.1, or 1 mT; 1 h continuously or 5 h intermittently | Ø DNA damage |  |  | Ø GSH/GSSG ratio |  |  |
| Wartenberg et al. (2008) | Oral mucosa cancer cell (UM-SCC-14-C) | DC EF, 4 V/m, 24 h |  |  |  | ↑ Cu/Zn SOD  Ø CAT  ↓GSH | Effects blocked by NAC. | Increased apoptosis and decreased cell proliferation. |
| Wolf et al. (2005) | HL-60 leukemia cells, Rat-1 fibroblast, WI-38 diploid fibroblast | 50-Hz EMF, 0.5-1 mT, 24-72 h | ↑ DNA oxidative damage | ↑ ROS in Rat-1 fibroblast |  |  | Effect blocked by alpha-tocopherol. | Dose-dependent increase in cell proliferation observed. |
| Wu et al. (2016) | Male mice, liver | Static E-field, 9.2-21.85 kV/m, 2.3-15.4 kV/m, and 0 kV/m, 35 days | Ø lipid peroxidation |  |  | ↑ SOD |  | No effect on glutathione-transferase and glutathione peroxidase |
| Yin et al. (2016) | Primary cultured rat hippocampal neurons | 50-Hz MF, 8 mT, 90 min | ↑ lipid peroxidation | ↑ ROS |  | ↓SOD |  |  |
| Yokus et al. (2005) | Female Wistar rat in vivo, leukocytes and plasma | 50-Hz MF, 0.97 mT, 3 h/day for 50 or 100 days | ↑ DNA oxidative damage  ↑lipid peroxidation |  |  |  |  | Larger effects with longer exposure. |
| Yokus et al. (2008) | Male Sprague-Dawley rat in vivo, leukocytes | 50-Hz MF, 0.1 and 0.5 mT, 2 h/day for 10 months | ↑ different forms of oxidative DNA damage in 0.1 mT group |  |  |  |  |  |
| \*Yoon et al. (2014) | Human lung fibroblast W138 and human lung epithelial L132 cells | 60-Hz MF, 1 or 2 mT, 6 h |  |  |  |  |  | MF did not enhance H2O2-induced double strand DNA breaks.(MF potentiated infra-red induced breaks). |
| \*Yoshikawa et al. (2000) | Male BALB/C mouse injected with lipopolysaccharide (LPS) in vivo, liver | 60-Hz MF, 0.1 mT, 5.5 h |  |  |  |  |  | MF did not induce NO generation, but enhanced LPS-induced NO generation. |
| Zhao et al. (2011) | Human-hamster hybrid(A9L)), mitochondria-deficient (p(0)A(L)) cells, double-strand break repair- deficient (XRS-5) cells | Static MF, 8.5 T, 3 h |  | ↑ ROS |  |  |  |  |
| Zwirska-Korczala et al. (2004) | Murine squamous carcinoma AT478 cell | Mixture of frequencies up to 400 Hz, MF, 0.11 mT, 16 min, assayed 24 and 72 h after exposure | ↑lipid peroxidation |  |  | ↑ MnSOD and Cu/ZnSOD  Ø GPx | Effects attenuated by melatonin |  |
| Zmylony et al. (2004a) | Rat lymphocytes stimulate by FeCl2 | 50-HZ MF, 20, 40, or 200 T, 5 or 60 min |  | ↓ ROS in Fe and 40 T MF exposed cells (AC MF has to be directed along the earth’s static MF). |  |  |  |  |
| Zmylony et al. (2004b) | Rat lymphocytes | 50-HZ MF, 40 T, 5 or 60 min | MF enhanced DNA damage caused by ultraviolet radiation (UVA). (UVA damages DNA via free radicals.) |  |  |  |  |  |

**Literature list (E= 162 (87%); NE= 24 (13%)) (E= paper reported effect; NE= paper reported no significant effect).**

**(VT = in vitro; VO= in vivo; HU= human study; CE = long-term/repeated exposure; AE= acute exposure; LI = low intensity; IFR= increase free radical; DFR= decrease free radical; IOD = increase oxidative damages; DOD = decrease oxidative damages; IAO =increase antioxidant activity; DAO= decrease antioxidant activity; AO= effect of antioxidant; IX= interaction with other factor; MC= mechanism)**

**Aïda Lahbib, Soumaya Ghodbane, Mohsen Sakly & Hafedh Abdelmelek. Vitamins and glucose metabolism: The role of static magnetic fields.  International Journal of Radiation Biology. Posted online on August 4, 2014. (doi:10.3109/09553002.2014.930537) (review)**  
  
Purpose: This review focuses on our own data and other data from the literature of static magnetic fields (SMF) bioeffects and vitamins and glucose metabolism. Three main areas of investigation have been covered: Static magnetic field and glucose metabolism, static magnetic field and vitamins and the role of vitamins on glucose metabolism. Conclusion: Considering these articles comprehensively, the conclusions are as follows: The primary cause of changes in cells after incubation in external SMF is disruption of free radical metabolism and elevation of their concentration. Such disruption causes oxidative stress leading to an unsteadiness of glucose level and insulin release. Moreover, based on available data, it was concluded that exposure to SMF alters plasma levels of vitamin A, C, D and E; these parameters can take part in disorder of glucose homeostasis and insulin release.

**(E) (VT, AE, IFR; IAO)** [**Akan Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akan%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=20809504)**,** [**Aksu B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Aksu%20B%5BAuthor%5D&cauthor=true&cauthor_uid=20809504)**,** [**Tulunay A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Tulunay%20A%5BAuthor%5D&cauthor=true&cauthor_uid=20809504)**,** [**Bilsel S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bilsel%20S%5BAuthor%5D&cauthor=true&cauthor_uid=20809504)**,** [**Inhan-Garip A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Inhan-Garip%20A%5BAuthor%5D&cauthor=true&cauthor_uid=20809504)**. Extremely low-frequency electromagnetic fields affect the immune response of monocyte-derived macrophages to pathogens.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/20809504) **31(8):603-612, 2010.**

This study aimed to determine the effect of extremely low-frequency electromagnetic fields (ELF-EMF) on the physiological response of phagocytes to an infectious agent. THP-1 cells (human monocytic leukemia cell line) were cultured and 50 Hz, 1 mT EMF was applied for 4-6 h to cells induced with Staphylococcus aureus or interferon gamma/lipopolysaccharide (IFγ/LPS). Alterations in nitric oxide (NO), inducible nitric oxide synthase (iNOS) levels, heat shock protein 70 levels (hsp70), cGMP levels, caspase-9 activation, and the growth rate of S. aureus were determined. The growth curve of exposed bacteria was lower than the control. Field application increased NO levels. The increase was more prominent for S. aureus-induced cells and appeared earlier than the increase in cells without field application. However, a slight decrease was observed in iNOS levels. Increased cGMP levels in response to field application were closely correlated with increased NO levels. ELF-EMF alone caused increased hsp70 levels in a time-dependent manner. When cells were induced with S. aureus or IFγ/LPS, field application produced higher levels of hsp70. ELF-EMF suppressed caspase-9 activation by a small extent. These data confirm that ELF-EMF affects bacterial growth and the response of the immune system to bacterial challenges, suggesting that ELF-EMF could be exploited for beneficial uses.

**(E) (VO, CE, DFR)** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Akdag%20MZ%22%5BAuthor%5D)**,** [**Bilgin MH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bilgin%20MH%22%5BAuthor%5D)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dasdag%20S%22%5BAuthor%5D)**,** [**Tumer C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tumer%20C%22%5BAuthor%5D)**. Alteration of nitric oxide production in rats exposed to a prolonged, extremely low-frequency magnetic field.** [**Electromagn Biol Med.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Electromagn%20Biol%20Med.');) **26(2):99-106, 2007.**

The purpose of this study is to investigate the possible effect of an extremely low-frequency magnetic field (ELF-MF) on nitric oxide (NO) level. In this study, 27 male Sprague-Dawley rats were used. The rats were divided into three groups: two experimental and one control (sham-exposed). The first and second experimental group (n = 10) were exposed to 100 microT and 500 microT ELF-MF during 10 months, 2 h a day, respectively, and the third (n = 7) group was treated like an experimental group except for ELF-MF exposure in methacrylate boxes. After ELF-MF and sham exposure, serum nitrite levels were measured by Griess reaction. A significant reduction was observed in nitrite levels among the first and second experimental groups of rats and sham-exposed rats after exposure for 10 months, 2 h a day, to ELF-MF of 100 and 500 microT (p < 0.01). These results suggest that prolonged ELF-MF exposure at intensities of exposure limits, determined by ICNIRP for public and occupational, may reduce NO production probably affected by NO generation pathways.

**(E)** **(VO, CE, DAO)** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Akdag%20MZ%22%5BAuthor%5D)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dasdag%20S%22%5BAuthor%5D)**,** [**Ulukaya E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ulukaya%20E%22%5BAuthor%5D)**,** [**Uzunlar AK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Uzunlar%20AK%22%5BAuthor%5D)**,** [**Kurt MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kurt%20MA%22%5BAuthor%5D)**,** [**Taşkın A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ta%C5%9Fk%C4%B1n%20A%22%5BAuthor%5D)**. Effects of extremely low-frequency magnetic field on caspase activities and oxidative stress values in rat brain.** [**Biol Trace Elem Res.**](javascript:AL_get(this,%20'jour',%20'Biol%20Trace%0d%0a%20Elem%20Res.');) **138(1):238-249, 2010.**

This study was aimed to investigate the effect of extremely low-frequency magnetic field (ELF-MF) on apoptosis and oxidative stress values in the brain of rat. Rats were exposed to 100 and 500 microT ELF-MF, which are the safety standards of public and occupational exposure for 2 h/day for 10 months. Brain tissues were immunohistochemically stained for the active (cleaved) caspase-3 in order to measure the apoptotic index by a semi-quantitative scoring system. In addition, the levels of catalase (CAT), malondialdehyde (MDA), myeloperoxidase (MPO), total antioxidative capacity (TAC), total oxidant status (TOS), and oxidative stress index (OSI) were measured in rat brain. Final score of apoptosis and MPO activity were not significantly different between the groups. CAT activity decreased in both exposure groups (p < 0.05), while TAC was found to be lower in ELF 500 group than those in ELF-100 and sham groups (p < 0.05). MDA, TOS, and OSI values were found to be higher in ELF-500 group than those in ELF-100 and sham groups (p < 0.05). In conclusion, apoptosis was not changed by long-term ELF-MF exposure, while both 100 and 500 microT ELF-MF exposure induced toxic effect in the rat brain by increasing oxidative stress and diminishing antioxidant defense system.

**(E) (VO, CE, IOD)** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akdag%20MZ%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Dasdag%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**,** [**Cakir DU**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cakir%20DU%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**,** [**Yokus B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yokus%20B%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**,** [**Kizil G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kizil%20G%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**,** [**Kizil M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kizil%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23324065)**. Do 100- and 500-μT ELF magnetic fields alter beta-amyloid protein, protein carbonyl and malondialdehyde in rat brains?** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23324065) **32(3):363-372, 2013a.**

Several studies still state that presently accepted safety standards for extremely low-frequency magnetic fields (ELF-MFs) do not provide adequate protection, and therefore the standards are still open to question. To help resolve this question, the aim of this study was to illuminate the interaction between biomolecules and ELF-MFs by investigating the effect of ELF-MFs on beta-amyloid protein (BAP), protein carbonyl (PC) and malondialdehyde (MDA) in rat brain. For this study, 30 adult male Sprague-Dawley rats were used, which were divided into two experimental groups and a sham exposed group. Rats in two experimental groups were exposed to 100- and 500-μT ELF-MFs (50 Hz) for 2 h/day for 10 months, which are the generally accepted safety standards for public and occupational exposures. The same procedures were applied to the rats in the sham group, but with the generator turned off. The results of this study showed that neither ELF-MFs used in this study altered BAP level significantly (p>0.05). However, PC and MDA levels were increased by the exposure to 100- and 500-μT ELF-MFs (p < 0.0001). In conclusion, both PC and MDA levels were altered by long-term exposure to either 100 or 500 μT ELF-MF. However, many further and more comprehensive studies will be required to elucidate the interaction mechanisms between ELF-MFs exposure and living organisms.

**(NE)** **(VO, CE)** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akdag%20MZ%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Dasdag%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Uzunlar AK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Uzunlar%20AK%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Ulukaya E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ulukaya%20E%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Oral AY**](http://www.ncbi.nlm.nih.gov/pubmed?term=Oral%20AY%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Celik N**](http://www.ncbi.nlm.nih.gov/pubmed?term=Celik%20N%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**,** [**Akşen F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ak%C5%9Fen%20F%5BAuthor%5D&cauthor=true&cauthor_uid=23786626)**. Can safe and long-term exposure to extremely low frequency (50 Hz) magnetic fields affect apoptosis, reproduction, and oxidative stress?** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/23786626) **89(12):1053-1060, 2013b.**

Abstract Purpose: The purpose of this study was to determine whether 50 Hz Extremely Low Frequency-Magnetic Fields (ELF-MFs) affects apoptotic processes, oxidative damage, and reproductive characteristics such as sperm count and morphology in rat testes. Materials and Methods: 30 male Sprague-Dawley rats were used in the present study, which were divided into three groups (sham group, n: 10, and two experimental groups, n: 10 for each group).Rats in the experimental group were exposed to 100 and 500 μT ELF-MF (2h/day, 7 days/week, for 10 months) corresponding to exposure levels that are considered safe for humans.. Same experimental procedures were applied to the sham group, but the ELF generator was turned off. Tissues from the testes were immunohistochemically stained for active (cleaved) caspase-3 in order to measure the apoptotic index by a semi-quantitative scoring system. The levels of catalase (CAT), malondialdehyde (MDA), myeloperoxidase (MPO), total antioxidative capacity (TAC), total oxidant status (TOS), and oxidative stress index (OSI) were also measured. Additionally, epididymal sperm count and sperm morphology was evaluated. Results: There were no significant differences in the reproductive and oxidative stress parameters between the sham group and the exposed groups (p>0.05). While no difference was observed between the final apoptosis score of the sham and the100 µT ELF-MF group (p>0.05), the final apoptosis score was higher in the 500 µT ELF-MF exposure group than in the sham group (p<0.05). Conclusion: Long-term exposure to 100 µT and 500 µT ELF-MF did not affect oxidative or antioxidative processes, lipid peroxidation, or reproductive components such as sperm count and morphology in testes tissue of rats. However, long-term exposure to 500 µT ELF-MF did affect active-caspase-3 activity, which is a well-known apoptotic indicator.

**(E)** **(VO, CE, IOD)** [**Akpinar D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akpinar%20D%5BAuthor%5D&cauthor=true&cauthor_uid=23045992)**,** [**Ozturk N**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ozturk%20N%5BAuthor%5D&cauthor=true&cauthor_uid=23045992)**,** [**Ozen S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ozen%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23045992)**,** [**Agar A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Agar%20A%5BAuthor%5D&cauthor=true&cauthor_uid=23045992)**,** [**Yargicoglu P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yargicoglu%20P%5BAuthor%5D&cauthor=true&cauthor_uid=23045992)**. The effect of different strengths of extremely low-frequency electric fields on antioxidant status, lipid peroxidation, and visual evoked potentials.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23045992) **31(4):436-448, 2012.**

The aim of the study was to investigate the effects of extremely low-frequency electric field (ELF EF) on visual evoked potential (VEP), thiobarbituric acid reactive substances (TBARS), total antioxidant status (TAS), total oxidant status (TOS), and oxidant stress index (OSI). Thirty female Wistar rats, aged 3 months, were divided into three equal groups: Control (C), the group exposed to EF at 12 kV/m strength (E12), and the group exposed to EF at 18 kV/m strength (E18). Electric field was applied to the E12 and E18 groups for 14 days (1 h/day). Brain and retina TBARS, TOS, and OSI were significantly increased in the E12 and E18 groups with respect to the control group. Also, TBARS levels were significantly increased in the E18 group compared with the E12 group. Electric fields significantly decreased TAS levels in both brain and retina in E12 and E18 groups with respect to the control group. All VEP components were significantly prolonged in rats exposed to electric fields compared to control group. In addition, all latencies of VEP components were increased in the E18 group with respect to the E12 group. It is conceivable to suggest that EF-induced lipid peroxidation may play an important role in changes of VEP parameters.

**(E) (VO, CE, DOD, IOD)** [**Akpınar D**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Akp%C4%B1nar%20D%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Kantar Gok D**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kantar%20Gok%20D%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Hidisoglu E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hidisoglu%20E%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Aslan M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Aslan%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Ozen S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ozen%20S%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Agar A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Agar%20A%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**,** [**Yargicoglu P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Yargicoglu%20P%5BAuthor%5D&cauthor=true&cauthor_uid=27070942)**. Effects of pre- and postnatal exposure to extremely low-frequency electric fields on mismatch negativity component of the auditory event-related potentials: Relation to oxidative stress.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/27070942) **2016 Apr 12:0. [Epub ahead of print]**

In our previous study, the developmental effects of extremely low-frequency electric fields (ELF-EF) on visual and somatosensory evoked potentials in adult rats were studied. There is no study so far examining the effects of 50 Hz electric field (EF) on mismatch negativity (MMN) recordings after exposure of rats during development. Therefore, our present study aimed to investigate MMN and oxidative brain damage in rats exposed to EF (12 kV/m, 1 h/day). Rats were divided into four groups, namely control (C), prenatal (Pr), postnatal (Po), and prenatal+postnatal (PP). Pregnant rats of Pr and PP groups were exposed to EF during pregnancy. Following birth, rats of PP and Po groups were exposed to EF for three months. After exposure to EF, MMN was recorded by electrodes positioned stereotaxically to the surface of the dura, and then brain tissues were removed for histological and biochemical analyses. The MMN amplitude was higher to deviant tones than to standard tones. It was decreased in all experimental groups compared with the C group. 4-Hydroxy-2-nonenal (4-HNE) levels were significantly increased in the Po group with respect to the C group, whereas they were significantly decreased in the PP group compared with Pr and Po groups. Protein carbonyl levels were significantly decreased in the PP group compared with C, Pr, and Po groups. EF decreased MMN amplitudes were possibly induced by lipid peroxidation.

**(E)(VO, CE, IOD)** [**Aksen F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Aksen%20F%22%5BAuthor%5D)**,** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Akdag%20MZ%22%5BAuthor%5D)**,** [**Ketani A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ketani%20A%22%5BAuthor%5D)**,** [**Yokus B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yokus%20B%22%5BAuthor%5D)**,** [**Kaya A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kaya%20A%22%5BAuthor%5D)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dasdag%20S%22%5BAuthor%5D)**. Effect of 50-Hz 1-mT magnetic field on the uterus and ovaries of rats (electron microscopy evaluation).** [**Med Sci Monit.**](javascript:AL_get(this,%20'jour',%20'Med%20Sci%20%0d%0aMonit.');) **12(6):BR215-220, 2006.**

BACKGROUND: The aim of this study was to investigate the effect of extremely low frequency magnetic fields (ELFMF) on the uterus and ovary of rats. MATERIAL/METHODS: Forty-eight female Wistar albino rats were divided into two groups, one for 50 and the other for 100 days of exposure. Each group was further divided into two groups, one sham exposed (n=12) and the other the experimental group (n=12). The experimental rats were exposed to 50-Hz 1-mT ELFMF for three hours/day for 50 or 100 days. The sham groups of rats were kept under the same circumstances without applying ELFMF. Electron microscopic examination was performed to evaluate the ovaries and uterus. RESULTS: Ultrastructural dissolution, decrease in cell organelles, cavities in cells, heterochromative appearance, and typical structural loss of the nucleus were observed in germinal epithelial cells of the rat ovaries in the 50-days ELFMF exposure group. Ultrastructural alterations in germinal epithelium and tunica albuginea of ovaries, irregularity in nucleus and nucleolus, increase in lipid vacuoles of cell cytoplasm and reduction in organelles were observed in rat ovaries in the 100-days ELFMF exposure group. Similar alterations were observed in uterus. Malondialdehyde concentration (MDA) of the ovaries and uterus increased in rats of the two exposure groups (p<0.001). CONCLUSIONS: The results of the study showed that 50 and 100 days of exposure to a 1-mT ELFMF can cause alterations at the cellular level and in MDA concentration.

**(NE)** **(VO, CE, AO)** [**Alcaraz M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Alcaraz%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23781994)**,** [**Olmos E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Olmos%20E%5BAuthor%5D&cauthor=true&cauthor_uid=23781994)**,** [**Alcaraz-Saura M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Alcaraz-Saura%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23781994)**,** [**Achel DG**](http://www.ncbi.nlm.nih.gov/pubmed?term=Achel%20DG%5BAuthor%5D&cauthor=true&cauthor_uid=23781994)**,** [**Castillo J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Castillo%20J%5BAuthor%5D&cauthor=true&cauthor_uid=23781994)**. Effect of long-term 50 Hz magnetic field exposure on the micronucleated polychromatic erythrocytes of mice.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23781994) **33(1):51-57, 2014.**

In recent years extremely low-frequency magnetic fields (ELF-EMF) have become widely used in human activities, leading to an increased chance of exposure to ELF-EMF. There are few reports on in vivo mammalian genotoxic effects using micronucleus (MN) assays, which generally have been used as a short-term screening system. We analyzed the possible genotoxic effect induced by long-term exposure (7, 14, 21, 28 d) of a 50 Hz ELM-MF to mice by measuring the increase in frequency of micronucleated polychromatic erythrocyte in their bone marrow (MNPCEs) and we compared it with that induced by 50 cGy of X-rays. Subsequently, we tried to reduce this chromosomal damage by administering four antioxidants substances with radioprotective capacities: dimethyl sulfoxide (DMSO), 6-n-propyl-2-thiouracil (PTU), grape-procyanidins (P) and citrus flavonoids extract (CE). The increase in micronucleated cells was higher in both physical treatments (Control < ELF-EMF (p < 0.01) <X-rays (p > 0.001)); however, the antioxidant substances only showed a genoprotective capacity against the damage induced by ionizing radiation (Ci > PTU = DMSO (p < 0.001) >P = CE (p < 0.001). The 50 Hz ELM-MF increased MNPCEs in mouse bone marrow, expressing a genotoxic capacity. Administration of antioxidant substances with radioprotective capacities known to act through the elimination of free radicals did not diminish the genotoxic effect induced by ELM-MF.

**(E) (VO, AE, CE, IAO)** [**Ansari AM**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ansari%20AM%5BAuthor%5D&cauthor=true&cauthor_uid=26764231)**,** [**Farzampour S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Farzampour%20S%5BAuthor%5D&cauthor=true&cauthor_uid=26764231)**,** [**Sadr A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sadr%20A%5BAuthor%5D&cauthor=true&cauthor_uid=26764231)**,** [**Shekarchi B**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Shekarchi%20B%5BAuthor%5D&cauthor=true&cauthor_uid=26764231)**,** [**Majidzadeh-A K**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Majidzadeh-A%20K%5BAuthor%5D&cauthor=true&cauthor_uid=26764231)**. Effects of short term and long term extremely low frequency magnetic field on depressive disorder in mice: Involvement of nitric oxide pathway.** [**Life Sci.**](http://www.ncbi.nlm.nih.gov/pubmed/26764231) **2016 Jan 4. pii: S0024-3205(15)30148-X. doi: 10.1016/j.lfs.2015.12.055. [Epub ahead of print]**

AIMS: Previous reports on the possible effects of Extremely Low Frequency Magnetic Fields (ELF MF) on mood have been paradoxical in different settings while no study has yet been conducted on animal behavior. In addition, it was shown that ELF MF exposure makes an increase in brain nitric oxide level. Therefore, in the current study, we aimed to assess the possible effect(s) of ELF MF exposure on mice Forced Swimming Test (FST) and evaluate the probable role of the increased level of nitric oxide in the observed behavior. MAIN METHODS: Male adult mice NMRI were recruited to investigate the short term and long term ELF MF exposure (0.5 mT and 50Hz, single 2h and 2weeks 2h a day). Loco motor behavior was assessed by using Open-Field Test (OFT) followed by FST to evaluate the immobility time. Accordingly, NΩ-nitro-L-arginine methyl ester 30mg/kg was used to exert anti-depressant like effect. KEY FINDINGS: According to the results, short term exposure did not alter the immobility time, whereas long term exposure significantly reduces immobility time (p<0.01). However, it was revealed that the locomotion did not differ among all experimental groups. Short term exposure reversed the anti-depressant like effect resulting from 30mg/kg of NΩ-nitro-L-arginine methyl ester (p<0.01). SIGNIFICANCE: It has been concluded that long term exposure could alter the depressive disorder in mice, whereas short term exposure has no significant effect. Also, reversing the anti-depressant activity of L-NAME indicates a probable increase in the brain nitric oxide.

**(E) (VO, AE, IAO)** [**Asghar T**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Asghar%20T%5BAuthor%5D&cauthor=true&cauthor_uid=27835746)**,** [**Jamil Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Jamil%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=27835746)**,** [**Iqbal M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Iqbal%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27835746)**,** [**Zia-Ul-Haq**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zia-Ul-Haq%5BAuthor%5D&cauthor=true&cauthor_uid=27835746)**,** [**Abbas M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Abbas%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27835746)**. Laser light and magnetic field stimulation effect on biochemical, enzymes activities and chlorophyll contents in soybean seeds and seedlings during early growth stages.** [**J Photochem Photobiol B.**](https://www.ncbi.nlm.nih.gov/pubmed/27835746) **165:283-290, 2016.**

Laser and magnetic field bio-stimulation attracted the keen interest of scientific community in view of their potential to enhance seed germination, seedling growth, physiological, biochemical and yield attributes of plants, cereal crops and vegetables. Present study was conducted to appraise the laser and magnetic field pre-sowing seed treatment effects on soybean sugar, protein, nitrogen, hydrogen peroxide (H2O2) ascorbic acid (AsA), proline, phenolic and malondialdehyde (MDA) along with chlorophyll contents (Chl "a" "b" and total chlorophyll contents). Specific activities of enzymes such as protease (PRT), amylase (AMY), catalyst (CAT), superoxide dismutase (SOD) and peroxides (POD) were also assayed. The specific activity of enzymes (during germination and early growth), biochemical and chlorophyll contents were enhanced significantly under the effect of both laser and magnetic pre-sowing treatments. Magnetic field treatment effect was slightly higher than laser treatment except PRT, AMY and ascorbic acid contents. However, both treatments (laser and magnetic field) effects were significantly higher versus control (un-treated seeds). Results revealed that laser and magnetic field pre-sowing seed treatments have potential to enhance soybean biological moieties, chlorophyll contents and metabolically important enzymes (degrade stored food and scavenge reactive oxygen species). Future study should be focused on growth characteristics at later stages and yield attributes.

**(E) (VT, CE, IFR)** [**Ayşe IG**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ay%C5%9Fe%20IG%5BAuthor%5D&cauthor=true&cauthor_uid=20707646)**,** [**Zafer A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zafer%20A%5BAuthor%5D&cauthor=true&cauthor_uid=20707646)**,** [**Sule O**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sule%20O%5BAuthor%5D&cauthor=true&cauthor_uid=20707646)**,** [**Işil IT**](http://www.ncbi.nlm.nih.gov/pubmed?term=I%C5%9Fil%20IT%5BAuthor%5D&cauthor=true&cauthor_uid=20707646)**,** [**Kalkan T**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kalkan%20T%5BAuthor%5D&cauthor=true&cauthor_uid=20707646)**. Differentiation of K562 cells under ELF-EMF applied at different time courses.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/20707646) **29(3):122-130, 2010.**

The time-course of ELF-EMF application to biological systems is thought to be an important parameter determining the physiological outcome. This study investigated the effect of ELF-EMF on the differentiation of K562 cells at different time courses. ELF-EMF (50 Hz, 5 mT, 1 h) was applied at two different time-courses; first at the onset of hemin induction for 1 h, and second, daily 1 h for four days. While single exposure to ELF-EMF resulted in a decrease in differentiation, ELF-EMF applied everyday for 1 h caused an increase in differentiation. The effect of co-stressors, magnesium, and heat-shock was also determined and similar results were obtained. ELF-EMF increased ROS levels in K562 cells not treated with hemin, however did not change ROS levels of hemin treated cells indicating that ROS was not the cause. Overall, these results imply that the time-course of application is an important parameter determining the physiological response of cells to ELF-EMF.

**(E) (VT, AC, IX)** [**Bawin SM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bawin%20SM%22%5BAuthor%5D)**,** [**Satmary WM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Satmary%20WM%22%5BAuthor%5D)**,** [**Jones RA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jones%20RA%22%5BAuthor%5D)**,** [**Adey WR**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Adey%20WR%22%5BAuthor%5D)**,** [**Zimmerman G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zimmerman%20G%22%5BAuthor%5D)**. Extremely-low-frequency magnetic fields disrupt rhythmic slow activity in rat hippocampal slices.** [**Bioelectromagnetics.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Bioelectromagnetics.');) **17(5):388-395, 1996.**

Several studies have indicated that weak, extremely-low-frequency (ELF; 1-100 Hz) magnetic fields affect brain electrical activity and memory processes in man and laboratory animals. Our studies sought to determine whether ELF magnetic fields could couple directly with brain tissue and affect neuronal activity in vitro. We used rat hippocampal slices to study field effects on a specific brain activity known as rhythmic slow activity (RSA), or theta rhythm, which occurs in 7-15 s bursts in the hippocampus during memory functions. RSA, which, in vivo, is a cholinergic activity, is induced in hippocampal slices by perfusion of the tissue with carbachol, a stable analog of acetylcholine. We previously demonstrated that the free radical nitric oxide (NO), synthesized in carbachol-treated hippocampal slices, lengthened and destabilized the intervals between successive RSA episodes. Here, we investigate the possibility that sinusoidal ELF magnetic fields could trigger the NO-dependent perturbation of the rate of occurrence of the RSA episodes. Carbachol-treated slices were exposed for 10 min epochs to 1 or 60 Hz magnetic fields with field intensities of 5.6, 56, or 560 microT (rms), or they were sham exposed. All exposures took place in the presence of an ambient DC field of 45 microT, with an angle of -66 degrees from the horizontal plane. Sinusoidal 1 Hz fields at 56 and 560 microT, but not at 5.6 microT, triggered the irreversible destabilization of RSA intervals. Fields at 60 Hz resulted in similar, but not statistically significant, trends. Fields had no effects on RSA when NO synthesis was pharmacologically inhibited. However, field effects could take place when extracellular NO, diffusing from its cell of origin to the extracellular space,was chelated by hemoglobin. These results suggest that ELF magnetic fields exert a strong influence on NO systems in the brain; therefore, they could modulate the functional state of a variety of neuronal ensembles.

**(E)** **(VO, CE, IOD)** [**Bediz CS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bediz%20CS%22%5BAuthor%5D)**,** [**Baltaci AK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Baltaci%20AK%22%5BAuthor%5D)**,** [**Mogulkoc R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mogulkoc%20R%22%5BAuthor%5D)**,** [**Oztekin E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Oztekin%20E%22%5BAuthor%5D)**. Zinc supplementation ameliorates electromagnetic field-induced lipid peroxidation in the rat brain.** [**Tohoku J Exp Med.**](javascript:AL_get(this,%20'jour',%20'Tohoku%20J%20%0d%0aExp%20Med.');) **208(2):133-140, 2006.**

Extremely low-frequency (0-300 Hz) electromagnetic fields (EMFs) generated by power lines, wiring and home appliances are ubiquitous in our environment. All populations are now exposed to EMF, and exposure to EMF may pose health risks. Some of the adverse health effects of EMF exposure are lipid peroxidation and cell damage in various tissues. This study has investigated the effects of EMF exposure and zinc administration on lipid peroxidation in the rat brain. Twenty-four male Sprague-Dawley rats were randomly allocated to three groups; they were maintained untreated for 6 months (control, n = 8), exposed to low-frequency (50 Hz) EMF for 5 minutes every other day for 6 months (n = 8), or exposed to EMF and received zinc sulfate daily (3 mg/kg/day) intraperitoneally (n = 8). We measured plasma levels of zinc and thiobarbituric acid reactive substances (TBARS), and levels of reduced glutathione (GSH) in erythrocytes. TBARS and GSH levels were also determined in the brain tissues. TBARS levels in the plasma and brain tissues were higher in EMF-exposed rats with or without zinc supplementation, than those in controls (p < 0.001). In addition, TBARS levels were significantly lower in the zinc-supplemented rats than those in the EMF-exposed rats (p < 0.001). GSH levels were significantly decreased in the brain and erythrocytes of the EMF-exposed rats (p < 0.01), and were highest in the zinc-supplemented rats (p < 0.001). Plasma zinc was significantly lower in the EMF-exposed rats than those in controls (p < 0.001), while it was highest in the zinc-supplemented rats (p < 0.001). The present study suggests that long-term exposure to low-frequency EMF increases lipid peroxidation in the brain, which may be ameliorated by zinc supplementation.

**(E) (VT, AE, IFR, DFR, MC)** [**Belova NA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Belova%20NA%22%5BAuthor%5D)**,** [**Potselueva MM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Potselueva%20MM%22%5BAuthor%5D)**,** [**Skrebnitskaia LK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Skrebnitskaia%20LK%22%5BAuthor%5D)**,** [**Znobishcheva AV**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Znobishcheva%20AV%22%5BAuthor%5D)**,** [**Lednev VV**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lednev%20VV%22%5BAuthor%5D)**. The influence of weak magnetic fields on the production of the reactive oxygen species in peritoneal neutrophils in mice. Biophysics (**[**Biofizika).**](http://www.ncbi.nlm.nih.gov/pubmed/20968078##) **55(4):586-591, 2010.**

The influence of weak magnetic fields of different types on the rate of the formation of reactive oxygen species in mouse peritoneal neutrophils has been studied. It was found that the exposure of neutrophils activated by phorbol 12-myristate 13-acetate to the magnetic field tuned to the parametric resonance for Ca2+ ions leads to a decrease in the rate of the reactive oxygen species (ROS) generation by 23%. Conversely, the generation of ROS in neutrophils exposed to the same field but stimulated by the bacterial peptide FMLP (N-formyl-L-methionyl-L-leucyl-L-phenylalanine) increased by about 21%. Pulsed magnetic fields also changed the rate of ROS generation in phorbol-stimulated neutrophils by about 20%, but the sign of the effects observed in this case was opposite to those induced by the magnetic field tuned to the parametric resonance for Ca2+ ions.

**(E) (VT, AE, IOD, IX)** [**Benassi B**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Benassi%20B%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Filomeni G**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Filomeni%20G%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Montagna C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Montagna%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Merla C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Merla%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Lopresto V**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lopresto%20V%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Pinto R**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pinto%20R%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Marino C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Marino%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**,** [**Consales C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Consales%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26223801)**. Extremely low frequency magnetic field (ELF-MF) exposure sensitizes SH-SY5Y cells to the pro-Parkinson's Disease toxin MPP.** [**Mol Neurobiol.**](http://www.ncbi.nlm.nih.gov/pubmed/26223801) **53(6):4247-4260, 2016.**

Parkinson's disease (PD) is a neurodegenerative disorder characterized by dopaminergic neuron loss, with an etiopathogenesis involving both genetic and environmental factors. The occupational/residential exposure to the electromagnetic fields has been recently associated with an increased risk of neurodegenerative diseases; it has been thus proposed that the extremely low frequency magnetic field (ELF-MF) may contribute to neurodegenerative etiopathogenesis, as its interaction with biological systems directly impairs redox homeostasis in specific areas of the brain. The molecular mechanisms elicited by ELF-MF, and their potential involvement in PD onset, still remain unclear. To this end, we set up a generator of ELF-MF able to stably and homogeneously reproduce environmental prolonged exposure to ELF-MF (50 Hz, 1 mT). Results obtained indicate that ELF-MF exposure alters cell response of SH-SY5Y cells to MPP+. We demonstrate that ELF-MF does not affect per se survival, shape, and morphology of both proliferating and differentiated SH-SY5Y cells but significantly impairs redox homeostasis and thiol content, triggering an increase in protein carbonylation. As a result, toxicity of MPP+, even at low doses, is highly enhanced in ELF-MF-exposed cells due to a significant increase in ROS levels, potentiation of oxidative damage, and induction of a caspase-dependent apoptosis. Pre-incubation with the thiol antioxidants N-acetyl-L-cysteine and GSH ethyl-ester significantly reduces the extent of oxidative damage and protects cells from death induced by the combined treatment ELF-MF/MPP+. Taken overall, our results demonstrate the redox-based molecular interaction between ELF-MF and PD neurotoxins in vitro, and open a new scenario for defining the synergy of environmental factors in PD onset.

**(E)** **(VT, AE, IOD)** [**Buczyński A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Buczy%C5%84ski%20A%22%5BAuthor%5D)**,** [**Pacholski K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pacholski%20K%22%5BAuthor%5D)**,** [**Dziedziczak-Buczyńska M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dziedziczak-Buczy%C5%84ska%20M%22%5BAuthor%5D)**,** [**Henrykowska G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Henrykowska%20G%22%5BAuthor%5D)**,** [**Jerominko A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jerominko%20A%22%5BAuthor%5D)**. The assessment of oxygen metabolism selected parameters of blood platelets exposed to low frequency magnetic radiation in cars--in vitro studies.** [**Rocz Akad Med Bialymst.**](javascript:AL_get(this,%20'jour',%20'Rocz%20Akad%20%0d%0aMed%20Bialymst.');) **50 Suppl 1:23-25, 2005.**

PURPOSE: The aim of the study was to determine how free radicals generation in blood platelets exposed to electromagnetic field (EMF) occurring in cars affects the process of these morphotic elements cell membranes phospholipid peroxidation. MATERIAL AND METHODS: The suspension of human blood platelets was exposed to EMF of proper characteristics in a specially arranged research stand. After 30, 60 and 90 min exposure of the platelet specimen to EMF, free radicals generation was measured with chemiluminescence and malondialdehyde concentration according to Placer et al. method. The obtained results were compared with the control values. RESULTS: The increase of free radicals generation was observed after 30 and 90 min exposure of platelets to magnetic field. Malondialdehyde reached the highest values also after 30 and 90 min exposure of the platelets to EMF as compared to the control. CONCLUSIONS: The increase in oxygen reactive species generation under the effect of exogenic magnetic radiation as well as proportional intensification of the peroxidation process determined on the basis of malondialdehyde concentration (the marker of this phenomenon) point to the platelet sensitivity to the investigated environmental factor.

**(E) (VT, AE, DOD, IAO, IX)** [**Bułdak RJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bu%C5%82dak%20RJ%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Polaniak R**](http://www.ncbi.nlm.nih.gov/pubmed?term=Polaniak%20R%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Bułdak L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bu%C5%82dak%20L%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Zwirska-Korczala K**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zwirska-Korczala%20K%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Skonieczna M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Skonieczna%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Monsiol A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Monsiol%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Kukla M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kukla%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Duława-Bułdak A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Du%C5%82awa-Bu%C5%82dak%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**,** [**Birkner E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Birkner%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22535669)**. Short-term exposure to 50 Hz ELF-EMF alters the cisplatin-induced oxidative response in AT478 murine squamous cell carcinoma cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/22535669) **33(8):641-651, 2012.**

The aim of this study was to assess the influence of cisplatin and an extremely low frequency electromagnetic field (ELF-EMF) on antioxidant enzyme activity and the lipid peroxidation ratio, as well as the level of DNA damage and reactive oxygen species (ROS) production in AT478 carcinoma cells. Cells were cultured for 24 and 72 h in culture medium with cisplatin. Additionally, the cells were irradiated with 50 Hz/1 mT ELF-EMF for 16 min using a solenoid as a source of the ELF-EMF. The amount of ROS, superoxide dismutase (SOD) isoenzyme activity, glutathione peroxidase (GSH-Px) activity, DNA damage, and malondialdehyde (MDA) levels were assessed. Cells that were exposed to cisplatin exhibited a significant increase in ROS and antioxidant enzyme activity. The addition of ELF-EMF exposure to cisplatin treatment resulted in decreased ROS levels and antioxidant enzyme activity. A significant reduction in MDA concentrations was observed in all of the study groups, with the greatest decrease associated with treatment by both cisplatin and ELF-EMF. Cisplatin induced the most severe DNA damage; however, when cells were also irradiated with ELF-EMF, less DNA damage occurred. Exposure to ELF-EMF alone resulted in an increase in DNA damage compared to control cells. ELF-EMF lessened the effects of oxidative stress and DNA damage that were induced by cisplatin; however, ELF-EMF alone was a mild oxidative stressor and DNA damage inducer. We speculate that ELF-EMF exerts differential effects depending on the exogenous conditions. This information may be of value for appraising the pathophysiologic consequences of exposure to ELF-EMF.

**(E)(VT, AE, IFR)** [**Calabrò E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Calabr%C3%B2%20E%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Condello S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Condello%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Currò M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Curr%C3%B2%20M%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Ferlazzo N**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ferlazzo%20N%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Caccamo D**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Caccamo%20D%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Magazù S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Magaz%C3%B9%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**,** [**Ientile R**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ientile%20R%5BAuthor%5D&cauthor=true&cauthor_uid=24217848)**. Effects of low intensity static magnetic field on FTIR spectra and ROS production in SH-SY5Y neuronal-like cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/24217848) **34(8):618-629, 2013.**

Biological effects of man-made electromagnetic fields (EMFs) have been studied so far by experimental approaches exposing animals and cell cultures to EMFs. However, the evidence for cell toxicity induced by static magnetic field (SMF) is still uncertain. We investigated the effects produced by the exposure of human SH-SY5Y neuronal-like cells to a uniform magnetic field at intensities of 2.2 mT, which is less than the recommended public exposure limits set by the International Commission on Non-Ionizing Radiation Protection (ICNIRP). A decrease of membrane mitochondrial potential up to 30% was measured after 24 h of exposure to SMF in SH-SY5Y cells, and this effect was associated with reactive oxygen species production increase. Fourier transform infrared spectroscopy (FTIR) analysis showed that exposure to a static magnetic intensity around 2.2 mT changed the secondary structure of cellular proteins and lipid components. The vibration bands relative to the methylene group increased significantly after 4 h of exposure, whereas further exposure up to 24 h produced evident shifts of amide I and II modes and a relative increase in β-sheet contents with respect to α-helix components. Our study demonstrated that a moderate SMF causes alteration in cell homeostasis, as indicated by FTIR spectroscopy observations of changes in protein structures that are part of cell response to magnetic field exposure.

**(E) (VT, AE, DFR)** [**Calota V**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Calota%20V%5BAuthor%5D&cauthor=true&cauthor_uid=16517219)**,** [**Dragoiu S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Dragoiu%20S%5BAuthor%5D&cauthor=true&cauthor_uid=16517219)**,** [**Meghea A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Meghea%20A%5BAuthor%5D&cauthor=true&cauthor_uid=16517219)**,** [**Giurginca M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Giurginca%20M%5BAuthor%5D&cauthor=true&cauthor_uid=16517219)**. Decrease of luminol chemiluminescence upon exposure of human blood serum to 50 Hz electric fields.** [**Bioelectrochemistry.**](http://www.ncbi.nlm.nih.gov/pubmed/16517219) **69(1):126-127, 2006.**

The chemiluminescence of luminol, after 1 and 2 h in vitro exposure of human serum to 50 Hz electric fields of different intensities, decreases as compared to the controls. This indicates a field-induced decrease in the concentration of the free radicals. The report is limited to the key kinetic and field data, inviting independent kinetic analysis of the data in terms of reaction moments or reaction susceptibilities for the various normal modes indicated by the data.

**(E)(VT, AE, IFR)** [**Calota V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Calota%20V%22%5BAuthor%5D)**,** [**Dragoiu S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dragoiu%20S%22%5BAuthor%5D)**,** [**Meghea A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Meghea%20A%22%5BAuthor%5D)**,** [**Giurginca M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Giurginca%20M%22%5BAuthor%5D)**. Effects of prooxidants on human serum exposed to 50 Hz magnetic fields.** [**Electromagn Biol Med.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Electromagn%20Biol%20Med.');)**26(2):135-140, 2007.**

The purpose of this article is to evaluate magnetic field effects (50 Hz, different magnetic intensities) on the chemiluminescence intensity of human serum. We find that 1 and 2 h of exposure increased the chemiluminescence emission. The addition to the serum of prooxidants FeCl(2) and H(2)O(2) in different concentrations increased the chemiluminescence intensity even more.

**(E)(VO, CE, DOD, IOD, DFR, IFR)** [**Canseven AG**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Canseven%20AG%22%5BAuthor%5D)**,** [**Coskun S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Coskun%20S%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**. Effects of various extremely low frequency magnetic fields on the free radical processes, natural antioxidant system and respiratory burst system activities in the heart and liver tissues.** [**Indian J Biochem Biophys.**](javascript:AL_get(this,%20'jour',%20'Indian%20J%20%0d%0aBiochem%20Biophys.');) **45(5):326-331, 2008.**

Magnetic fields (MFs) can affect biological systems by increasing the release of free radicals that are able to alter cell defense systems and breakdown tissue homeostasis. In the present study, the effects of extremely low frequency (ELF) electromagnetic fields (EMF) were investigated on free radical levels, natural antioxidant systems and respiratory burst system activities in heart and liver tissues of guinea pigs exposed to 50 Hz MFs of 1, 2 and 3 mT for 4 h/day and 8 h/day for 5 days by measuring malondialdehyde (MDA), nitric oxide (NO), glutathione (GSH) levels and myeloperoxidase (MPO) activity. A total of sixty-two male guinea pigs, 10-12 weeks old were studied in seven groups as control and exposure groups: Group I (control), II (1 mT, 4 h/day), III (1 mT, 8 h/day), IV (2 mT, 4 h/day), V (2 mT, 8 h/day), VI (3 mT, 4 h/day), and VII (3 mT, 8 h/day). Controls were kept under the same conditions without any exposure to MF. MDA levels increased in liver in groups II and IV, but decreased in group VII for both liver and heart tissues. NOx levels declined in heart in groups II and III and in liver in groups III, V, and VI, but increased in liver in group VII. GSH levels increased in heart in groups II, IV, V, and in liver in groups V and VI and VI, but decreased in groups II and IV in liver. MPO activity decreased in liver in groups III, IV, VI and VII with respect to controls and in heart tissues in groups II, III and IV; however, there was a significant increase MPO activity in heart in group VII. From the results, it can be concluded that the intensity and exposure duration of MFs are among the effective conditions on the formation of free radicals and behaviour of antioxidant enzymes.

**(E) (VT, AE, IFR)** [**Chen Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=25450462)**,** [**Hong L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hong%20L%5BAuthor%5D&cauthor=true&cauthor_uid=25450462)**,** [**Zeng Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zeng%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=25450462)**,** [**Shen Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Shen%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=25450462)**,** [**Zeng Q**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zeng%20Q%5BAuthor%5D&cauthor=true&cauthor_uid=25450462)**. Power frequency magnetic fields induced reactive oxygen species-related autophagy in mouse embryonic fibroblasts.** [**Int J Biochem Cell Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/25450462) **57:108-114, 2014.**

Power frequency magnetic fields (PFMF) have been reported to affect several cellular functions, such as cell proliferation and apoptosis. In this study, we investigated the effects of PFMF on mouse embryonic fibroblasts (MEF) autophagy. After cells were exposed to 50 Hz PFMF at 2 mT for 0.5 h, 2 h, 6 h, 12 h, and 24 h, we observed a significant increase in autophagic markers at 6 h, including (i) higher microtubule-associated protein 1 light chain 3-II (LC3-II), (ii) the increased formation of GFP-LC3 puncta, and (iii) increased numbers of autophagic vacuoles under transmission electron microscope. Moreover, we provide convincing evidence using chloroquine (CQ) that the increase of autophagic markers was the result of enhanced autophagic flux and not the suppression of lysosomal function. In a search for molecular mechanisms underlying PFMF-mediated autophagy, we observe that the autophagic process involved reactive oxygen species (ROS) and was independent of the mammalian target of rapamycin (mTOR) signaling pathway.

**(E)(VT, AE, IX)** [**Cheun BS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Cheun%20BS%22%5BAuthor%5D)**,** [**Yi SH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yi%20SH%22%5BAuthor%5D)**,** [**Baik KY**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Baik%20KY%22%5BAuthor%5D)**,** [**Lim JK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lim%20JK%22%5BAuthor%5D)**,** [**Yoo JS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yoo%20JS%22%5BAuthor%5D)**,** [**Shin HW**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Shin%20HW%22%5BAuthor%5D)**,** [**Soh KS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22soh%20KS%22%5BAuthor%5D)**. Biophoton emission of MDCK cell with hydrogen peroxide and 60 Hz AC magnetic field.** [**J Environ Biol.**](javascript:AL_get(this,%20'jour',%20'J%20Environ%20%0d%0aBiol.');) **28(4):735-740, 2007.**

We studied biophoton characteristics of Madin-Darby canine kidney (MDCK) cells under the influence of H2O2 by employing a photomultiplier tube (PMT) and a fluorescence microscope. H2O2 was used for producing reactive oxygen species (ROS) in the measurement. Images from a fluorescence microscope show an increase of photon intensity emitted from the sample due to H2O2. By using a PMT we measured quantitative change in biophoton emission with application of H2O2 to the MDCK cell culture, found that the increase of the biophoton is dependent upon the amount of H2O2. The agreement between the results of the PMT and the fluorescence microscope suggests the possibility of quantitative measurement of the influence of ROS on living tissue or cell. In addition we applied a 60 Hz AC magnetic field on the cells to investigate the change in reaction between MDCK cell and ROS. It showed that a decay of chemiluminescence intensity has taken a different path following exposure to the magnetic field. As a result, the PMT measurement might be considered as a useful tool for studying biochemical characteristics in relation to ROS.

**(E)** **(VT, AE, IFR, IOD, IAO**) [**Chu LY**](http://www.ncbi.nlm.nih.gov/pubmed?term=Chu%20LY%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Lee JH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20JH%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Nam YS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Nam%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Lee YJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20YJ%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Park WH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Park%20WH%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Lee BC**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20BC%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Kim D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kim%20D%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Chung YH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Chung%20YH%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**,** [**Jeong JH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jeong%20JH%5BAuthor%5D&cauthor=true&cauthor_uid=22131325)**. Extremely low frequency magnetic field induces oxidative stress in mouse cerebellum.** [**Gen Physiol Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/22131325) **30(4):415-421, 2011.**

We have investigated whether extremely low frequency magnetic field (ELF-MF) induces lipid peroxidation and reactive oxygen species in mouse cerebellum. After exposure to 60 Hz ELF-MF at 2.3 mT intensity for 3 hours, there was a significant increase in malondialdehyde level and hydroxyl radical. ELF-MF significantly induced concomitant increase in superoxide dismutase without alteration in glutathione peroxidase activity. While glutathione contents were not altered, ascorbic acid levels were significantly decreased by ELF-MF exposure. These results indicate that ELF-MF may induce oxidative stress in mouse cerebellum. However, the mechanism remains further to be characterized.

**(E)** **(VO, AE, IFR)** [**Chung YH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chung%20YH%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Lee YJ**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20YJ%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Lee HS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20HS%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Chung SJ**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chung%20SJ%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Lim CH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lim%20CH%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Oh KW**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Oh%20KW%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Sohn UD**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sohn%20UD%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Park ES**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Park%20ES%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**,** [**Jeong JH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jeong%20JH%5BAuthor%5D&cauthor=true&cauthor_uid=25605992)**. Extremely low frequency magnetic field modulates the level of neurotransmitters.** [**Korean J Physiol Pharmacol.**](http://www.ncbi.nlm.nih.gov/pubmed/25605992) **19(1):15-20, 2015.**

This study was aimed to observe that extremely low frequency magnetic field (ELF-MF) may be relevant to changes of major neurotransmitters in rat brain. After the exposure to ELF-MF (60 Hz, 2.0 mT) for 2 or 5 days, we measured the levels of biogenic amines and their metabolites, amino acid neurotransmitters and nitric oxide (NO) in the cortex, striatum, thalamus, cerebellum and hippocampus. The exposure of ELF-MF for 2 or 5 days produced significant differences in norepinephrine and vanillyl mandelic acid in the striatum, thalamus, cerebellum and hippocampus. Significant increases in the levels of serotonin and 5-hydroxyindoleacetic acid were also observed in the striatum, thalamus or hippocampus. ELF-MF significantly increased the concentration of dopamine in the thalamus. ELF-MF tended to increase the levels of amino acid neurotransmitters such as glutamine, glycine and γ -aminobutyric acid in the striatum and thalamus, whereas it decreased the levels in the cortex, cerebellum and hippocampus. ELF-MF significantly increased NO concentration in the striatum, thalamus and hippocampus. The present study has demonstrated that exposure to ELF-MFs may evoke the changes in the levels of biogenic amines, amino acid and NO in the brain although the extent and property vary with the brain areas. However, the mechanisms remain further to be characterized.

**(E) (VO, HU, CE, IAO)** [**Cichoń N**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cicho%C5%84%20N%5BAuthor%5D&cauthor=true&cauthor_uid=28430370)**,** [**Bijak M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Bijak%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28430370)**,** [**Miller E**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Miller%20E%5BAuthor%5D&cauthor=true&cauthor_uid=28430370)**,** [**Saluk J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Saluk%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28430370)**. Extremely low frequency electromagnetic field (ELF-EMF) reduces oxidative stress and improves functional and psychological status in ischemic stroke patients.** [**Bioelectromagnetics.**](https://www.ncbi.nlm.nih.gov/pubmed/28430370) **2017 Apr 21. doi: 10.1002/bem.22055. [Epub ahead of print]**

As a result of ischaemia/reperfusion, massive generation of reactive oxygen species occurs, followed by decreased activity of antioxidant enzymes. Extremely low frequency electromagnetic fields (ELF-EMF) can modulate oxidative stress, but there are no clinical antioxidant studies in brain stroke patients. The aim of our study was to investigate the effect of ELF-EMF on clinical and antioxidant status in post-stroke patients. Fifty-seven patients were divided into two groups: ELF-EMF and non-ELF-EMF. Both groups underwent the same 4-week rehabilitation program. Additionally, the ELF-EMF group was exposed to an ELF-EMF field of 40 Hz, 7 mT for 15 min/day for 4 weeks (5 days a week). The activity of catalase and superoxide dismutase was measured in hemolysates, and total antioxidant status (TAS) determined in plasma. Functional status was assessed before and after the series of treatments using Activities of Daily Living (ADL), Mini-Mental State Examination (MMSE), and Geriatric Depression Scale (GDS). Applied ELF-EMF significantly increased enzymatic antioxidant activity; however, TAS levels did not change in either group. Results show that ELF-EMF induced a significant improvement in functional (ADL) and mental (MMSE, GDS) status. Clinical parameters had positive correlation with the level of enzymatic antioxidant protection

**(E)** **(VO, CE, IAO, DAO)** [**Ciejka EB**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ciejka%20EB%22%5BAuthor%5D)**,** [**Goraca A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Goraca%20A%22%5BAuthor%5D)**. The influence of low-frequency magnetic field on plasma antioxidant capacity and heart rate.** [**Wiad Lek.**](javascript:AL_get(this,%20'jour',%20'Wiad%20Lek.');) **62(2):81-86, 2009.**

INTRODUCTION: Low-frequency magnetic field is widely applied as magnetotherapy in physiotherapeutic treatment. Recognition of positive and negative effects of the magnetic field has been the subject of numerous studies. Experimental studies concern, among others, the effect of this field on the heart rate and plasma antioxidant capacity. The aim of the study was to check whether a time-variable magnetic field of constant frequency and induction affects the heart rate and plasma antioxidant capacity. MATERIAL AND METHODS: The tests were performed on Spraque-Dawley rats exposed to the magnetic field of the following parameters: frequency - 40 Hz, induction - 7 mT, time of exposure - 30 and 60 minutes. The measurements of ECG and plasma antioxidant capacity expressed in the number of reduced iron ions were performed on experimental animals: before, after a single exposure and after 14 days of exposure. RESULTS: A significant decrease of the heart rate was observed after 14 days of exposure. A variable magnetic field of the parameters: frequency - 40 Hz, induction - 7 mT and exposure time of 14 days caused an increase of the organism antioxidant defence, whereas a variable magnetic field of the frequency of 40 Hz, induction - 7 mT and exposure time 60 minutes for 14 days caused a significant decrease of the organism antioxidant defence. CONCLUSIONS: The exposure time affects heart rate, plasma antioxidant capacity and the organism defense ability against free radicals.

**(E)** **(VO, CE, IFR)** [**Ciejka E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ciejka%20E%22%5BAuthor%5D)**,** [**Skibska B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Skibska%20B%22%5BAuthor%5D)**,** [**Kleniewska P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kleniewska%20P%22%5BAuthor%5D)**,** [**Goraca A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Goraca%20A%22%5BAuthor%5D)**. [Influence of low frequency magnetic field on chosen parameters of oxidative stress in rat's muscles].** [**Pol Merkur Lekarski.**](http://www.ncbi.nlm.nih.gov/pubmed/21298985##) **29(174):361-364, 2010. [Article in Polish]**

Free radicals are atoms, molecules or their fragments, which excess leads to the development of the oxidative stress, which is caused of many neoplasmic, neurodegenerative, inflammatory diseases and aging the organism. The main of exogenous sources of free radicals are among others: industrial pollution, tobacco smoke, ionizing radiation, ultrasound and magnetic field. The low magnetic field is applied in the physician therapy. The aim of this study was to evaluate the influence of low magnetic field on the parameters of oxidative stress in rat's muscles. **MATERIALS AND METHODS:**  Thirty male rats, weight of 280-300 g were randomly divided into three experimental groups: control I and treatment II and III (ELFMF-exposed), each containing seven animals. Animals in treat group II were exposed to 40 Hz, 7 mT for 0.5 h/day for 14 days (this kind of the ELFMF is mostly use in magnetotherapy) while, group III was exposed to 40 Hz, 7 mT for 1 h/day for 14 days. Control rats were in separate room without exposing to ELFMF. Immediately after the last exposure, the part of muscles was taken under pentobarbital anaesthesia. The effects of exposure to ELFMF on oxidative states were assessed on the measurements of concentration of -SH group, H2O2, and the concentration of proteins in muscles homogenates. **RESULTS:** Exposure to ELFMF: 40 Hz, 7 mT, 30 and 60 min/day used for 2 weeks caused significant increase in -SH group concentration and decrease of the protein concentration in the muscles homogenates. **CONCLUSION:**  Low magnetic field used in magnetotherapy causes the significant changes of the generating the reactive forms of oxygen in the muscles which depend on the parameters of low magnetic field.

**(E)** **(VO, CE, IOD)** [**Ciejka E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ciejka%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22314568)**,** [**Kleniewska P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kleniewska%20P%5BAuthor%5D&cauthor=true&cauthor_uid=22314568)**,** [**Skibska B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Skibska%20B%5BAuthor%5D&cauthor=true&cauthor_uid=22314568)**,** [**Goraca A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Goraca%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22314568)**. Effects of extremely low frequency magnetic field on oxidative balance in brain of rats.** [**J Physiol Pharmacol.**](http://www.ncbi.nlm.nih.gov/pubmed/22314568) **62(6):657-661, 2011.**

Extremely low frequency magnetic field (ELF-MF) may result in oxidative DNA damage and lipid peroxidation with an ultimate effect on a number of systemic disturbances and cell death. The aim of the study is to assess the effect of ELF-MF parameters most frequently used in magnetotherapy on reactive oxygen species generation (ROS) in brain tissue of experimental animals depending on the time of exposure to this field. The research material included adult male Sprague-Dawley rats, aged 3-4 months. The animals were divided into 3 groups: I - control (shame) group; II - exposed to the following parameters of the magnetic field: 7 mT, 40 Hz, 30 min/day, 10 days; III - exposed to the ELF-MF parameters of 7 mT, 40 Hz, 60 min/day, 10 days. The selected parameters of oxidative stress: thiobarbituric acid reactive substances (TBARS), hydrogen peroxide (H(2)O(2)), total free sulphydryl groups (-SH groups) and protein in brain homogenates were measured after the exposure of rats to the magnetic field. ELF-MF parameters of 7 mT, 40 Hz, 30 min/day for 10 days caused a significant increase in lipid peroxidation and insignificant increase in H(2)O(2) and free -SH groups. The same ELF-MF parameters but applied for 60 min/day caused a significant increase in free -SH groups and protein concentration in the brain homogenates indicating the adaptive mechanism. The study has shown that ELF-MF applied for 30 min/day for 10 days can affect free radical generation in the brain. Prolongation of the exposure to ELF-MF (60/min/day) caused adaptation to this field. The effect of ELF-MF irradiation on oxidative stress parameters depends on the time of animal exposure to magnetic field.

**(E) (VO, CE, IAO)** [**Ciejka E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ciejka%20E%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**,** [**Jakubowska E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jakubowska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**,** [**Zelechowska P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zelechowska%20P%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**,** [**Huk-Kolega H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Huk-Kolega%20H%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**,** [**Kowalczyk A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kowalczyk%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**,** [**Goraca A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Goraca%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25230563)**. [Effect of extremely low frequency magnetic field on glutathione in rat muscles].** [**Med Pr.**](http://www.ncbi.nlm.nih.gov/pubmed/25230563) **65(3):343-349, 2014. [Article in Polish]**

BACKGROUND: Free radicals (FR) are atoms, molecules or their fragments. Their excess leads to the development of oxidizing stress, the cause of many neoplastic, neurodegenerative and inflammatory diseases, and aging of the organism. Industrial pollution, tobacco smoke, ionizing radiation, ultrasound and magnetic field are the major FR exogenous sources. The low frequency magnetic field is still more commonly applied in the physical therapy. The aim of the presented study was to evaluate the effect of extremely low frequency magnetic field used in the magnetotherapy on the level of total glutathione, oxidized and reduced, and the redox state of the skeletal muscle cells, depending on the duration of exposure to magnetic field. MATERIAL AND METHODS: The male rats, weight of 280-300 g, were randomly devided into 3 experimental groups: controls (group I) and treatment groups exposed to extremely low frequency magnetic field (ELF-MF) (group II exposed to 40 Hz, 7 mT for 0.5 h/day for 14 days and group III exposed to 40 Hz, 7 mT for 1 h/day for 14 days). Control rats were kept in a separate room not exposed to extremely low frequency magnetic field. Immediately after the last exposure, part of muscles was taken under pentobarbital anesthesia. Total glutathione, oxidized and reduced, and the redox state in the muscle tissue of animals were determined after exposure to magnetic fields. RESULTS: Exposure to low magnetic field: 40 Hz, 7 mT for 30 min/day and 60 min/day for 2 weeks significantly increased the total glutathione levels in the skeletal muscle compared to the control group (p < 0.001). CONCLUSIONS: Exposure to magnetic fields used in the magnetic therapy plays an important role in the development of adaptive mechanisms responsible for maintaining the oxidation-reduction balance in the body and depends on exposure duration.

**(VT, AE, IFR, IOD, DAO)** [**Cinzia C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Cinzia%20C%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Umberto M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Umberto%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Roberta B**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Roberta%20B%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Rita DA**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rita%20DA%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Maddalena M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Maddalena%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Luigi C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Luigi%20C%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Piero S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Piero%20S%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Vilberto S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Vilberto%20S%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**,** [**Lucia P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lucia%20P%5BAuthor%5D&cauthor=true&cauthor_uid=27001710)**. Effect of extremely low frequency electromagnetic fields on antioxidant activity in the human keratinocyte cell line NCTC 2544.** [**Biotechnol Appl Biochem.**](http://www.ncbi.nlm.nih.gov/pubmed/27001710) **2016 Mar 22. doi: 10.1002/bab.1495. [Epub ahead of print]**

Some epidemiological studies have suggested possible associations between exposure to extremely low frequency electromagnetic fields (ELF-EMFs) and various diseases. Recently ELF-EMF has been considered as a therapeutic agent. In order to support ELF-EMF use in regenerative medicine, in particular in the treatment of skin injuries, we investigated whether significant cell damage occurs after ELF-EMF exposure. Reactive Oxygen Species production was evaluated in the human keratinocyte exposed for 1 h to 50 Hz ELF-EMF in a range of field strengths from 0.25 G to 2 G. Significant ROS increases resulted at 0.5 G and 1 G and under these flux densities ROS production, Glutathione content, antioxidant defense activity and lipid peroxidation markers were assessed for different lengths of time. Analyzed parameters of antioxidant defense and membrane integrity showed a different trend at two selected magnetic fluxes, with a greater sensitivity of the cells exposed to 0.5 G, especially after 1 h. All significant alterations observed in the first four hours of exposure reverted to controls 24 h after suggesting that under these conditions ELF-EMF induces a slight oxidative stress that does not overwhelm the metabolic capacity of the cells or have a cytotoxic effect.

**(E) (VT, AE, IFR)** [**Consales C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Consales%20C%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Cirotti C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cirotti%20C%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Filomeni G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Filomeni%20G%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Panatta M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Panatta%20M%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Butera A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Butera%20A%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Merla C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Merla%20C%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Lopresto V**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Lopresto%20V%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Pinto R**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Pinto%20R%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Marino C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Marino%20C%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**,** [**Benassi B**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Benassi%20B%5BAuthor%5D&cauthor=true&cauthor_uid=29039021)**. Fifty-Hertz Magnetic Field Affects the Epigenetic Modulation of the miR-34b/c in Neuronal Cells.** [**Mol Neurobiol.**](https://www.ncbi.nlm.nih.gov/pubmed/29039021) **2017 Oct 16. doi: 10.1007/s12035-017-0791-0. [Epub ahead of print]**

The exposure to extremely low-frequency magnetic fields (ELF-MFs) has been associated to increased risk of neurodegenerative diseases, although the underlying molecular mechanisms are still undefined. Since epigenetic modulation has been recently encountered among the key events leading to neuronal degeneration, we here aimed at assessing if the control of gene expression mediated by miRNAs, namely miRs-34, has any roles in driving neuronal cell response to 50-Hz (1 mT) magnetic field in vitro. We demonstrate that ELF-MFs drive an early reduction of the expression level of miR-34b and miR-34c in SH-SY5Y human neuroblastoma cells, as well as in mouse primary cortical neurons, by affecting the transcription of the common pri-miR-34. This modulation is not p53 dependent, but attributable to the hyper-methylation of the CpG island mapping within the miR-34b/c promoter. Incubation with N-acetyl-l-cysteine or glutathione ethyl-ester fails to restore miR-34b/c expression, suggesting that miRs-34 are not responsive to ELF-MF-induced oxidative stress. By contrast, we show that miRs-34 control reactive oxygen species production and affect mitochondrial oxidative stress triggered by ELF-MFs, likely by modulating mitochondria-related miR-34 targets identified by in silico analysis. We finally demonstrate that ELF-MFs alter the expression of the α-synuclein, which is specifically stimulated upon ELF-MFs exposure via both direct miR-34 targeting and oxidative stress. Altogether, our data highlight the potential of the ELF-MFs to tune redox homeostasis and epigenetic control of gene expression in vitro and shed light on the possible mechanism(s) producing detrimental effects and predisposing neurons to degeneration.

**(E)** **(VO, CE, IFR, IOD)** [**Coşkun S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Co%C5%9Fkun%20S%22%5BAuthor%5D)**,** [**Balabanli B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Balabanli%20B%22%5BAuthor%5D)**,** [**Canseven A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Canseven%20A%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**. Effects of continuous and intermittent magnetic fields on oxidative parameters in vivo.** [**Neurochem Res.**](javascript:AL_get(this,%20'jour',%20'Neurochem%20%0d%0aRes.');) **34(2):238-243, 2009.**

Continuous and intermittent 50 Hz, 1.5 mT magnetic field with the exposure period of 4 h/day for 4 days was used to investigate its possible effect on adult guinea pigs. Tissues and plasma specimens were assessed by biochemical parameters. Malondialdehyde (MDA), glutathione (GSH), nitric oxide (NO) levels and myeloperoxidase activity (MPO) were examined in plasma, liver and brain tissues. All parameters were determined by spectrophotometer. While intermittent magnetic field was effective on plasma lipid peroxidation, continuous magnetic field was found to be effective on plasma MPO activity and NO levels. Augmentation of lipid peroxidation was also observed in liver tissue both intermittent and continuous magnetic field exposures. These results indicate that both the intermittent and continuous magnetic field exposures affect various tissues in a distinct manner because of having different tissue antioxidant status and responses.

**(E)** **(VO,** **CE, IOD, DAO)** [**Cui Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cui%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Ge Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ge%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Rizak JD**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rizak%20JD%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Zhai C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zhai%20C%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Zhou Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zhou%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Gong S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Gong%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**,** [**Che Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Che%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=22570685)**. Deficits in water maze performance and oxidative stress in the hippocampus and striatum induced by extremely low frequency magnetic field exposure.** [**PLoS One.**](http://www.ncbi.nlm.nih.gov/pubmed/22570685) **7(5):e32196, 2012.**

The exposures to extremely low frequency magnetic field (ELF-MF) in our environment have dramatically increased. Epidemiological studies suggest that there is a possible association between ELF-MF exposure and increased risks of cardiovascular disease, cancers and neurodegenerative disorders. Animal studies show that ELF-MF exposure may interfere with the activity of brain cells, generate behavioral and cognitive disturbances, and produce deficits in attention, perception and spatial learning. Although, many research efforts have been focused on the interaction between ELF-MF exposure and the central nervous system, the mechanism of interaction is still unknown. In this study, we examined the effects of ELF-MF exposure on learning in mice using two water maze tasks and on some parameters indicative of oxidative stress in the hippocampus and striatum. We found that ELF-MF exposure (1 mT, 50 Hz) induced serious oxidative stress in the hippocampus and striatum and impaired hippocampal-dependent spatial learning and striatum-dependent habit learning. This study provides evidence for the association between the impairment of learning and the oxidative stress in hippocampus and striatum induced by ELF-MF exposure.

**(NE) (VT, AE)** [**de Groot MW**](http://www.ncbi.nlm.nih.gov/pubmed?term=de%20Groot%20MW%5BAuthor%5D&cauthor=true&cauthor_uid=25111744)**,** [**Kock MD**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kock%20MD%5BAuthor%5D&cauthor=true&cauthor_uid=25111744)**,** [**Westerink RH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Westerink%20RH%5BAuthor%5D&cauthor=true&cauthor_uid=25111744)**. Assessment of the neurotoxic potential of exposure to 50Hz extremely low frequency electromagnetic fields (ELF-EMF) in naïve and chemically-stressed PC12 cells.** [**Neurotoxicology.**](http://www.ncbi.nlm.nih.gov/pubmed/25111744) **44:358-364, 2014.**

Increasing exposure to extremely low frequency electromagnetic fields (ELF-EMF), generated by power lines and electric appliances, raises concern about potential adverse health effects of ELF-EMF. The central nervous system is expected to be particularly vulnerable to ELF-EMF as its function strongly depends on electrical excitability. We therefore investigated effects of acute (30min) and sub-chronic (48h) exposure to 50Hz ELF-EMF on naïve and chemically-stressed pheochromocytoma (PC12) cells. The latter have higher levels of iron and/or reactive oxygen species (ROS) and display increased vulnerability to environmental insults. Effects of ELF-EMF on Ca2+-homeostasis, ROS production and membrane integrity were assessed using Fura-2 single cell fluorescence microscopy, H2-DCFDA and CFDA assays, respectively. Our data demonstrate that acute exposure of naïve PC12 cells to 50 Hz ELF-EMF up to 1000 μT fails to affect basal or depolarization-evoked [Ca2+]i. Moreover, sub-chronic ELF-EMF exposure up to 1000μT has no consistent effects on Ca2+-homeostasis in naïve PC12 cells and does not affect ROS production and membrane integrity. Notably, in chemically-stressed PC12 cells both acute and sub-chronic ELF-EMF exposure also failed to exert consistent effects on Ca2+-homeostasis, ROS production and membrane integrity. Our combined findings thus indicate that exposure to 50Hz ELF-EMF up to 1000 μT, i.e. 10,000 times above background exposure, does not induce neurotoxic effects in vitro, neither in naïve nor in chemically-stressed PC12 cells. Though our data require confirmation, e.g. in developing neuronal cells in vitro or (developing) animals, it appears that the neurotoxic risk of ELF-EMF exposure is limited.

**(NE)** **(VT, AE, IX)** [**De Mattei M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22De%20Mattei%20M%22%5BAuthor%5D)**,** [**Pasello M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pasello%20M%22%5BAuthor%5D)**,** [**Pellati A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pellati%20A%22%5BAuthor%5D)**,** [**Stabellini G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Stabellini%20G%22%5BAuthor%5D)**,** [**Massari L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Massari%20L%22%5BAuthor%5D)**,** [**Gemmati D**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Gemmati%20D%22%5BAuthor%5D)**,** [**Caruso A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Caruso%20A%22%5BAuthor%5D)**. Effects of electromagnetic fields on proteoglycan metabolism of bovine articular cartilage explants.** [**Connect Tissue Res.**](javascript:AL_get(this,%20'jour',%20'Connect%20%0d%0aTissue%20Res.');) **44(3-4):154-159, 2003.**

Electromagnetic field (EMF) exposure has been proposed for the treatment of osteoarthritis. In this study, we investigated the effects of EMF (75 Hz, 2,3 mT) on proteoglycan (PG) metabolism of bovine articular cartilage explants cultured in vitro, both under basal conditions and in the presence of interleukin-1beta (IL-1beta) in the culture medium. Proteoglycan synthesis and the residual PG tissue content resulted significantly higher in EMF-exposed explants than in controls, whereas no effect was observed on PG release and nitric oxide (NO) production. IL-1beta induced both a reduction in PG synthesis and an increase in PG release, related to a strong stimulation of NO production, which resulted in a net loss of tissue PG content. In IL-1beta-treated explants, EMF increased PG synthesis, whereas in spite of a slight stimulation of NO production EMF did not modify PG release. This resulted in the residual PG tissue content being maintained at the control level. In both experimental conditions, the effects of EMF were associated with an increase in lactate production. The results of our study show that EMFs are able to promote anabolic activities and PG synthesis in bovine articular cartilage explants. This effect also is maintained in the presence of IL-1beta, thus counteracting the catabolic activity of the cytokine. Altogether, these data suggest that EMF exposure exerts a chondroprotective effect on articular cartilage in vitro.

**(E)(VT, AE, IFR, IAO)** [**De Nicola M**](http://www.ncbi.nlm.nih.gov/pubmed?term=De%20Nicola%20M%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Cordisco S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cordisco%20S%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Cerella C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cerella%20C%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Albertini MC**](http://www.ncbi.nlm.nih.gov/pubmed?term=Albertini%20MC%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**D'Alessio M**](http://www.ncbi.nlm.nih.gov/pubmed?term=D'Alessio%20M%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Accorsi A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Accorsi%20A%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Bergamaschi A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bergamaschi%20A%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Magrini A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Magrini%20A%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**,** [**Ghibelli L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghibelli%20L%5BAuthor%5D&cauthor=true&cauthor_uid=17384247)**. Magnetic fields protect from apoptosis via redox alteration.** [**Ann N Y Acad Sci.**](http://www.ncbi.nlm.nih.gov/pubmed/17384247) **1090:59-68, 2006.**

Magnetic fields (MFs) are receiving much attention in basic research due to their emerging ability to alter intracellular signaling. We show here that static MFs with intensity of 6 mT significantly alter the intracellular redox balance of U937 cells. A strong increase of reactive oxygen species (ROS) and a decrease of glutathione (GSH) intracellular levels were found after 2 h of MF exposure and maintained thereafter. We found that also other types of MFs, such as extremely-low-frequency (ELF) MFs affect intracellular GSH starting from a threshold at 0.09 mT. We previously reported that static MFs in the intensity range of 0.3-60 mT reduce apoptosis induced by damaging agents (Fanelli et al., 1998). Here, we show that ELF-MFs are also able to protect U937 from apoptosis. Interestingly, this ability is limited to the ELF intensities able to alter redox equilibrium, indicating a link between MF's antiapoptotic effect and the MF alteration of intracellular redox balance. This suggests that MF-produced redox alterations may be part of the signaling pathway leading to apoptosis antagonism. Thus, we tested whether MFs may still exert an antiapoptotic action in cells where the redox state was artificially altered in both directions, that is, by creating an oxidative (via GSH depletion with BSO) or a reducing (with DTT) cellular environment. In both instances, MFs fail to affect apoptosis. Thus, a correct intracellular redox state is required in order for MFs to exert their antiapoptotic effect.

**(E) (VT, VO, AE, IOD, DAO, IX)** [**Deng B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Deng%20B%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Xu H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Xu%20H%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Zhang J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zhang%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Wang J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Wang%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Han LC**](http://www.ncbi.nlm.nih.gov/pubmed?term=Han%20LC%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Li LY**](http://www.ncbi.nlm.nih.gov/pubmed?term=Li%20LY%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Wu GL**](http://www.ncbi.nlm.nih.gov/pubmed?term=Wu%20GL%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Hou YN**](http://www.ncbi.nlm.nih.gov/pubmed?term=Hou%20YN%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Guo GZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Guo%20GZ%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Wang Q**](http://www.ncbi.nlm.nih.gov/pubmed?term=Wang%20Q%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Sang HF**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sang%20HF%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**,** [**Xu LX**](http://www.ncbi.nlm.nih.gov/pubmed?term=Xu%20LX%5BAuthor%5D&cauthor=true&cauthor_uid=24614080)**. Neuroprotective effects of sevoflurane against electromagnetic pulse-induced brain injury through inhibition of neuronal oxidative stress and apoptosis.** [**PLoS One.**](http://www.ncbi.nlm.nih.gov/pubmed/24614080) **9(3):e91019, 2014.**

Electromagnetic pulse (EMP) causes central nervous system damage and neurobehavioral disorders, and sevoflurane protects the brain from ischemic injury. We investigated the effects of sevoflurane on EMP-induced brain injury. Rats were exposed to EMP and immediately treated with sevoflurane. The protective effects of sevoflurane were assessed by Nissl staining, Fluoro-Jade C staining and electron microscopy. The neurobehavioral effects were assessed using the open-field test and the Morris water maze. Finally, primary cerebral cortical neurons were exposed to EMP and incubated with different concentration of sevoflurane. The cellular viability, lactate dehydrogenase (LDH) release, superoxide dismutase (SOD) activity and malondialdehyde (MDA) level were assayed. TUNEL staining was performed, and the expression of apoptotic markers was determined. The cerebral cortexes of EMP-exposed rats presented neuronal abnormalities. Sevoflurane alleviated these effects, as well as the learning and memory deficits caused by EMP exposure. In vitro, cell viability was reduced and LDH release was increased after EMP exposure; treatment with sevoflurane ameliorated these effects. Additionally, sevoflurane increased SOD activity, decreased MDA levels and alleviated neuronal apoptosis by regulating the expression of cleaved caspase-3, Bax and Bcl-2. These findings demonstrate that Sevoflurane conferred neuroprotective effects against EMP radiation-induced brain damage by inhibiting neuronal oxidative stress and apoptosis.

**(E)** **(VO, CE, IOD, DAO)** [**Deng Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Deng%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Zhang Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zhang%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Jia S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jia%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Liu J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Liu%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Liu Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Liu%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Xu W**](http://www.ncbi.nlm.nih.gov/pubmed?term=Xu%20W%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**,** [**Liu L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Liu%20L%5BAuthor%5D&cauthor=true&cauthor_uid=24158621)**. Effects of aluminum and extremely low frequency electromagnetic radiation on oxidative stress and memory in brain of mice.** [**Biol Trace Elem Res.**](http://www.ncbi.nlm.nih.gov/pubmed/24158621) **156(1-3):243-252, 2013.**

This study was aimed to investigate the effect of aluminum and extremely low-frequency magnetic fields (ELF-MF) on oxidative stress and memory of SPF Kunming mice. Sixty male SPF Kunming mice were divided randomly into four groups: control group, ELF-MF group (2 mT, 4 h/day), load aluminum group (200 mg aluminum/kg, 0.1 ml/10 g), and ELF-MF + aluminum group (2 mT, 4 h/day, 200 mg aluminum/kg). After 8 weeks of treatment, the mice of three experiment groups (ELF-MF group, load aluminum group, and ELF-MF + aluminum group) exhibited firstly the learning memory impairment, appearing that the escaping latency to the platform was prolonged and percentage in the platform quadrant was reduced in the Morris water maze (MWM) task. Secondly are the pathologic abnormalities including neuronal cell loss and overexpression of phosphorylated tau protein in the hippocampus and cerebral cortex. On the other hand, the markers of oxidative stress were determined in mice brain and serum. The results showed a statistically significant decrease in superoxide dismutase activity and increase in the levels of malondialdehyde in the ELF-MF group (P < 0.05 or P < 0.01), load aluminum group (P < 0.01), and ELF-MF + aluminum group (P < 0.01). However, the treatment with ELF-MF + aluminum induced no more damage than ELF-MF and aluminum did, respectively. In conclusion, both aluminum and ELF-MF could impact on learning memory and pro-oxidative function in Kunming mice. However, there was no evidence of any association between ELF-MF exposure with aluminum loading.

**(E) (VT, AE,** [**DRF) Di S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Di%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Tian Z**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Tian%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Qian A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Qian%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Li J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Li%20J%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Wu J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Wu%20J%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Wang Z**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Wang%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Zhang D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20D%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Yin D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Yin%20D%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Brandi ML**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Brandi%20ML%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**,** [**Shang P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Shang%20P%5BAuthor%5D&cauthor=true&cauthor_uid=22642494)**. Large gradient high magnetic field affects FLG29.1 cells differentiation to form osteoclast-like cells.** [**Int J Radiat Biol.**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Di+S+and+Tian+Z+and+2012) **88(11):806-813, 2012.**

PURPOSE: We aimed to investigate the effects of different apparent gravities (μ g, 1 g and 2 g) produced by large gradient high magnetic field (LGHMF) on human preosteoclast FLG29.1 cells. MATERIALS AND METHODS: FLG29.1 cells were cultured in Roswell Park Memorial Institute (RPMI)-1640 medium. Cells were exposed to LGHMF for 72 h. On culture day 1, 2, 3, cell proliferation was detected by 3-(4,5)-dimethylthiahi-azo (-z-y1)-3,5-di-phenytetrazoliumromide (MTT) method. On day 3, cell apoptosis and necrosis were assayed by Hoechst and propidium iodide (PI) staining. After cells were exposed to LGHMF for 72 h with the induction of 12-o-tetradecanoylphorbol 13-acetate (TPA), Tartrate-Resistant Acid Phosphatase (TRAP) positive cells and nitric oxide (NO) release were detected by TRAP staining and Griess method, respectively. Intracellular TRAP activity was measured using nitrophenylphosphate (pNPP) as the substrate. RESULTS: MTT detection revealed that compared to control, FLG 29.1 cell proliferation in the μ g and 2 g groups were promoted. However, there is no obvious difference between the 1 g and control groups. Hoechst-PI staining showed that LGHMF promoted cell apoptosis and necrosis, especially in the 2 g group. Exposure to LGHMF inhibited the NO concentration of supernatant. Both the TRAP activity and the number of TRAP positive cells were higher in cells of μ g group than those in 2 g group. In the 1 g group, they were decreased significantly compared to control. CONCLUSIONS: These findings indicate that LGHMF could directly affect human preosteoclast FLG29.1 cells survival and differentiation. High magnetic flux inhibited osteoclasts formation and differentiation while reduced apparent gravity enhanced osteoclastogenesis.

**(NE)** **(VT, CE)** [**Di Loreto S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Di%20Loreto%20S%22%5BAuthor%5D)**,** [**Falone S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Falone%20S%22%5BAuthor%5D)**,** [**Caracciolo V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Caracciolo%20V%22%5BAuthor%5D)**,** [**Sebastiani P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sebastiani%20P%22%5BAuthor%5D)**,** [**D'Alessandro A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22D%27Alessandro%20A%22%5BAuthor%5D)**,** [**Mirabilio A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mirabilio%20A%22%5BAuthor%5D)**,** [**Zimmitti V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zimmitti%20V%22%5BAuthor%5D)**,** [**Amicarelli F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Amicarelli%20F%22%5BAuthor%5D)**. Fifty hertz extremely low-frequency magnetic field exposure elicits redox and trophic response in rat-cortical neurons.** [**J Cell Physiol.**](javascript:AL_get(this,%20'jour',%20'J%20Cell%20%0d%0aPhysiol.');) **219(2):334-343, 2009.**

Large research activity has raised around the mechanisms of interaction between extremely low-frequency magnetic fields (ELF-MFs) and biological systems. ELF-MFs may interfere with chemical reactions involving reactive oxygen species (ROS), thus facilitating oxidative damages in living cells. Cortical neurons are particularly susceptible to oxidative stressors and are also highly dependent on the specific factors and proteins governing neuronal development, activity and survival. The aim of the present work was to investigate the effects of exposures to two different 50 Hz sinusoidal ELF-MFs intensities (0.1 and 1 mT) in maturing rat cortical neurons' major anti-oxidative enzymatic and non-enzymatic cellular protection systems, membrane peroxidative damage, as well as growth factor, and cytokine expression pattern. Briefly, our results showed that ELF-MFs affected positively the cell viability and concomitantly reduced the levels of apoptotic death in rat neuronal primary cultures, with no significant effects on the main anti-oxidative defences. Interestingly, linear regression analysis suggested a positive correlation between reduced glutathione (GSH) and ROS levels in 1 mT MF-exposed cells. On this basis, our hypothesis is that GSH could play an important role in the antioxidant defence towards the ELF-MF-induced redox challenge. Moreover, the GSH-based cellular response was achieved together with a brain-derived neurotrophic factor over-expression as well as with the interleukin 1beta-dependent regulation of pro-survival signaling pathways after ELF-MF exposure.

**(E) (VT, AE, IX)** [**Ding GR**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ding%20GR%22%5BAuthor%5D)**,** [**Nakahara T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nakahara%20T%22%5BAuthor%5D)**,** [**Hirose H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hirose%20H%22%5BAuthor%5D)**,** [**Koyama S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Koyama%20S%22%5BAuthor%5D)**,** [**Takashima Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Takashima%20Y%22%5BAuthor%5D)**,** [**Miyakoshi J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Miyakoshi%20J%22%5BAuthor%5D)**. Extremely low frequency magnetic fields and the promotion of H2O2-induced cell death in HL-60 cells.** [**Int J Radiat Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aRadiat%20Biol.');) **80(4):317-324, 2004.**

PURPOSE: To test whether exposure to an extremely low frequency magnetic field (60 Hz, 5 mT) affects hydrogen peroxide (H2O2)-induced cell death in human leukaemia HL-60 cells. MATERIALS AND METHODS: Cells were treated with H2O2 with or without exposure to an extremely low frequency magnetic fields. Viable cells, apoptotic and necrotic cells were determined by annexin V flow cytometry assay. The levels of apoptosis-related proteins (caspase-3, caspase-7, Bcl-2 and Bax) and poly(ADP-ribose) polymerase were detected using Western blotting. RESULTS: Simultaneous treatment with exposure to the magnetic field and H2O2 (85 or 100 microM) for 24 h increased the number of apoptotic and necrotic cells significantly, and significantly decreased the number of viable cells compared with cells treated with H2O2 alone. The protein levels of Bax and Bcl-2 showed no differences between H2O2-treated cells and those treated with both H2O2 and an extremely low frequency magnetic field. Exposure to the magnetic field also had no effect on H2O2-induced caspase-3 activation. However, the protein levels of active caspase-7 in cells simultaneously exposed to an extremely low frequency magnetic field and H2O2 for 2 and 8 h was higher than that of H2O2 treatment alone. In addition, simultaneous exposure to an extremely low frequency magnetic field and H2O2 caused poly(ADP-ribose) polymerase cleavage and induced early inactivation at 2 h, while H2O2 treatment alone did not produce this effect until 4 h. CONCLUSIONS: The data suggest that although the magnetic field itself cannot induce apoptosis and necrosis, it exerts a promoting effect on H2O2-induced cell death, and it demonstrates that caspase-7 as well as poly(ADP-ribose) polymerase might be involved in this process.

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In recent years, extremely low-frequency electromagnetic field (ELF-EMF) has received considerable attention for its potential biological effects. Numerous studies have shown the role of ELF-EMF in behaviour modulation. The aim of this study was to investigate the effect of short-term ELF-EMF (50 Hz) in the development of anxiety-like behaviour in rats through change hypothalamic oxidative stress and NO. Ten adult male rats (Wistar albino) were divided in two groups: control group-without exposure to ELF-EMF and experimental group-exposed to ELF-EMF during 7 days. After the exposure, time open field test and elevated plus maze were used to evaluate the anxiety-like behaviour of rats. Upon completion of the behavioural tests, concentrations of superoxide anion (O2·-), nitrite (NO2-, as an indicator of NO) and peroxynitrite (ONOO-) were determined in the hypothalamus of the animals. Obtained results show that ELF-EMF both induces anxiety-like behaviour and increases concentrations of O2·- and NO, whereas it did not effect on ONOO- concentration in hypothalamus of rats. In conclusion, the development of anxiety-like behaviour is mediated by oxidative stress and increased NO concentration in hypothalamus of rats exposed to ELF-EMF during 7 days.

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The present study investigated the effects of lotus seedpod procyanidins (LSPCs) administered by oral gavage on the cognitive deficits and oxidative damage of mice at extremely low frequency electromagnetic field (ELF-EMF) exposure (50 Hz, 8 mT, 28 days). The results showed that 90 mg kg-1 LSPCs treatment significantly increased body weight compared with the ELF-EMF group at ELF-EMF exposure and effectively maintained liver index, thymus index, kidney index and spleen index close to normal. A water maze test indicated that learning and memory abilities of the ELF-EMF group deteriorated significantly with ELF-EMF exposure when compared with the control group, but the ELF-EMF + LSPCs90 group had remarkably improved learning and memory abilities compared with the ELF-EMF group. Malondialdehyde (MDA), reactive oxygen species (ROS), nitric oxide (NO) and nitric oxide synthase (NOS) mostly exhibited significant increases, while the activities of glutathione peroxidase (GPx), catalase (CAT) and superoxide dismutase (SOD) decreased significantly under ELF-EMF exposure in the ELF-EMF group. LSPCs (especially 60, 90 mg kg-1) administration decreased MDA, ROS, NO content and lowered NOS activity in LSPCs treatment groups. Furthermore, LSPCs (60, 90 mg kg-1) treatment significantly augmented GPx, CAT, SOD activity in the hippocampus and serum. Pathological observation showed that number of pyramidal cells of the CA1 and CA3 regions of the hippocampus of the LSPCs treatment groups was significantly greater than the ELF-EMF group. All the data suggested that the LSPCs can effectively prevent learning and memory damage and oxidative damage caused by the ELF-EMF, most likely through the ability of LSPCs to scavenge oxygen free radicals and to stimulate antioxidant enzyme activity.

**(NE)** **(VT, AE)** [**Duan W**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Duan%20W%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Liu C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Liu%20C%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Zhang L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20L%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**He M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=He%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Xu S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Xu%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Chen C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20C%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Pi H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pi%20H%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Gao P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Gao%20P%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Zhang Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Zhong M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zhong%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Yu Z**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Yu%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**,** [**Zhou Z**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zhou%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=25688995)**. Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells.** [**Radiat Res.**](http://www.ncbi.nlm.nih.gov/pubmed/25688995) **183(3):305-314, 2015.**

Extremely low-frequency electromagnetic fields (ELF-EMF) and radiofrequency electromagnetic fields (RF-EMF) have been considered to be possibly carcinogenic to humans. However, their genotoxic effects remain controversial. To make experiments controllable and results comparable, we standardized exposure conditions and explored the potential genotoxicity of 50 Hz ELF-EMF and 1800 MHz RF-EMF. A mouse spermatocyte-derived GC-2 cell line was intermittently (5 min on and 10 min off) exposed to 50 Hz ELF-EMF at an intensity of 1, 2 or 3 mT or to RF-EMF in GSM-Talk mode at the specific absorption rates (SAR) of 1, 2 or 4 W/kg. After exposure for 24 h, we found that neither ELF-EMF nor RF-EMF affected cell viability using Cell Counting Kit-8. Through the use of an alkaline comet assay and immunofluorescence against γ-H2AX foci, we found that ELF-EMF exposure resulted in a significant increase of DNA strand breaks at 3 mT, whereas RF-EMF exposure had insufficient energy to induce such effects. Using a formamidopyrimidine DNA glycosylase (FPG)-modified alkaline comet assay, we observed that RF-EMF exposure significantly induced oxidative DNA base damage at a SAR value of 4 W/kg, whereas ELF-EMF exposure did not. Our results suggest that both ELF-EMF and RF-EMF under the same experimental conditions may produce genotoxicity at relative high intensities, but they create different patterns of DNA damage. Therefore, the potential mechanisms underlying the genotoxicity of different frequency electromagnetic fields may be different.

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Purpose: The aim of this research was to demonstrate the protective effects of electromagnetic field (EMF) exposure on the human microglial cell line, HMO6, against ischemic cell death induced by in vitro oxygen-glucose deprivation (OGD). Materials and methods: HMO6 cells were cultured for 4 h under OGD with or without exposure to EMF with different combinations of frequencies and intensities (10, 50, or 100 Hz/1 mT and 50 Hz/0.01, 0.1, or 1 mT). Cell survival, intracellular calcium and reactive oxygen species (ROS) levels were measured. Results: OGD caused significant HMO6 cell death as well as elevation of intracellular Ca2+ and ROS levels. Among different combinations of EMF frequencies and intensities, 50 Hz/1 mT EMF was the most potent to attenuate OGD-induced cell death and intracellular Ca2+ and ROS levels. A significant but less potent protective effect was also found at 10 Hz/1 mT, whereas no protective effect was found at other combinations of EMF. A xanthine oxidase inhibitor reversed OGD-induced ROS production and cell death, while NADPH oxidase and mitochondrial respiration chain complex II inhibitors did not affect cell death. Conclusions: 50 Hz/1 mT EMF protects human microglial cells from OGD-induced cell death by interfering with OGD-induced elevation of intracellular Ca2+ and ROS levels, and xanthine oxidase is one of the main mediators involved in OGD-induced HMO6 cell death. Non-invasive treatment of EMF radiation may be clinically useful to attenuate hypoxic-ischemic brain injury.

**(E)** **(VO, CE, IOD, IAO)** [**Emre M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Emre%20M%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**,** [**Cetiner S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cetiner%20S%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**,** [**Zencir S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Zencir%20S%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**,** [**Unlukurt I**](http://www.ncbi.nlm.nih.gov/pubmed?term=Unlukurt%20I%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**,** [**Kahraman I**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kahraman%20I%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**,** [**Topcu Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Topcu%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=20824388)**. Oxidative stress and apoptosis in relation to exposure to magnetic field.** [**Cell Biochem Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/20824388) **59(2):71-77, 2011.**

We investigated the effect of extremely low-frequency electromagnetic field (ELF-EMF) with pulse trains exposure on lipid peroxidation, and, hence, oxidative stress in the rat liver tissue. The parameters that we measured were the levels of plasma alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase as well as plasma albumin, bilirubin, and total protein levels in 30 adult male Wistar rats exposed to ELF. We also determined the percentage of apoptotic and necrotic cells of the kidney extracts from the animals by flow cytometry method. Apoptotic cell death was further characterized by monitoring DNA degradation using gel electrophoresis. The results showed an increase in the levels of oxidative stress indicators, and the flow cytometric data suggested a possible relationship between the exposure to magnetic field and the cell death. We showed significantly lower necrotic cell percentages in experimental animals compared to either unexposed or sham control groups. However, DNA ladder analyses did not differentiate between the groups. Our results were discussed in relation to the response of biological systems to EMF.

**(E) (VO, CE, IFR)** [**Erdal N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Erdal%20N%22%5BAuthor%5D)**,** [**Gürgül S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22G%C3%BCrg%C3%BCl%20S%22%5BAuthor%5D)**,** [**Tamer L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tamer%20L%22%5BAuthor%5D)**,** [**Ayaz L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ayaz%20L%22%5BAuthor%5D)**. Effects of long-term exposure of extremely low frequency magnetic field on oxidative/nitrosative stress in rat liver.** [**J Radiat Res (Tokyo).**](javascript:AL_get(this,%20'jour',%20'J%20Radiat%20%0d%0aRes%20(Tokyo).');) **49(2):181-187, 2008.**

Thirty-two adult Wistar-Albino female and male rats were used to investigate the long-term (45 days) effects of extremely low frequency magnetic field (ELF-MF; 50Hz, 1mT, 4h/day) exposure on oxidative/nitrosative stress in liver tissues of rats. The rats were divided randomly into four groups: female control (FC; n = 8) and MF-exposed female rats (F-MF; n = 8); male control (MC; n = 8) and MF-exposed male rats (M-MF; n = 8). Liver tissue from each animal was harvested and utilized for malondialdehyde (MDA) and 3-nitrotyrosine (3-NT) detection. MDA levels were measured by MDA-TBA method, while the 3-NT levels were determined by the HPLC-UV system. There were no significant differences between the MDA levels of the control (FC; MC) and MF-exposed (F-MF; M-MF) rats (P > 0.05). In the F-MF rats, 3-NT levels were significantly increased when compared to those of the FC rats (P < 0.05). There were no significant differences between the 3-NT levels of the MC and M-MF rats. In conclusion, our study suggests that the long-term ELF-MF exposure may enhance the oxidative/nitrosative stress in liver tissue of the female rats and could have a deteriorative effect on cellular proteins rather than lipids by enhancing 3-NT formation.

**(E)** **(VO, CE, IAO, DAO)** [**Falone S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Falone%20S%22%5BAuthor%5D)**,** [**Mirabilio A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mirabilio%20A%22%5BAuthor%5D)**,** [**Carbone MC**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Carbone%20MC%22%5BAuthor%5D)**,** [**Zimmitti V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zimmitti%20V%22%5BAuthor%5D)**,** [**Di Loreto S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Di%20Loreto%20S%22%5BAuthor%5D)**,** [**Mariggiò MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mariggi%C3%B2%20MA%22%5BAuthor%5D)**,** [**Mancinelli R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mancinelli%20R%22%5BAuthor%5D)**,** [**Di Ilio C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Di%20Ilio%20C%22%5BAuthor%5D)**,** [**Amicarelli F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Amicarelli%20F%22%5BAuthor%5D)**. Chronic exposure to 50 Hz magnetic fields causes a significant weakening of antioxidant defence systems in aged rat brain.** [**Int J Biochem Cell Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aBiochem%20Cell%20Biol.');) **40(12):2762-2770, 2008.**

Several studies suggest that extremely low-frequency magnetic fields (ELF-MFs) may enhance the free radical endogenous production. It is also well known that one of the unavoidable consequences of ageing is an overall oxidative stress-based decline in several physiological functions and in the general resistance to stressors. On the basis of these assumptions, the aim of this study was to establish whether the ageing process can increase susceptibility towards widely present ELF-MF-mediated pro-oxidative challenges. To this end, female Sprague-Dawley rats were continuously exposed to a sinusoidal 50 Hz, 0.1 mT magnetic field for 10 days. Treatment-induced changes in the major antioxidant protection systems and in the neurotrophic support were investigated, as a function of the age of the subjects. All analyses were performed in brain cortices, due to the high susceptibility of neuronal cells to oxidative injury. Our results indicated that ELF-MF exposure significantly affects anti-oxidative capability, both in young and aged animals, although in opposite ways. Indeed, exposed young individuals enhanced their neurotrophic signalling and anti-oxidative enzymatic defence against a possible ELF-MF-mediated increase in oxygen radical species. In contrast, aged subjects were not capable of increasing their defences in response to ELF-MF treatment but, on the contrary, they underwent a significant decrease in the major antioxidant enzymatic activities. In conclusion, our data seem to suggest that the exposure to ELF-MFs may act as a risk factor for the occurrence of oxidative stress-based nervous system pathologies associated with ageing.

**(E) (VT, AE, IAO, IX)** [**Falone S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Falone%20S%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Marchesi N**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Marchesi%20N%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Osera C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Osera%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Fassina L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Fassina%20L%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Comincini S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Comincini%20S%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Amadio M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Amadio%20M%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**,** [**Pascale A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pascale%20A%5BAuthor%5D&cauthor=true&cauthor_uid=26940444)**. Pulsed electromagnetic field (PEMF) prevents pro-oxidant effects of H2O2 in SK-N-BE(2) human neuroblastoma cells.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/26940444) **2016 Mar 4:1-6. [Epub ahead of print]**

Purpose: The redox milieu, together with reactive oxygen species (ROS) accumulation, may play a role in mediating some biological effects of extremely-low-frequency electromagnetic fields (ELF-EMF). Some of us have recently reported that a pulsed EMF (PEMF) improves the antioxidant response of a drug-sensitive human neuroblastoma SH-SY5Y cell line to pro-oxidants. Since drug resistance may affect cell sensitivity to redox-based treatments, we wanted to verify whether drug-resistant human neuroblastoma SK-N-BE(2) cells respond to a PEMF in a similar fashion. Materials and methods: SK-N-BE(2) cells were exposed to repeated 2 mT, 75 Hz PEMF (15 min each, repeated 3 times over 5 days), and ROS production, Mn-dependent superoxide dismutase (MnSOD)-based antioxidant protection and viability were assessed after 10 min or 30 min 1 mM hydrogen peroxide. Sham controls were kept at the same time in identical cell culture incubators. Results: The PEMF increased the MnSOD-based antioxidant protection and reduced the ROS production in response to a pro-oxidant challenge. Conclusions: Our work might lay foundation for the development of non-invasive PEMF-based approaches aimed at elevating endogenous antioxidant properties in cellular or tissue models.

**(E) (VT, AE, IAO)** [**Falone S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Falone%20S%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Santini S Jr**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Santini%20S%20Jr%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Cordone V**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cordone%20V%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Cesare P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cesare%20P%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Bonfigli A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Bonfigli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Grannonico M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Grannonico%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Di Emidio G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Di%20Emidio%20G%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Tatone C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Tatone%20C%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Cacchio M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cacchio%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**,** [**Amicarelli F**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Amicarelli%20F%5BAuthor%5D&cauthor=true&cauthor_uid=28904402)**. Power frequency magnetic field promotes a more malignant phenotype in neuroblastoma cells via redox-related mechanisms.** [**Sci Rep.**](https://www.ncbi.nlm.nih.gov/pubmed/28904402) **7(1):11470, 2017.**

In accordance with the classification of the International Agency for Research on Cancer, extremely low frequency magnetic fields (ELF-MF) are suspected to promote malignant progression by providing survival advantage to cancer cells through the activation of critical cytoprotective pathways. Among these, the major antioxidative and detoxification defence systems might be targeted by ELF-MF by conferring cells significant resistance against clinically-relevant cytotoxic agents. We investigated whether the hyperproliferation that is induced in SH-SY5Y human neuroblastoma cells by a 50 Hz, 1 mT ELF magnetic field was supported by improved defence towards reactive oxygen species (ROS) and xenobiotics, as well as by reduced vulnerability against both H2O2 and anti-tumor ROS-generating drug doxorubicin. ELF-MF induced a proliferative and survival advantage by activating key redox-responsive antioxidative and detoxification cytoprotective pathways that are associated with a more aggressive behavior of neuroblastoma cells. This was coupled with the upregulation of the major sirtuins, as well as with increased signaling activity of the erythroid 2-related nuclear transcription factor 2 (NRF2). Interestingly, we also showed that the exposure to 50 Hz MF as low as 100 µT may still be able to alter behavior and responses of cancer cells to clinically-relevant drugs.

**(VT, AE, IFR, AO)** [**Feng B**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Feng%20B%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**,** [**Qiu L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Qiu%20L%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**,** [**Ye C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ye%20C%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**,** [**Chen L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20L%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**,** [**Fu Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Fu%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**,** [**Sun W**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20W%5BAuthor%5D&cauthor=true&cauthor_uid=26850078)**. Exposure to a 50-Hz magnetic field induced mitochondrial permeability transition through the ROS/GSK-3β signaling pathway.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/26850078) **92:148-155, 2016a.**

Purpose To investigate the biological effects of a 50-Hz magnetic field (MF) on mitochondrial permeability. Materials and methods Human amniotic epithelial cells were exposed to MF (50 Hz, 0.4 mT) for different durations. Mitochondrial permeability, mitochondrial membrane potential (ΔΨm), cytochrome c (Cyt-c) release and the related mechanisms were explored. Results Exposure to the MF at 0.4 mT for 60 min transiently induced mitochondrial permeability transition (MPT) and Cyt-c release, although there was no significant effect on mitochondrial membrane potential (ΔΨm). Other than decreasing the total Bcl-2 associated X protein (Bax) level, MF exposure did not significantly affect the levels of Bax and B-cell lymphoma-2 (Bcl-2) in mitochondria. In addition, cells exposed to the MF showed increased intracellular reactive oxidative species (ROS) levels and glycogen synthase kinase-3β (GSK-3β) dephosphorylation at 9 serine residue (Ser9). Moreover, the MF-induced MPT was attenuated by ROS scavenger (N-acetyl-L-cysteine, NAC) or GSK-3β inhibitor, and NAC pretreatment prevented GSK-3β dephosphorylation (Ser9) caused by MF exposure. Conclusion MPT induced by MF exposure was mediated through the ROS/GSK-3β signaling pathway.

**(E) (VT, AE, IFR)** [**Feng B**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Feng%20B%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**,** [**Dai A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Dai%20A%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**,** [**Chen L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20L%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**,** [**Qiu L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Qiu%20L%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**,** [**Fu Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Fu%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**,** [**Sun W**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20W%5BAuthor%5D&cauthor=true&cauthor_uid=27442448)**. NADPH oxidase-produced superoxide mediated a 50-Hz magnetic field-induced epidermal growth factor receptor clustering.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/27442448) **92:596-602, 2016b.**

PURPOSE: A 50-Hz magnetic field (MF) was found to induce epidermal growth factor receptor (EGFR) clustering in our previous study. The aim of this work was to investigate the molecular mechanisms that mediated MF-induced EGFR clustering. MATERIALS AND METHODS: Human amniotic epithelial (FL) cells were exposed to a 50-Hz MF. Total reactive oxygen species (ROS), cytoplasmic and mitochondrial superoxide production were detected by DCFH-DA, DHE and MitoSOX, respectively. EGFR clustering was analyzed using confocal microscopy after indirect immunofluorescence staining. RESULTS: Results showed that exposing FL cells to MF at intensity higher than 0.2 mT for 15 min enhanced total ROS production. Additionally, enhanced total ROS and cytoplasmic superoxide production were observed after exposing cells to MF at 0.4 mT for 5, 15, or 30 min, while mitochondrial superoxide production for 15 or 30 min. Pretreatment with Nox inhibitor, DPI, effectively inhibited MF-induced cytoplasmic superoxide production and subsequent EGFR clustering while mitochondrial superoxide production was not affected. CONCLUSIONS: Nox-produced superoxide mediated a 50-Hz magnetic field-induced EGFR clustering.

**(E) (VT, AE, IFR, MC)** [**Feng B**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Feng%20B%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**,** [**Ye C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ye%20C%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**,** [**Qiu L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Qiu%20L%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**,** [**Chen L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20L%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**,** [**Fu Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Fu%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**,** [**Sun W**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20W%5BAuthor%5D&cauthor=true&cauthor_uid=27310130)**. Mitochondrial ROS release and subsequent Akt Activation potentially mediated the anti-apoptotic effect of a 50-Hz magnetic field on FL cells.** [**Cell Physiol Biochem.**](https://www.ncbi.nlm.nih.gov/pubmed/27310130) **38(6):2489-2499, 2016c.**

BACKGROUND/AIMS: Our previous study showed that exposure to a 50-Hz magnetic field (MF) could induce transient mitochondrial permeability transition (MPT) in cells. In the present study, the aim was to explore the possible biological implications of MF-induced transient MPT. MATERIALS AND METHODS: Human amniotic (FL) cells were exposed to MF for different durations or intensities followed by incubation with staurosporine for 4 h. After MF exposure, cell early apoptosis, cell viability, mitochondrial ROS and the level of phosphorylated Akt were assessed. After MF exposure followed by incubation with staurosporine, cell early apoptosis was assessed. RESULTS: MF exposure had a protective effect against early apoptosis induced by staurosporine, which could be abolished by MPT inhibitors, although MF exposure alone had no significant effect on early apoptosis or viability of cells. In addition, exposing cells to MF increased the level of mitochondrial ROS which were released into cytoplasm through mitochondrial permeability transition pores (mPTP), and induced ROS-dependent phosphorylation of Akt. Furthermore, the anti-apoptotic effect of MF exposure was completely eliminated when Akt was inhibited. CONCLUSIONS: The present study indicated a possibility that mitochondrial ROS release through mPTP and subsequent Akt activation were necessary for the anti-apoptotic effect of MF.

**(E)** **(VO, CE, DAO)** [**Fernie KJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fernie%20KJ%22%5BAuthor%5D)**,** [**Bird DM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bird%20DM%22%5BAuthor%5D)**. Evidence of oxidative stress in American kestrels exposed to electromagnetic fields.** [**Environ Res.**](javascript:AL_get(this,%20'jour',%20'Environ%20%0d%0aRes.');) **86(2):198-207, 2001.**

Exposure to electromagnetic fields (EMFs) alters melatonin, behavior, growth, and reproduction of captive American kestrels (Falco sparverius), particularly of males. EMF exposure is a "possible" human carcinogen and associated with some neurodegenerative diseases. Oxidative stress contributes to cancer, neurodegenerative diseases, and immune disorders. We tested whether EMF exposure elicits an avian immune response and alters oxidative stress levels. Captive male kestrels were bred under control or EMF conditions equivalent to those experienced by wild kestrels. Short-term EMF exposure (one breeding season) suppressed plasma total proteins, hematocrits, and carotenoids in the first half of the breeding season. It also suppressed erythrocyte cells and lymphocyte proportions, but elevated granulosa proportions at the end of the breeding season. Long-term EMF exposure (two breeding seasons) suppressed hematocrits in the first half of the reproductive period too. Results indicate that only short-term EMF birds experience an immune response, particularly during the early half of the breeding season. The elevation of granulocytes, and the suppression of carotenoids, total proteins, and previously melatonin in the same kestrels, signifies that the short-term EMF male kestrels had higher levels of oxidative stress, due to an immune response and/or EMF exposure. Long-term EMF exposure may be linked to higher levels of oxidative stress through EMF exposure only.

**(E) (VT, AE, IX)** [**Fiorani M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fiorani%20M%22%5BAuthor%5D)**,** [**Biagiarelli B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Biagiarelli%20B%22%5BAuthor%5D)**,** [**Vetrano F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Vetrano%20F%22%5BAuthor%5D)**,** [**Guidi G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Guidi%20G%22%5BAuthor%5D)**,** [**Dachà M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dach%C3%A0%20M%22%5BAuthor%5D)**,** [**Stocchi V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Stocchi%20V%22%5BAuthor%5D)**. In vitro effects of 50 Hz magnetic fields on oxidatively damaged rabbit red blood cells.** [**Bioelectromagnetics.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Bioelectromagnetics.');) **18(2):125-131, 1997.**

The aim of this study was to investigate the effects of 50 Hz magnetic fields (0.2-0.5 mT) on rabbit red blood cells (RBCs) that were exposed simultaneously to the action of an oxygen radical-generating system, Fe(II)/ascorbate. Previous data obtained in our laboratory showed at the exposure of rabbit erythrocytes or reticulocytes to Fe(II)/ascorbate hexokinase inactivation, whereas the other glycolytic enzymes do not show any decay. We also observed depletion of reduced glutathione (GSH) content with a concomitant intracellular and extracellular increase in oxidized glutathione (GSSG) and a decrease in energy charge. In this work we investigated whether 50 Hz magnetic fields could influence the intracellular impairments that occur when erythrocytes or reticulocytes are exposed to this oxidant system, namely, inactivation of hexokinase activity, GSH depletion, a change in energy charge, and hemoglobin oxidation. The results obtained indicate the a 0.5 mT magnetic field had no effect on intact RBCs, whereas it increased the damage with Fe(II)/ascorbate to a 0.5 mT magnetic field induced a significant further decay in hexokinase activity (about 20%) as well as a twofold increase in methemoglobin production compared with RBCs that were exposed to the oxidant system alone. Although further studies will be needed to determine the physiological implications of these data, the results reported in this study demonstrate that the effects of the magnetic fields investigated are able to potentiate the cellular damage induced in vitro by oxidizing agents.

**(E)** **(VT, AE, IFR)** [**Fitzsimmons RJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fitzsimmons%20RJ%22%5BAuthor%5D)**,** [**Gordon SL**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Gordon%20SL%22%5BAuthor%5D)**,** [**Kronberg J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kronberg%20J%22%5BAuthor%5D)**,** [**Ganey T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ganey%20T%22%5BAuthor%5D)**,** [**Pilla AA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pilla%20AA%22%5BAuthor%5D)**. A pulsing electric field (PEF) increases human chondrocyte proliferation through a transduction pathway involving nitric oxide signaling.** [**J Orthop Res.**](javascript:AL_get(this,%20'jour',%20'J%20Orthop%20%0d%0aRes.');) **26(6):854-859, 2008.**

A potential treatment modality for joint pain due to cartilage degradation is electromagnetic fields (EMF) that can be delivered, noninvasively, to chondrocytes buried within cartilage. A pulsed EMF in clinical use for recalcitrant bone fracture healing has been modified to be delivered as a pulsed electric field (PEF) through capacitive coupling. It was the objective of this study to determine whether the PEF signal could have a direct effect on chondrocytes in vitro. This study shows that a 30-min PEF treatment can increase DNA content of chondrocyte monolayer by approximately 150% at 72 h poststimulus. Studies intended to explore the biological mechanism showed this PEF signal increased nitric oxide measured in culture medium and cGMP measured in cell extract within the 30-min exposure period. Increasing calcium in the culture media or adding the calcium ionophore A23187, without PEF treatment, also significantly increased short-term nitric oxide production. The inhibitor W7, which blocks calcium/calmodulin, prevented the PEF-stimulated increase in both nitric oxide and cGMP. The inhibitor L-NAME, which blocks nitric oxide synthase, prevented the PEF-stimulated increase in nitric oxide, cGMP, and DNA content. An inhibitor of guanylate cyclase (LY83583) blocked the PEF-stimulated increase in cGMP and DNA content. A nitric oxide donor, when present for only 30 min, increased DNA content 72 h later. Taken together, these results suggest the transduction pathway for PEF-stimulated chondrocyte proliferation involves nitric oxide and the production of nitric oxide may be the result of a cascade that involves calcium, calmodulin, and cGMP production.

**(E)** **(VT, AE, IFR)** [**Frahm J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Frahm%20J%22%5BAuthor%5D)**,** [**Lantow M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lantow%20M%22%5BAuthor%5D)**,** [**Lupke M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lupke%20M%22%5BAuthor%5D)**,** [**Weiss DG**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Weiss%20DG%22%5BAuthor%5D)**,** [**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Simk%C3%B3%20M%22%5BAuthor%5D)**. Alteration in cellular functions in mouse macrophages after exposure to 50 Hz magnetic fields.** [**J Cell Biochem.**](javascript:AL_get(this,%20'jour',%20'J%20Cell%20%0d%0aBiochem.');) **99(1):168-177, 2006.**

The aim of the present study is to investigate whether extremely low frequency electromagnetic fields (ELF-EMF) affect certain cellular functions and immunologic parameters of mouse macrophages. In this study, the influence of 50 Hz magnetic fields (MF) at 1.0 mT was investigated on the phagocytic activity and on the interleukin-1beta (IL-1beta) production in differentiated macrophages. MF-exposure led to an increased phagocytic activity after 45 min, shown as a 1.6-fold increased uptake of latex beads in MF-exposed cells compared to controls. We also demonstrate an increased IL-1beta release in macrophages after 24 h exposure (1.0 mT MF). Time-dependent IL-1beta formation was significantly increased already after 4 h and reached a maximum of 12.3-fold increase after 24 h compared to controls. Another aspect of this study was to examine the genotoxic capacity of 1.0 mT MF by analyzing the micronucleus (MN) formation in long-term (12, 24, and 48 h) exposed macrophages. Our data show no significant differences in MN formation or irregular mitotic activities in exposed cells. Furthermore, the effects of different flux densities (ranging from 0.05 up to 1.0 mT for 45 min) of 50 Hz MF was tested on free radical formation as an endpoint of cell activation in mouse macrophage precursor cells. All tested flux densities significantly stimulated the formation of free radicals. Here, we demonstrate the capacity of ELF-EMF to stimulate physiological cell functions in mouse macrophages shown by the significantly elevated phagocytic activity, free radical release, and IL-1beta production suggesting the cell activation capacity of ELF-EMF in the absence of any genotoxic effects.

**(E) (VT, AE, IFR)** [**Frahm J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Frahm%20J%5BAuthor%5D&cauthor=true&cauthor_uid=19913603)**,** [**Mattsson MO**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mattsson%20MO%5BAuthor%5D&cauthor=true&cauthor_uid=19913603)**,** [**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Simk%C3%B3%20M%5BAuthor%5D&cauthor=true&cauthor_uid=19913603)**. Exposure to ELF magnetic fields modulate redox related protein expression in mouse macrophages.** [**Toxicol Lett.**](http://www.ncbi.nlm.nih.gov/pubmed/19913603) **192(3):330-336, 2010.**

The interaction of extremely low frequency (ELF) magnetic fields (MF) with cells can induce alterations in various cell physiological processes. Here, we present evidence that exposure of mouse macrophages to 50 Hz, 1.0 mT MF lead to immune cell activation seen as increased production of reactive oxygen species (ROS), and also to modulation on the expression level of important proteins acting in redox regulatory processes and thus explaining the noted changes in ROS levels seen after exposure. The MF exposure caused slight and transient decreases after short term exposures (2h or less) of clathrin, adaptin, PI3-kinase, protein kinase B (PKB) and PP2A, whereas longer exposures had no effect. The levels of the NAD(P)H oxidase subunit gp91phox oscillated between increased and normal levels compared to controls. The stress proteins Hsp70 and Hsp110 exhibited increased levels at certain time points, but not generally. The effects of MF on protein levels are different from the effects exerted by 12-O-tetradecanolyphobol-13-acetate (TPA) or LPS, although all three factors cause increases in ROS release. This suggests that ELF MF interacts with other cellular constituents than these chemicals, although induced pathways at least partially converge.

**(E)** **(VT, AE, IFR)** [**Garip AI**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Garip%20AI%22%5BAuthor%5D)**,** [**Akan Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Akan%20Z%22%5BAuthor%5D)**. Effect of ELF-EMF on number of apoptotic cells; correlation with reactive oxygen species and HSP.** [**Acta Biol Hung.**](javascript:AL_get(this,%20'jour',%20'Acta%20Biol%20%0d%0aHung.');) **61(2):158-167, 2010.**

It is by now accepted that extremely low frequency electromagnetic fields ELF-EMF (0-300 Hz) affect biological systems although the mechanism has not been elucidated yet. In this study the effect of ELFEMF on the number of apoptotic cells of K562 human leukemia cell line induced or not with oxidative stress and the correlation with heat-shock protein 70 (hsp70) levels was investigated. One sample was treated with H 2 O 2 while the other was left untreated. ELF-EMF (1 mT, 50 Hz) was applied for 3 hours. ELF-EMF alone caused a decrease in the number of apoptotic cells and a slight increase in viability. However, it increased the number of apoptotic cells. In cells treated with H 2 O 2 . hsp70 and reactive oxygen species (ROS) levels were increased by ELF-EMF. These results show that the effect of ELF-EMF on biological systems depends on the status of the cell: while in cells not exposed to oxidative stress it is able to decrease the number of apoptotic cells by inducing an increase in hsp levels, it increases the number of apoptotic cells in oxidative stress-induced cells.

**(E)(VO, CE, IAO, IX)** [**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Amara S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Amara%20S%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Arnaud J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Arnaud%20J%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Garrel C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Garrel%20C%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Faure H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Faure%20H%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Favier A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Favier%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=21505001)**. Effect of selenium pre-treatment on plasma antioxidant vitamins A (retinol) and E (α-tocopherol) in static magnetic field-exposed rats.** [**Toxicol Ind Health.**](http://www.ncbi.nlm.nih.gov/pubmed/21505001) **27(10):949-955, 2011a.**

In the present study, we evaluate the effect of the co-exposure to static magnetic field (SMF) and selenium (Se) on the antioxidant vitamins A and E levels and some other parameters of oxidative stress in rat. Sub-acute exposure of male adult rats to a uniform SMF (128 mT, 1 h/day during 5 consecutive days) increased plasma activity of glutathione peroxidase (+35%) but decreased α-tocopherol (-67%) and retinol levels (-41%). SMF exposure failed to alter the plasmatic thiobarbituric acid-reactive species (TBARs), total thiol groups and selenium concentrations. Sub-chronic administration of Se (Na(2)SeO(3), 0.2 mg/L, for 30 consecutive days, per os) ameliorated the antioxidant capacities in SMF-treated rats. Our investigation demonstrated that sub-acute exposure to SMF induced oxidative stress, which may be prevented by a pretreatment with selenium.

**(E)** **(VO, CE, IAO, DAO)** [**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Amara S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Amara%20S%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Garrel C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Garrel%20C%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Arnaud J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Arnaud%20J%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Ducros V**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ducros%20V%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Favier A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Favier%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=21787674)**. Selenium supplementation ameliorates static magnetic field-induced disorders in antioxidant status in rat tissues.** [**Environ Toxicol Pharmacol.**](http://www.ncbi.nlm.nih.gov/pubmed/21787674) **31(1):100-106, 2011b.**

The aim of this study was to investigate the effect of selenium supplementation on the antioxidant enzymatic system (such as GPx, GR and SOD), GSH and selenium level in liver, kidney, muscle and brain of static magnetic field (SMF) exposed rats. Male adult rats were divided into control rats (n=6), SMF-exposed rats (128 mT; 1h/day for 5 days), selenium-treated rats (Na(2)SeO(3), 0.2mg/l, in drinking water for 4 weeks) and co-exposed rats (selenium for 4 weeks and SMF during the last 5 consecutive days). Sub-acute exposure to SMF induces a decrease of selenium levels in kidney, muscle and brain. Our results also revealed a decrease of GPx activities in kidney and muscle. By contrast, SMF exposure increased total GSH levels and total SOD activities in liver, while glutathione reductase activity is unaffected. Selenium supplementation in SMF-exposed rats restored selenium levels in kidney, muscle and brain and elevated the activities of GPx in kidney and muscle to those of control group. In the liver, selenium supplementation failed to bring down the elevated levels of total GSH and SOD activity. Our investigations suggested that sub-acute exposure to SMF altered the antioxidant response by decreasing the level of total selenium in kidney, muscle and brain. Interestingly, selenium supplementation ameliorates antioxidant capacity in rat tissues exposed to SMF.

[**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24027759)**,** [**Lahbib A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lahbib%20A%5BAuthor%5D&cauthor=true&cauthor_uid=24027759)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=24027759)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=24027759)**. Bioeffects of static magnetic fields: oxidative stress, genotoxic effects, and cancer studies.** [**Biomed Res Int.**](http://www.ncbi.nlm.nih.gov/pubmed/24027759) **2013:602987, 2013. (review)**

The interaction of static magnetic fields (SMFs) with living organisms is a rapidly growing field of investigation. The magnetic fields (MFs) effect observed with radical pair recombination is one of the well-known mechanisms by which MFs interact with biological systems. Exposure to SMF can increase the activity, concentration, and life time of paramagnetic free radicals, which might cause oxidative stress, genetic mutation, and/or apoptosis. Current evidence suggests that cell proliferation can be influenced by a treatment with both SMFs and anticancer drugs. It has been recently found that SMFs can enhance the anticancer effect of chemotherapeutic drugs; this may provide a new strategy for cancer therapy. This review focuses on our own data and other data from the literature of SMFs bioeffects. Three main areas of investigation have been covered: free radical generation and oxidative stress, apoptosis and genotoxicity, and cancer. After an introduction on SMF classification and medical applications, the basic phenomena to understand the bioeffects are described. The scientific literature is summarized, integrated, and critically analyzed with the help of authoritative reviews by recognized experts; international safety guidelines are also cited.

**(E) (VO, CE, IX)** **Ghodbane S1, Amara S, Lahbib A, Louchami K, Sener A, Sakly M, Abdelmelek H. Vitamin E prevents glucose metabolism alterations induced by static magnetic field in rats. Environ Sci Pollut Res Int. 21(22):12731-12738, 2014.**

In the present study, we investigate the effects of a possible protective role of vitamin E (vit E) or selenium (Se) on glucose metabolism disruption induced by static magnetic field (SMF) in rats. Rats have been exposed to SMF (128 mT, 1 h/day during 5 days). Our results showed that SMF failed to alter body weight and relative liver weight. Our data demonstrated that exposure to SMF increased (+21 %) blood glucose level and caused a decrease (-15 %) in liver glycogen content. Moreover, the same treatment induced a reduction of pancreatic islet area. Interestingly, supplementation with vit E (DL α-tocopherol acetate, 150 mg/kg per os during 5 days) prevented alterations induced by SMF on glucose metabolism and liver glycogen content, whereas supplementation with Se (Na2SeO3, 0.20 mg/l, in drinking water for 4 weeks) restored only hepatic glycogen contents. By contrast, both vit E and Se failed to correct the area of pancreatic islets.



**(E) (VO, CE, IAO, AO)** [**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=26062464)**,** [**Ammari M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ammari%20M%5BAuthor%5D&cauthor=true&cauthor_uid=26062464)**,** [**Lahbib A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lahbib%20A%5BAuthor%5D&cauthor=true&cauthor_uid=26062464)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=26062464)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=26062464)**. Static magnetic field exposure-induced oxidative response and caspase-independent apoptosis in rat liver: effect of selenium and vitamin E supplementations.** [**Environ Sci Pollut Res Int.**](http://www.ncbi.nlm.nih.gov/pubmed/26062464) **22(20):16060-16066, 2015a.**

In the present study, we investigated the implication of oxidative stress and apoptosis under static magnetic field (SMF) in the brain and liver. Moreover, we estimated the protective role of selenium and vitamin E in rat tissues against disorders induced by SMF. Exposure of rats to SMF (128 mT, 1 h/day during five consecutive days) increased the activity of catalase (CAT) (+24 %) in the liver but not in the brain. By contrast, the same treatment failed to alter malondialdehyde (MDA) concentration in the brain and liver. Exposure to SMF also induced hepatocyte apoptosis through a caspase-independent pathway involving mitochondrial apoptosis-inducing factor (AIF) but not in the brain. Selenium and vitamin E supplementations to SMF-exposed rats restored liver CAT activity but failed to minimize liver apoptosis.

**(E) (VO, CE, IAO, AO)** [**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25395602)**,** [**Lahbib A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lahbib%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25395602)**,** [**Ammari M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ammari%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25395602)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25395602)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=25395602)**. Does static magnetic field-exposure induced oxidative stress and apoptosis in rat kidney and muscle? Effect of vitamin E and selenium supplementations.** [**Gen Physiol Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/25395602) **34(1):23-32, 2015b.**

Static magnetic fields (SMFs) effect observed with radical pair recombination is one of the well-known mechanisms by which SMFs interact with biological systems. Our aim was to study whether SMF induces oxidative stress and apoptosis in rat tissues and to evaluate the possible protector effect of selenium (Se) and vitamin E (vit E) supplementations. Rats were randomly divided into control, SMF-exposed, Se-treated, vit E-treated, SMF exposed rats and co-treated with Se, and SMF exposed rats and co-treated with vit E. After animal sacrifice, catalase (CAT) activity and malondialdehyde (MDA) concentration were measured and apoptosis inducing factor (AIF) immunohistochemical labeling was performed in kidney and muscle. Exposure of rats to SMF (128 mT, 1 h/day for 5 days) increased the MDA concentrations (+25%) and CAT activities (+34%) in kidney but not in muscle. By contrast, the same treatment failed to induce a caspase-independent pathway apoptosis in both tissues. Interestingly, Se pre-treatment inhibited the increase of MDA concentrations and CAT activities in kidney in SMF-exposed rats. However, vit E administration corrected only MDA levels in rat kidney. In conclusion, exposure to SMF induced oxidative stress in kidney that can be prevented by treatment with Se or vit E.

**(NE)(VT, AE, IX)** [**Giorgi G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Giorgi%20G%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Lecciso M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lecciso%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Capri M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Capri%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Lukas Yani S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lukas%20Yani%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Virelli A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Virelli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Bersani F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bersani%20F%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**,** [**Del Re B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Del%20Re%20B%5BAuthor%5D&cauthor=true&cauthor_uid=25435353)**. An evaluation of genotoxicity in human neuronal-type cells subjected to oxidative stress under an extremely low frequency pulsed magnetic field.** [**Mutat Res Genet Toxicol Environ Mutagen.**](http://www.ncbi.nlm.nih.gov/pubmed/25435353) **775-776:31-37, 2014.**

The possible genotoxicity of extremely low frequency magnetic field (ELF-MF) exposure is still a controversial topic. The most of the reported data suggests that it alone does not affect DNA integrity, but several recent reports have suggested that sinusoidal ELF-MF may increase the effect of known genotoxic agents. Only a few studies deal with non sinusoidal ELF-MF, including pulsed magnetic field (PMF), which are produced by several devices. The aim of this study is to investigate whether PMF exposure can interfere with DNA damage and repair in the presence of a genotoxic oxidative agent in neuronal type cells. To this purpose gamma-H2AX foci formation, which is a sensitive marker of DNA double strand breaks (DSB), was investigated at different points of time (1, 24, 48, 72h) after the H2O2 treatment (300μM for 1h) under PMF exposure (1mT, 50Hz) in human neuroblastoma BE(2)C cells. Moreover, cytotoxicity evaluation, by MTT assay and cell cycle analysis, was performed at various points of time after the treatment. Taken together, results suggest that PMF exposure does not interfere with genotoxicity and cytotoxicity induced by oxidative stress.

**(E) (VO, CE, DOD, IAO)** [**Glinka M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Glinka%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23323798)**,** [**Sieroń A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Siero%C5%84%20A%5BAuthor%5D&cauthor=true&cauthor_uid=23323798)**,** [**Birkner E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Birkner%20E%5BAuthor%5D&cauthor=true&cauthor_uid=23323798)**,** [**Cieślar G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cie%C5%9Blar%20G%5BAuthor%5D&cauthor=true&cauthor_uid=23323798)**. Influence of extremely low-frequency magnetic field on the activity of antioxidant enzymes during skin wound healing in rats.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23323798) **32(4):463-470, 2013.**

The aim of this study was to evaluate the activity of the antioxidant enzymes mitochondrial and cytosolic superoxide dismutase (EC 1.15.1.1), glutathione peroxidase (POX, EC 1.11.1.9) and glutathione S-transferase (EC 3.1.2.7), as well as the concentration of malone dialdehyde (MDA), as an indicator of lipid peroxidation rate in the liver tissue homogenates and blood serum of male rats exposed to extremely low-frequency magnetic field (ELF-MF) in order to improve the healing process of an experimental cut wound on the back of each animal. The exposure to ELF-MF with frequency 40 Hz and magnetic flux density 10 mT induced an increase in POX serum activity and a decrease in MDA contents in the liver tissue, which suggests the inhibition of phospholipid peroxidation and subsequent stabilization of cellular membranes, as a result of ELF-MF action. Based on the results obtained, it seems that ELF-MF could be a useful supplement in the complex treatment of prolonged wound healing, due to the activation of endogenous enzymatic antioxidant system.

**(E)** **(VO, CE, IOD)** [**Gok DK**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Gok%20DK%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**,** [**Akpinar D**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Akpinar%20D%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**,** [**Hidisoglu E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hidisoglu%20E%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**,** [**Ozen S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ozen%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**,** [**Agar A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Agar%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**,** [**Yargicoglu P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Yargicoglu%20P%5BAuthor%5D&cauthor=true&cauthor_uid=25496054)**. The developmental effects of extremely low frequency electric fields on visual and somatosensory evoked potentials in adult rats.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/25496054) **2014 Dec 11:1-10. [Epub ahead of print]**

The purpose of our study was to investigate the developmental effects of extremely low frequency electric fields (ELF-EFs) on visual evoked potentials (VEPs) and somatosensory-evoked potentials (SEPs) and to examine the relationship between lipid peroxidation and changes of these potentials. In this context, thiobarbituric acid reactive substances (TBARS) levels were determined as an indicator of lipid peroxidation. Wistar albino female rats were divided into four groups; Control (C), gestational (prenatal) exposure (Pr), gestational+ postnatal exposure (PP) and postnatal exposure (Po) groups. Pregnant rats of Pr and PP groups were exposed to 50 Hz electric field (EF) (12 kV/m; 1 h/day), while those of C and Po groups were placed in an inactive system during pregnancy. Following parturition, rats of PP and Po groups were exposed to ELF-EFs whereas rats of C and Pr groups were kept under the same experimental conditions without being exposed to any EF during 68 days. On postnatal day 90, rats were prepared for VEP and SEP recordings. The latencies of VEP components in all experimental groups were significantly prolonged versus C group. For SEPs, all components of PP group, P2, N2 components of Pr group and P1, P2, N2 components of Po group were delayed versus C group. As brain TBARS levels were significantly increased in Pr and Po groups, retina TBARS levels were significantly elevated in all experimental groups versus C group. In conclusion, alterations seen in evoked potentials, at least partly, could be explained by lipid peroxidation in the retina and brain.

**(E)** **(VT, CE, IRF, IOD, DAO)** [**Goraca A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Goraca%20A%22%5BAuthor%5D)**,** [**Ciejka E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ciejka%20E%22%5BAuthor%5D)**,** [**Piechota A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Piechota%20A%22%5BAuthor%5D)**. Effects of extremely low frequency magnetic field on the parameters of oxidative stress in heart.** [**J Physiol Pharmacol.**](javascript:AL_get(this,%20'jour',%20'J%20Physiol%20%0d%0aPharmacol.');) **61(3):333-338, 2010.**

Increasing production of free radicals in organisms is one of the putative mechanisms by which a extremely low frequency magnetic field (ELF-MF) may affect biological systems. The present study was designated to assess if ELF-MF applied in the magnetotherapy, affects generation of reactive oxygen species (ROS) in heart tissue and antioxidant capacity of plasma according to its working time. The experiments were performed on 3 groups of animals: group I - control; group II - exposed to 40 Hz, 7 mT, 30 min/day for 14 days (this field is commonly applied in magnetotherapy); group III - exposed to 40 Hz, 7 mT, 60 min/day for 14 days. Control rats were housed in a separate room without exposure to ELF-MF. Immediately after the last exposure, blood was taken from the tail vein and hearts were removed under anesthesia. The effect of the exposure to ELF-MF on oxidative stress was assessed on the basis of the measurements of thiobarbituric acid reactive substances (TBARS), hydrogen peroxide (H(2)O(2)), total free sulphydryl groups (-SH groups) and reduced glutathione (GSH) concentrations in heart homogenates. The total antioxidant capacity of plasma was measured using ferric reducing ability method (FRAP). Exposure to ELF-MF (40 Hz, 7 mT, 30 min/day for 2 weeks) did not significantly alter tissue TBARS, H(2)O(2), total free -SH groups, reduced glutathione (GSH) and total antioxidant capacity of plasma. By contrast, ELF-MF with the same frequency and induction but used for 60 min/day for 14 days caused significant increase in TBARS and H(2)O(2) concentration (P<0.01) and decrease in the concentration of GSH (P<0.05) and total free -SH groups in heart homogenates. Moreover, exposure of rats to ELF-MF (40 Hz, 7 mT, 60 min/day for 2 weeks) resulted in the decrease of plasma antioxidant capacity. Our results indicate that effects of ELF-MF on ROS generation in the heart tissue and antioxidant capacity of plasma depend on its working time.

**(E)** **(VO, CE, IFR, IOD, DAO)** [**Guler G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Guler%20G%22%5BAuthor%5D)**,** [**Turkozer Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Turkozer%20Z%22%5BAuthor%5D)**,** [**Tomruk A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tomruk%20A%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**. The protective effects of N-acetyl-L-cysteine and epigallocatechin-3-gallate on electric field-induced hepatic oxidative stress. I**[**nt J Radiat Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aRadiat%20Biol.');) **84(8):669-680, 2008.**

PURPOSE: To investigate the effects of 12 kV/m electric (E) field sourced by power lines on oxidative and nitrosative stress, and antioxidant status. Furthermore, the study aimed to examine the protective effects of N-Acetyl-L-cysteine (NAC) and epigallocatechin-gallate (EGCG) in the liver tissues of guinea pigs against the possible detriments of electromagnetic field exposure. MATERIALS AND METHODS: Guinea pigs were exposed to 50 Hz 12 kV/m E-field. NAC and EGCG were administered intraperitoneally. Malonedialdehyde (MDA), a product of lipid peroxidation (LPO), and nitric oxide derivatives (nitrate (NO(3)), nitrite (NO(2)), total level of nitric oxide (NO(x)) were estimated as biomarkers of oxidative and nitrosative stress, respectively. Superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), and myeloperoxidase (MPO) were evaluated as endogenous antioxidant enzymes in liver tissues of the guinea pigs. RESULTS: The results of our study indicated a significant increase in the levels of oxidant products (MDA, NO(3), NO(2), NO(x)), and a significant decrease in antioxidant enzyme (SOD, GSH-Px and MPO) activities. We also found that the individual or plus application of NAC and EGCG resulted in the reduction of oxidative stress prior to E field application. CONCLUSION: To conclude, extremely low frequency (ELF) electric field has potential harmful effects on the living organisms by enhancing the free radical production. NAC and EGCG might have hepatoprotective effects in ELF-E field induced oxidative and nitrosative stress.

**(NE)** **(VO, CE)** [**Güler G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22G%C3%BCler%20G%22%5BAuthor%5D)**,** [**Türközer Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22T%C3%BCrk%C3%B6zer%20Z%22%5BAuthor%5D)**,** [**Ozgur E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ozgur%20E%22%5BAuthor%5D)**,** [**Tomruk A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tomruk%20A%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**,** [**Karasu C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Karasu%20C%22%5BAuthor%5D)**. Protein oxidation under extremely low frequency electric field in guinea pigs. Effect of N-acetyl-L-cysteine treatment.** [**Gen Physiol Biophys.**](javascript:AL_get(this,%20'jour',%20'Gen%20%0d%0aPhysiol%20Biophys.');) **28(1):47-55, 2009a.**

Modern age exposes humans to an increasing level of electromagnetic activity in their environment due to overhead power lines and transformers around residential areas. Studies have shown that treatment with antioxidants can suppress the oxidative damage induced by electromagnetic fields in various frequencies of the non-ionizing radiation band. In this study, we detected protein carbonyl content (PCO), advanced oxidation protein products (AOPP) in liver and 3-nitrotyrosine (3-NT) levels in plasma of guinea pigs in order to investigate the effects of N-acetyl-L-cysteine (NAC) administration on oxidative protein damage induced by power frequency electric (E) field (50 Hz, 12 kV/m, 7 days/8 h/day). We also analyzed hepatic hydroxyproline level to study protein synthesis. According to the findings of the present study, no statistically significant changes occurred in PCO, AOPP and 3-NT levels of the guinea pigs that were exposed to the E field with respect to the control group. However, liver hydroxyproline level was significantly diminished in the E field exposure group compared to the control and PCO, hydroxyproline and 3-NT levels changed significantly in the NAC-administrated groups.

**(NE)** **(VO, CE)** [**Güler G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22G%C3%BCler%20G%22%5BAuthor%5D)**,** [**Türközer Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22T%C3%BCrk%C3%B6zer%20Z%22%5BAuthor%5D)**,** [**Ozgur E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ozgur%20E%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**. Antioxidants alleviate electric field-induced effects on lung tissue based on assays of heme oxygenase-1, protein carbonyl content, malondialdehyde, nitric oxide, and hydroxyproline.** [**Sci Total Environ.**](javascript:AL_get(this,%20'jour',%20'Sci%20Total%20%0d%0aEnviron.');) **407(4):1326-1332, 2009b.**

In order to test whether antioxidants have beneficiary effects on electric field induced damage, we determined the pulmonary levels of heme oxygenase-1 (HO-1), protein carbonyl content (PCO), malondialdehyde (MDA), nitric oxide (NO) and hydroxyproline (HP) under extremely low frequency (ELF) electric (E) field exposure (50 Hz, 12 kV/m, 7 days/for 8 h/day). While PCO levels significantly increased (p<0.05), insignificant changes (p>0.05) were observed in HO-1, MDA, NO and HP levels for electric field exposure groups compared to the control group. We have not observed any significant change in these parameters on the electric field group compared to the group where NAC and EGCG were separately applied along with electric field. However, during our previous studies, we have concluded that NAC and EGCG are potent antioxidants and we believe that new studies should be established by way of setting up different experimental conditions.

**(E)** **(VO, CE, IAO, LI)** [**Hajnorouzi A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Hajnorouzi%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21227536)**,** [**Vaezzadeh M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Vaezzadeh%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21227536)**,** [**Ghanati F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghanati%20F%5BAuthor%5D&cauthor=true&cauthor_uid=21227536)**,** [**Jamnezhad H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jamnezhad%20H%5BAuthor%5D&cauthor=true&cauthor_uid=21227536)**,** [**Nahidian B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Nahidian%20B%5BAuthor%5D&cauthor=true&cauthor_uid=21227536)**. Growth promotion and a decrease of oxidative stress in maize seedlings by a combination of geomagnetic and weak electromagnetic fields.** [**J Plant Physiol.**](http://www.ncbi.nlm.nih.gov/pubmed/21227536) **168(10):1123-1128, 2011.**

In the present study, we hypothesized that an appropriate combination of a geomagnetic field (as a static field) and an alternative magnetic field may result in the promotion of maize seedling growth by an alleviation of an excess production of reactive oxygen species. First, we determined the applicable range of frequencies by theoretical calculations, and a combined magnetic field was designed. The seeds were germinated in the magnetic field for 4 days, and the seedlings were allowed to grow in a nutrient solution for another 4 days. The magnetic field-treated maize seeds produced seedlings with a faster growth rate than the control seeds. The activity of superoxide dismutase in the magnetic field-treated seedlings was lower, while the total antioxidant capacity of these seedlings was higher than that of the control group. The maintenance of membrane integrity and a decrease of iron content in the magnetic field-treated seedlings suggest that a combination of both static and alternative magnetic fields promotes the growth of the plants by lowering iron absorption, a reduction in the Fenton chemistry, and lowering the risk of oxidative burst.

**(E) (VO, AE, IOD, IAO)** [**Hanini R**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Hanini%20R%5BAuthor%5D&cauthor=true&cauthor_uid=28523373)**,** [**Chatti A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chatti%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28523373)**,** [**Ghorbel SB**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ghorbel%20SB%5BAuthor%5D&cauthor=true&cauthor_uid=28523373)**,** [**Landoulsi A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Landoulsi%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28523373)**. Role of SOD gene in response to static magnetic fields in Pseudomonas aeruginosa.** [**Curr Microbiol.**](https://www.ncbi.nlm.nih.gov/pubmed/28523373) **2017 May 18. doi: 10.1007/s00284-017-1264-4. [Epub ahead of print]**

The protective role of superoxide dismutase (SOD) against non-ionizing radiation such as static electromagnetic field (200 mT) has been studied in wild-type and mutant strain of Pseudomonas aeruginosa lacking cytosolic Mn-SOD (sodM), Fe-SOD (sodB), or both SODs (sodMB). Our results showed that inactivation of sodM and/or sodB genes increases the sensitivity of P. aeruginosa toward stress induced by the static magnetic field (200 mT). Furthermore, our results showed an enhancement of SOD, catalase, and peroxidases after exposure to the magnetic field. However, wild-type cells maintained significantly higher activities of antioxidant enzymes than mutant strains. The malondialdehyde produced by the oxidative degradation of unsaturated lipids and fatty acids showed significant increase in mutant strains compared to the wild-type. The overall results showed that the SOD has a protective role against a stress induced by static electromagnetic field in P. aeruginosa.

**(NE)** **(VO, CE)** [**Harakawa S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Harakawa%20S%22%5BAuthor%5D)**,** [**Inoue N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Inoue%20N%22%5BAuthor%5D)**,** [**Hori T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hori%20T%22%5BAuthor%5D)**,** [**Tochio K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tochio%20K%22%5BAuthor%5D)**,** [**Kariya T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kariya%20T%22%5BAuthor%5D)**,** [**Takahashi K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Takahashi%20K%22%5BAuthor%5D)**,** [**Doge F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Doge%20F%22%5BAuthor%5D)**,** [**Suzuki H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Suzuki%20H%22%5BAuthor%5D)**,** [**Nagasawa H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nagasawa%20H%22%5BAuthor%5D)**. Effects of a 50 Hz electric field on plasma lipid peroxide level and antioxidant activity in rats.** [**Bioelectromagnetics.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Bioelectromagnetics.');) **26(7):589-594, 2005.**

The effects of exposure to extremely low frequency electric fields (ELF EFs) on plasma lipid peroxide levels and antioxidant activity (AOA) in Sprague-Dawley rats were studied. The test was based on comparisons among rats treated with a combination of the oxidizing agent, 2,2'-azobis(2-aminopropane) dihydrochloride (AAPH) and 50 Hz EF of 17.5 kV/m intensity for 15 min per day for 7 days, AAPH alone, EF alone or no treatment. EF significantly decreased the plasma peroxide level in rats treated with AAPH, similar to treatment by ascorbic acid or the superoxide dismutase. Ascorbic acid increased AOA; however, EF and superoxide dismutase did not change AOA compared with sham exposure in stressed rats. No influence on the lipid peroxide level and AOA in unstressed rats was observed with EF exposure alone. Although the administration of AAPH decreased AOA, this decrease did not change when EF was added. These data indicate that the ELF EF used in this study influenced the lipid peroxide level in an oxidatively stressed rat.

**(E)** **(VO, CE, IOD, DAO)** [**Hashish AH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hashish%20AH%22%5BAuthor%5D)**,** [**El-Missiry MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22El-Missiry%20MA%22%5BAuthor%5D)**,** [**Abdelkader HI**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Abdelkader%20HI%22%5BAuthor%5D)**,** [**Abou-Saleh RH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Abou-Saleh%20RH%22%5BAuthor%5D)**. Assessment of biological changes of continuous whole body exposure to static magnetic field and extremely low frequency electromagnetic fields in mice.** [**Ecotoxicol Environ Saf.**](javascript:AL_get(this,%20'jour',%20'Ecotoxicol%0d%0a%20Environ%20Saf.');) **71(3):895-902. 2008.**

The question whether static magnetic fields (SMFs) and extremely low frequency electromagnetic fields (ELF-EMF) cause biological effects is of special interest. We investigated the effects of continuous whole body exposure to both fields for 30 days on some liver and blood parameters in mice. Two exposure systems were designed; the first produced a gradient SMF while the second generated uniform 50Hz ELF-EMF. The results showed a gradual body weight loss when mice were exposed to either field. This is coupled with a significant decrease (P<0.05) in the levels of glucose, total protein and the activity of alkaline phosphatase in serum. A significant increase in lactate dehydrogenase activity was demonstrated in serum and liver paralleled with a significant elevation in hepatic gamma-glutamyl transferase activity. The glutathione-S-transferase activity and lipid peroxidation level in the liver were significantly increased while a significant decrease in hepatic gluthathione content was recorded. A significant decrease in the counts of monocytes, platelets, peripheral lymphocytes as well as splenic total, T and B lymphocytes levels was observed for SMF and ELF-EMF exposed groups. The granulocytes percentage was significantly increased. The results indicate that there is a relation between the exposure to SMF or ELF-EMF and the oxidative stress through distressing redox balance leading to physiological disturbances.

**(E) (VT, AE, IOD, IFR, IAO, DAO)** [**Henrykowska G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Henrykowska%20G%22%5BAuthor%5D)**,** [**Jankowski W**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jankowski%20W%22%5BAuthor%5D)**,** [**Pacholski K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pacholski%20K%22%5BAuthor%5D)**,** [**Lewicka M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lewicka%20M%22%5BAuthor%5D)**,** [**Smigielski J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Smigielski%20J%22%5BAuthor%5D)**,** [**Dziedziczak-Buczyńska M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dziedziczak-Buczy%C5%84ska%20M%22%5BAuthor%5D)**,** [**Buczyński A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Buczy%C5%84ski%20A%22%5BAuthor%5D)**. The effect of 50 Hz magnetic field of different shape on oxygen metabolism in blood platelets: in vitro studies.** [**Int J Occup Med Environ Health.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aOccup%20Med%20Environ%20Health.');) **22(3):269-276, 2009.**

OBJECTIVES: The aim of the study was to assess the influence that the shape of low frequency magnetic field may have on catalase and superoxide dismutase activity, malondialdehyde concentration and free radicals generation in human blood platelets. MATERIALS AND METHODS: The suspension of human blood platelets was exposed for 15 min to 50 Hz magnetic field of different shape, and flux density of 10 mT. RESULTS: The determinations of free radicals, malondialdehyde and catalase showed increased values compared with the initial level, regardless of the shape of the magnetic field applied. In contrast, superoxide dismutase activity was lower than at the onset of the experiment. CONCLUSIONS: The findings indicate that the oxidative stress resulting from exposure to 50 Hz magnetic field of 10 mT induction may produce a number of adverse effects within the cell and thus may lead to systemic disturbances in the human body.

**(NE) (VT, AE)** [**Hong MN**](http://www.ncbi.nlm.nih.gov/pubmed?term=Hong%20MN%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Han NK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Han%20NK%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Lee HC**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20HC%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Ko YK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ko%20YK%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Chi SG**](http://www.ncbi.nlm.nih.gov/pubmed?term=Chi%20SG%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Gimm YM**](http://www.ncbi.nlm.nih.gov/pubmed?term=Gimm%20YM%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=22302048)**. Extremely low frequency magnetic fields do not elicit oxidative stress in MCF10A cells.** [**Radiat Res.**](http://www.ncbi.nlm.nih.gov/pubmed/22302048) **53(1):79-86, 2012.**

The aim of this study was to determine whether extremely low frequency magnetic fields (ELF-MF) could affect intracellular reactive oxygen species (ROS) levels and antioxidant enzyme activity. After MCF10A human breast epithelial cells were exposed to 1 mT of 60 Hz ELF-MF for 4 hours, intracellular ROS level, superoxide dismutase (SOD) activity, and reduced to oxidized glutathione (GSH/GSSG) ratio were measured. The cells exposed to ELF-MF did not evidence statistically significant changes in the above-mentioned biological parameters as compared to either the incubator controls or sham-exposed cells. By way of contrast, the IR-exposed cells exhibited marked changes in ROS level, SOD activity, and GSH/GSSG ratio. When we assessed morphological changes and senescence-associated beta-galactosidase (SA-β-Gal) activity, only the IR-exposed cells were positive. According to our results, it could be concluded that ELF-MF has no effect on intracellular ROS level, SOD activity, and GSH/GSSG ratio under our exposure condition.

**(E) (VT, AE, IFR)** [**Höytö A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=H%C3%B6yt%C3%B6%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28264623)**,** [**Herrala M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Herrala%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28264623)**,** [**Luukkonen J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Luukkonen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28264623)**,** [**Juutilainen J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Juutilainen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28264623)**,** [**Naarala J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Naarala%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28264623)**. Cellular detection of 50 Hz magnetic fields and weak blue light: effects on superoxide levels and genotoxicity.** [**Int J Radiat Biol.**](https://www.ncbi.nlm.nih.gov/pubmed/28264623) **7:1-7, 2017.**

PURPOSE: We tested the hypothesis that the effects of 50 Hz magnetic fields (MFs) on superoxide levels and genotoxicity depend on the presence of blue light. MATERIALS AND METHODS: Human SH-SY5Y neuroblastoma cells were exposed to a 50 Hz, 100 μT MF with or without non-phototoxic level of blue light for 24 h. We also studied whether these treatments alter responses to menadione, an agent that induces mitochondrial superoxide (O2• -) production and DNA damage. Micronuclei, proliferation, viability, cytosolic and mitochondrial O2• - levels were assessed. RESULTS: MF (without blue light) increased cytosolic O2• - production and blue light suppressed this effect. Mitochondrial O2• - production was reduced by both MF and blue light, but these effects were not additive. Micronucleus frequency was not affected by blue light or MF alone, but blue light (significantly when combined with MF) enhanced menadione-induced micronuclei. CONCLUSIONS: The original simple hypothesis (blue light is needed for MF effects) was not supported, but interaction of MF and blue light was nevertheless observed. The results are consistent with MF effects on light-independent radical reactions.

**(E)** **(VT, AE, IX, AO)** [**Jajte J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jajte%20J%22%5BAuthor%5D)**,** [**Zmyślony M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zmy%C5%9Blony%20M%22%5BAuthor%5D)**,** [**Palus J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Palus%20J%22%5BAuthor%5D)**,** [**Dziubałtowska E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dziuba%C5%82towska%20E%22%5BAuthor%5D)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Rajkowska%20E%22%5BAuthor%5D)**. Protective effect of melatonin against in vitro iron ions and 7 mT 50 Hz magnetic field-induced DNA damage in rat lymphocytes.** [**Mutat Res.**](javascript:AL_get(this,%20'jour',%20'Mutat%20%0d%0aRes.');) **483(1-2):57-64, 2001.**

We have previously shown that simultaneous exposure of rat lymphocytes to iron ions and 50Hz magnetic field (MF) caused an increase in the number of cells with DNA strand breaks. Although the mechanism of MF-induced DNA damage is not known, we suppose that it involves free radicals. In the present study, to confirm our hypothesis, we have examined the effect of melatonin, an established free radicals scavenger, on DNA damage in rat peripheral blood lymphocytes exposed in vitro to iron ions and 50Hz MF. The alkaline comet assay was chosen for the assessment of DNA damage. During pre-incubation, part of the cell samples were supplemented with melatonin (0.5 or 1.0mM). The experiments were performed on the cell samples incubated for 3h in Helmholtz coils at 7mT 50Hz MF. During MF exposure, some samples were treated with ferrous chloride (FeCl2, 10microg/ml), while the rest served as controls. A significant increase in the number of cells with DNA damage was found only after simultaneous exposure of lymphocytes to FeCl2 and 7mT 50Hz MF, compared to the control samples or those incubated with FeCl2 alone. However, when the cells were treated with melatonin and then exposed to iron ions and 50Hz MF, the number of damaged cells was significantly reduced, and the effect depended on the concentration of melatonin. The reduction reached about 50% at 0.5mM and about 100% at 1.0mM. Our results indicate that melatonin provides protection against DNA damage in rat lymphocytes exposed in vitro to iron ions and 50Hz MF (7mT). Therefore, it can be suggested that free radicals may be involved in 50Hz magnetic field and iron ions-induced DNA damage in rat blood lymphocytes. The future experimental studies, in vitro and in vivo, should provide an answer to the question concerning the role of melatonin in the free radical processes in the power frequency magnetic field.

**(E)** **(VT, AE, IX)** [**Jajte J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jajte%20J%5BAuthor%5D&cauthor=true&cauthor_uid=12160605)**,** [**Grzegorczyk J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Grzegorczyk%20J%5BAuthor%5D&cauthor=true&cauthor_uid=12160605)**,** [**Zmyślony M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zmy%C5%9Blony%20M%5BAuthor%5D&cauthor=true&cauthor_uid=12160605)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rajkowska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=12160605)**. Effect of 7 mT static magnetic field and iron ions on rat lymphocytes: apoptosis, necrosis and free radical processes.** [**Bioelectrochemistry.**](http://www.ncbi.nlm.nih.gov/pubmed/12160605) **57(2):107-111, 2002.**

Simultaneous exposure of rat lymphocytes to 7 mT static magnetic field (SMF) and iron ions caused an increase in the number of cells with DNA damage. The mechanism by which MF induces DNA damage and the possible cytotoxic consequences are not known. However, we suppose that free radicals are involved. Potentially, the deterioration of DNA molecules by simultaneous exposure to 7 mT SMF and iron ions may lead to cell death: apoptosis or necrosis. The possible prooxidative properties of these two agents may result in an induction of the lipid peroxidation process as a marker of free radical mechanism in the cells. Experiments were performed on rat blood lymphocytes incubated for 3 h in Helmholtz coils at SMF of flux density 7 mT. During SMF exposure, some samples were treated with ferrous chloride (10 microg/ml), the rest serving as controls. We used the dye exclusion method with the DNA-fluorochromes: ethidium bromide and acridine orange. No significant differences were observed between unexposed lymphocytes incubated with medium alone and lymphocytes exposed to 7 mT SMF. Three-hour incubation with FeCl(2) (10 microg/ml) did not affect cell viability. However, when lymphocytes were exposed to 7 mT SMF and simultaneously treated with FeCl(2), there was a significant increase in the percentage of apoptotic and necrotic cells accompanied by significant alterations in cell viability. As compared to lipid peroxidation, there is a significant increase in the amount of lipid peroxidation end products MDA+4 HNE in rat lymphocytes after simultaneous exposure to 7 mT SMF and FeCl(2) (vs. to the control samples and those exposed to SMF alone). This suggests that 7 mT static magnetic field in the presence of Fe(2+) ions can increase the concentration of oxygen free radicals and thus may lead to cell death.

**(E) (VT, AE, IX, AO)** [**Jajte J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jajte%20J%5BAuthor%5D&cauthor=true&cauthor_uid=12731401)**,** [**Zmyślony M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zmy%C5%9Blony%20M%5BAuthor%5D&cauthor=true&cauthor_uid=12731401)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rajkowska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=12731401)**. [Protective effect of melatonin and vitamin E against prooxidative action of iron ions and static magnetic field].** [**Med Pr.**](http://www.ncbi.nlm.nih.gov/pubmed/12731401) **54(1):23-28, 2003. [Article in Polish]**

The purpose of this study was to examine the effect of melatonin and vitamin E (trolox) on the level of lipid peroxidation in rat blood lymphocytes after in vitro (3 h) exposure to iron ions and/or 7mT static magnetic field (SMF). The lipid peroxidation process was chosen as a marker of free radical mechanism of SMF in cells. The cells were supplemented with (0.5 mM) melatonin or (0.1 mM) vitamin E (trolox) in preincubation. During SMF exposure in Helmholtz coils some samples were treated with ferrous chloride (10 mg/ml or 20 mg/ml), while the rest served as controls. There is a significant increase in the amount of lipid peroxidation end-products (4-HNE + MDA) in rat lymphocytes after simultaneous exposure to 7 mT SMF and iron ions (versus control samples and those exposed to SMF alone). Instead, when the cells were treated with melatonin or trolox and then exposed to iron ions and 7 mT SMF, the level of lipid peroxidation was significantly reduced. The results also indicated that melatonin is less effective than vitamin E (trolox) in inhibiting lipid peroxidation under the experimental conditions used.

**(E)** **(VO, CE, IFR, IOD, IAO)** [**Jelenković A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jelenkovi%C4%87%20A%22%5BAuthor%5D)**,** [**Janać B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jana%C4%87%20B%22%5BAuthor%5D)**,** [**Pesić V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pesi%C4%87%20V%22%5BAuthor%5D)**,** [**Jovanović DM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jovanovi%C4%87%20DM%22%5BAuthor%5D)**,** [**Vasiljević I**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Vasiljevi%C4%87%20I%22%5BAuthor%5D)**,** [**Prolić Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Proli%C4%87%20Z%22%5BAuthor%5D)**. Effects of extremely low-frequency magnetic field in the brain of rats.** [**Brain Res Bull.**](javascript:AL_get(this,%20'jour',%20'Brain%20Res%20%0d%0aBull.');) **68(5):355-360, 2006.**

An extremely low-frequency magnetic field (50 Hz, 0.5 mT) was used to investigate its possible effect on the brain of adult male Wistar rats following a 7-day exposure. The control rats were sham-exposed. Superoxide dismutase activities and production of superoxide radicals, lipid peroxidation, and nitric oxide were examined in the frontal cortex, striatum, basal forebrain, hippocampus, brainstem, and cerebellum. Significantly increased superoxide radical contents were registered in all the structures examined. Production of nitric oxide, which can oppose superoxide radical activities, was significantly increased in some structures: the frontal cortex, basal forebrain, hippocampus, and brainstem. Augmentation of lipid peroxydation was also observed, with significance only in the basal forebrain and frontal cortex, in spite of the significantly increased superoxide dismutase activities and nitric oxide production in the basal forebrain, and increased production of nitric oxide in the frontal cortex. The results obtained indicate that a 7-day exposure to extremely low-frequency magnetic field can be harmful to the brain, especially to the basal forebrain and frontal cortex due to development of lipid peroxidation. Also, high production of superoxide anion in all regions may compromise nitric oxide signaling processes, due to nitric oxide consumption in the reaction with the superoxide radical.

**(E)** **(VO, AE, IFR)** [**Jeong JH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jeong%20JH%22%5BAuthor%5D)**,** [**Kum C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kum%20C%22%5BAuthor%5D)**,** [**Choi HJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Choi%20HJ%22%5BAuthor%5D)**,** [**Park ES**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Park%20ES%22%5BAuthor%5D)**,** [**Sohn UD**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sohn%20UD%22%5BAuthor%5D)**. Extremely low frequency magnetic field induces hyperalgesia in mice modulated by nitric oxide synthesis.** [**Life Sci.**](javascript:AL_get(this,%20'jour',%20'Life%20%0d%0aSci.');) **78(13):1407-1412, 2006.**

We investigated an effect of extremely low frequency magnetic field (ELF-MF, 60 Hz) on hyperalgesia using hot plate test. The level of nitric oxide (NO) and the expression of nitric oxide synthase (NOS) were measured to determine if ELF-MF is engaged in NO mediated pain mechanism. Additionally, the involvement of Ca2+-dependent NO pathway in ELF-MF induced hyperalgesia was evaluated by blocking Ca2+ sources with NMDA receptor antagonist and Ca2+ channel blocker. The exposure of mice to ELF-MF lowered pain threshold and elevated NO synthesis in brain and spinal cord. An NOS inhibitor blocked these effects of ELF-MF with attenuating the reduction of pain threshold and the rise of NO level in brain and spine by the exposure of ELF-MF. The hyperalgesic effects of ELF-MF were also blocked by a Ca2+ channel blocker, nimodipine, but not by a NMDA receptor antagonist, MK-801. The expression of Ca2+ -dependent nNOS and eNOS and Ca2+ -independent iNOS were not changed by ELF-MF. These results indicated that the exposure of ELF-MF might cause Ca2+ -dependent NOS activation, which then induces hyperalgesia with the increase in NO synthesis. In conclusion, ELF-MF may produce hyperalgesia by modulating NO synthesis via Ca2+ -dependent NOS.

**(NE) (VT, AE)** [**Jin H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jin%20H%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**,** [**Yoon HE**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Yoon%20HE%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**,** [**Kim JK**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kim%20JK%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=25729273)**. Effects on g2/m phase cell cycle distribution and aneuploidy formation of exposure to a 60 Hz electromagnetic field in combination with ionizing radiation or hydrogen peroxide in l132 nontumorigenic human lung epithelial cells.** [**Korean J Physiol Pharmacol.**](http://www.ncbi.nlm.nih.gov/pubmed/25729273) **19(2):119-124, 2015.**

The aim of the present study was to assess whether exposure to the combination of an extremely low frequency magnetic field (ELF-MF; 60 Hz, 1 mT or 2 mT) with a stress factor, such as ionizing radiation (IR) or H2O2, results in genomic instability in non-tumorigenic human lung epithelial L132 cells. To this end, the percentages of G2/M-arrested cells and aneuploid cells were examined. Exposure to 0.5 Gy IR or 0.05 mM H2O2 for 9 h resulted in the highest levels of aneuploidy; however, no cells were observed in the subG1 phase, which indicated the absence of apoptotic cell death. Exposure to an ELF-MF alone (1 mT or 2 mT) did not affect the percentages of G2/M-arrested cells, aneuploid cells, or the populations of cells in the subG1 phase. Moreover, when cells were exposed to a 1 mT or 2 mT ELF-MF in combination with IR (0.5 Gy) or H2O2 (0.05 mM), the ELF-MF did not further increase the percentages of G2/M-arrested cells or aneuploid cells. These results suggest that ELF-MFs alone do not induce either G2/M arrest or aneuploidy, even when administered in combination with different stressors.

**(NE) (VT, AE)** [**Jin YB**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jin%20YB%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Kang GY**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kang%20GY%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Choi JI**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Choi%20JI%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Lee JW**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20JW%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Hong SC**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hong%20SC%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=22191540)**. Effects on micronuclei formation of 60-Hz electromagnetic field exposure with ionizing radiation, hydrogen peroxide, or c-Myc overexpression.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/22191540) **88(4):374-80, 2012.**

PURPOSE: Epidemiological studies have demonstrated a possible correlation between exposure to extremely low-frequency magnetic fields (ELF-MF) and cancer. However, this correlation has yet to be definitively confirmed by epidemiological studies. The principal objective of this study was to assess the effects of 60 Hz magnetic fields in a normal cell line system, and particularly in combination with various external factors, via micronucleus (MN) assays. MATERIALS AND METHODS: Mouse embryonic fibroblast NIH3T3 cells and human lung fibroblast WI-38 cells were exposed for 4 h to a 60 Hz, 1 mT uniform magnetic field with or without ionizing radiation (IR, 2 Gy), H(2)O(2) (100 μM) and cellular myelocytomatosis oncogene (c-Myc) activation. RESULTS: The results obtained showed no significant differences between the cells exposed to ELF-MF alone and the unexposed cells. Moreover, no synergistic effects were observed when ELF-MF was combined with IR, H(2)O(2), and c-Myc activation. CONCLUSIONS: Our results demonstrate that ELF-MF did not enhance MN frequency by IR, H(2)O(2) and c-Myc activation.

**(NE)** **(VT, AE)** [**Jin YB**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jin%20YB%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Choi SH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Choi%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Kim JK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kim%20JK%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Lee JW**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20JW%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Hong SC**](http://www.ncbi.nlm.nih.gov/pubmed?term=Hong%20SC%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=24305851)**. Absence of DNA damage after 60-Hz electromagnetic field exposure combined with ionizing radiation, hydrogen peroxide, or c-Myc overexpression.** [**Radiat Environ Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/24305851) **53(1):93-101, 2014.**

The principal objective of this study was to assess the DNA damage in a normal cell line system after exposure to 60 Hz of extremely low frequency magnetic field (ELF-MF) and particularly in combination with various external factors, via comet assays. NIH3T3 mouse fibroblast cells, WI-38 human lung fibroblast cells, L132 human lung epithelial cells, and MCF10A human mammary gland epithelial cells were exposed for 4 or 16 h to a 60-Hz, 1 mT uniform magnetic field in the presence or absence of ionizing radiation (IR, 1 Gy), H2O2 (50 μM), or c-Myc oncogenic activation. The results obtained showed no significant differences between the cells exposed to ELF-MF alone and the unexposed cells. Moreover, no synergistic or additive effects were observed after 4 or 16 h of pre-exposure to 1 mT ELF-MF or simultaneous exposure to ELF-MF combined with IR, H2O2, or c-Myc activation.

**(E) (VO, CE, IOD, DAO)** [**Jouni FJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jouni%20FJ%5BAuthor%5D&cauthor=true&cauthor_uid=22108253)**,** [**Abdolmaleki P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Abdolmaleki%20P%5BAuthor%5D&cauthor=true&cauthor_uid=22108253)**,** [**Ghanati F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghanati%20F%5BAuthor%5D&cauthor=true&cauthor_uid=22108253)**. Oxidative stress in broad bean (Vicia faba L.) induced by static magnetic field under natural radioactivity.** [**Mutat Res.**](http://www.ncbi.nlm.nih.gov/pubmed/22108253) **741(1-2):116-121, 2012.**

The investigation was performed to evaluate the influence of the static magnetic field on oxidative stress in Vicia faba cultivated in soil from high background natural radioactivity in Iran. Soil samples were collected from Ramsar, Iran where the annual radiation absorbed dose from background radiation is substantially higher than 20 mSv/year. The soil samples were then divided into 2 separate groups including high and low natural radioactivity. The plants were continuously exposed to static magnetic field of 15 mT for 8 days, each 8h/day. The results showed that in the plants cultivated in soils with high background natural radioactivity and low background natural radioactivity the activity of antioxidant enzymes as well as flavonoid content were lower than those of the control. Treatment of plants with static magnetic field showed similar results in terms of lowering of antioxidant defense system and increase of peroxidation of membrane lipids. Accumulation of ROS also resulted in chromosomal aberration and DNA damage. This phenomenon was more pronounced when a combination of natural radiation and treatment with static magnetic field was applied. The results suggest that exposure to static magnetic field causes accumulation of reactive oxygen species in V. faba and natural radioactivity of soil exaggerates oxidative stress.

**(E) (VO, CE, IOD)** [**Kantar Gok D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kantar%20Gok%20D%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Akpinar D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akpinar%20D%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Yargicoglu P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yargicoglu%20P%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Ozen S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ozen%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Aslan M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Aslan%20M%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Demir N**](http://www.ncbi.nlm.nih.gov/pubmed?term=Demir%20N%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Derin N**](http://www.ncbi.nlm.nih.gov/pubmed?term=Derin%20N%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**,** [**Agar A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Agar%20A%5BAuthor%5D&cauthor=true&cauthor_uid=24811084)**. Effects of extremely low-frequency electric fields at different intensities and exposure durations on mismatch negativity.** [**Neuroscience.**](http://www.ncbi.nlm.nih.gov/pubmed/24811084) **272C:154-166, 2014.**

The effects of extremely low-frequency electric fields (ELF-EFs, 3-300Hz) on lipid peroxidation levels and antioxidant enzyme activities have been shown in many tissues and plasma after exposure to 50-Hz alternating current (AC) electric fields. However, similar studies investigating brain lipid peroxidation status are limited. Moreover and as far as we know, no study has been conducted to examine mismatch negativity (MMN) response in rats following exposure to a 50-Hz AC electric field. Therefore, the purpose of the study was to investigate different intensities and exposure durations of ELF-EFs on MMN component of event-related potentials (ERPs) as well as apoptosis and oxidative brain damage in rats. Ninety male rats, aged 3months were used in our study. A total of six groups, composed of 15 animals each, was formed as follows: sham-exposed rats for 2weeks (C2), sham-exposed rats for 4weeks (C4), rats exposed to 12-kV/m and 18-kV/m electric fields for 2weeks (E12-2 and E18-2), rats exposed to 12- and 18-kV/m electric fields for 4weeks (E12-4 and E18-4). At the end of the experimental period, MMN responses were recorded in urethane-anesthetized rats by electrodes positioned stereotaxically to the surface of the dura. After MMN recordings, animals were killed by exsanguination and their brain tissues were removed for 4-hydroxy-2-nonenal (4-HNE), protein carbonyl and TUNEL analysis. In the current study, different change patterns in ERP parameters were observed dependent on the intensity and exposure duration of ELF-EFs. There were differences in the amplitudes of ERP between the responses to the standard and the deviant tones in all groups. When peak-to-peak amplitude of the difference curves was evaluated, MMN amplitude was significantly decreased in the E18-4 group compared with the C4 group. Additionally, the amount of 4-HNE was increased in all experimental groups compared with the control group. Consequently, it could be concluded that electric field decreased MMN amplitudes possibly induced by lipid peroxidation.

**(E)** **(VO, AE, IFR)** [**Kavaliers M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kavaliers%20M%22%5BAuthor%5D)**,** [**Choleris E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Choleris%20E%22%5BAuthor%5D)**,** [**Prato FS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Prato%20FS%22%5BAuthor%5D)**,** [**Ossenkopp K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ossenkopp%20K%22%5BAuthor%5D)**. Evidence for the involvement of nitric oxide and nitric oxide synthase in the modulation of opioid-induced antinociception and the inhibitory effects of exposure to 60-Hz magnetic fields in the land snail.** [**Brain Res.**](javascript:AL_get(this,%20'jour',%20'Brain%20%0d%0aRes.');) **809(1):50-57, 1998.**

The attenuation of opioid peptide-mediated antinociception is a well-established effect of extremely low frequency (ELF) electromagnetic fields with alterations in calcium channel function and/or calcium ion flux and protein kinase C activity being implicated in the mediation of these effects. The present study was designed to examine the effects of nitric oxide (NO) and calcium ion/calmodulin-dependent nitric oxide synthase (NOS) on opioid-induced antinociception and their involvement in mediating the inhibitory effects of exposure to ELF magnetic fields. We observed that enkephalinase (SCH 34826)-induced, and likely enkephalin-mediated, antinociception in the land snail, Cepaea nemoralis, as measured by the enhanced latency of a foot withdrawal response to a thermal (40 degreesC) stimulus, was reduced by the NO releasing agent, S-nitro-N-acetylpenicillamide (SNP), and enhanced by the NO synthase inhibitor, NG-nitro-l-arginine methyl ester (l-NAME). Exposure of snails to an ELF magnetic field (15 min, 60 Hz, 141 microT peak) also reduced the enkephalinase-induced antinociception. The inhibitory effects of the 60-Hz magnetic field were significantly reduced by the NO synthase inhibitor, l-NAME, and significantly enhanced by the NO releasing agent, SNP, at dosages which by themselves had no evident effects on nociceptive sensitivity. These results suggest that: (1) NO and NO synthase have antagonistic effects on opioid-induced analgesia in the snail, Cepaea and (2) the inhibitory effects of ELF magnetic fields on opioid analgesia involve alteration in NO and NO synthase activity.

**(NE) (VT, AE) Kesari KK, Luukkonen J, Juutilainen J, Naarala J. Genomic instability induced by 50Hz magnetic fields is a dynamically evolving process not blocked by antioxidant treatment. Mutat Res Genet Toxicol Environ Mutagen. 794:46-51, 2015.**   
  
Increased level of micronuclei was observed in SH-SY5Y cells in a previous study at 8 and 15 days after exposure to extremely low frequency (ELF) magnetic fields (MF), indicating possible induction of genomic instability in the progeny of the exposed cells. The aim of this study was to further explore the induction of genomic instability by ELF MFs by increasing the follow-up time up to 45 days after exposure. Human SH-SY5Y neuroblastoma cells were exposed to a 50Hz, 100μT MF for 24h with or without co-exposure to menadione (MQ), a chemical agent that increases cellular superoxide production. Micronuclei, reactive oxygen species (ROS) and lipid peroxidation (LPO) were measured at 15, 30 and 45 days after exposure. To study the possible causal role of ROS in the delayed effects of MF, the antioxidant N-acetylcysteine (NAC) was administered before MF exposure. Consistently with the previous study, the level of micronuclei was statistically significantly elevated 15 days after exposure. A similar effect was observed at 30 days, but not at 45 days after exposure. The level of LPO was statically significantly decreased 30 and 45 days after exposure. Consistently with our previous findings, the MF effect did not depend on co-exposure to MQ. Treatment with NAC effectively decreased cellular ROS level and suppressed the effect of MQ on ROS, but it did not block the MF effect, indicating that increase in ROS is not needed as a causal link between MF exposure and induction of delayed effects. The results presented here are consistent with genomic instability that persists in the progeny of MF-exposed cells up to at least 30 days after exposure. Changes in LPO observed at 30 and 45 days after exposure indicates that the MF-initiated process may continue up to at least 45 days after exposure.

**(E) (VT, AE, IFR)** [**Kesari KK**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kesari%20KK%5BAuthor%5D&cauthor=true&cauthor_uid=26791000)**,** [**Juutilainen J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Juutilainen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=26791000)**,** [**Luukkonen J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Luukkonen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=26791000)**,** [**Naarala J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Naarala%20J%5BAuthor%5D&cauthor=true&cauthor_uid=26791000)**. Induction of micronuclei and superoxide production in neuroblastoma and glioma cell lines exposed to weak 50 Hz magnetic fields.** [**J R Soc Interface.**](http://www.ncbi.nlm.nih.gov/pubmed/26791000) **2016 Jan;13(114). pii: 20150995. doi: 10.1098/rsif.2015.0995.**

Extremely low-frequency (ELF) magnetic fields (MF) have been associated with adverse health effects in epidemiological studies. However, there is no known mechanism for biological effects of weak environmental MFs. Previous studies indicate MF effects on DNA integrity and reactive oxygen species, but such evidence is limited to MFs higher (greater than or equal to 100 µT) than those generally found in the environment. Effects of 10 and 30 µT fields were studied in SH-SY5Y and C6 cells exposed to 50-Hz MFs for 24 h. Based on earlier findings, menadione (MQ) was used as a cofactor. Responses to MF were observed in both cell lines, but the effects differed between the cell lines. Micronuclei were significantly increased in SH-SY5Y cells at 30 µT. This effect was largest at the highest MQ dose used. Increased cytosolic and mitochondrial superoxide levels were observed in C6 cells. The effects on superoxide levels were independent of MQ, enabling further mechanistic studies without co-exposure to MQ. The micronucleus and mitochondrial superoxide data were consistent with a conventional rising exposure-response relationship. For cytosolic superoxide, the effect size was unexpectedly large at 10 µT. The results indicate that the threshold for biological effects of ELF MFs is 10 µT or less.

**(E) (VT, AE, IFR)** [**Khadir R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Khadir%20R%22%5BAuthor%5D)**,** [**Morgan JL**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Morgan%20JL%22%5BAuthor%5D)**,** [**Murray JJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Murray%20JJ%22%5BAuthor%5D)**. Effects of 60 Hz magnetic field exposure on polymorphonuclear leukocyte activation.** [**Biochim Biophys Acta.**](javascript:AL_get(this,%20'jour',%20'Biochim%20%0d%0aBiophys%20Acta.');) **1472(1-2):359-367, 1999.**

We have investigated the effects of a sinusoidal 60 Hz magnetic field on free radical (superoxide anion) production, degranulation (beta-glucuronidase and lysozyme release) and viability in human neutrophils (PMNs). Experiments were performed blindly in very controlled conditions to examine the effects of a magnetic field in resting PMNs and in PMNs stimulated with a tumor promoter: phorbol 12-myristate 13-acetate (PMA). Exposure of unstimulated human PMNs to a 60 Hz magnetic field did not affect the functions examined. In contrast, exposure of PMNs to a 22 milliTesla (mT), 60 Hz magnetic field induced significant increases in superoxide anion (O2-) production (26.5%) and in beta-glucuronidase release (53%) when the cells were incubated with a suboptimal stimulating dose of PMA. Release of lysozyme and lactate dehydrogenase was unchanged by the magnetic field, whether the cells were stimulated or not. A 60 Hz magnetic field did not have any effect on O2- generation by a cell-free system xanthine/xanthine oxidase, suggesting that a magnetic field could upregulate common cellular events (signal transduction) leading to O2- generation and beta-glucuronidase release. In conclusion, exposure of PMNs to a 22 mT, 60 Hz magnetic field potentiates the effect of PMA on O2- generation and beta-glucuronidase release. This effect could be the result of an alteration in the intracellular signaling.

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In recent years, there has been a dramatic increase in the number and variety of electronic devices that emit electromagnetic waves. Because people live and work in close proximity to these pieces of electrical equipment, there is growing concern surrounding the destruction of homeostasis by electromagnetic field exposure. In the present study, the effects of 60 Hz 0.8 mT extremely low-frequency electromagnetic fields (ELF-EMF) on a macrophage cell line (RAW 264.7) were examined. Under defined ELF-EMF exposure conditions, the production of nitric oxide and pro-inflammatory cytokines, TNF-α, IL-1β, and IL-6, were increased in RAW 264.7 cells and the expression of those genes was also upregulated. However, cell proliferation was not altered. Translocation of NF-κB (nuclear factor kappa B), molecules that act downstream of the pro-inflammatory cytokines, were increased to the nucleus under ELF-EMF exposure conditions. In addition, we found that ELF-EMF exposure elevated activation of nuclear factor of activated T cells (NFAT) 2, as well as positively affected the influx of calcium. Furthermore, with both the presence of a potent antioxidant (Resveratrol) and downregulation of the antioxidant-related gene Prx-1 (Peroxiredoxin-1), ELF-EMF was associated with higher inflammatory responses of macrophages. These results suggest that an ELF-EMF amplifies inflammatory responses through enhanced macrophage activation and can decrease the effectiveness of antioxidants.

**(E)** **(VT, AE, IFR, AO)** [**Koh EK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Koh%20EK%22%5BAuthor%5D)**,** [**Ryu BK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ryu%20BK%22%5BAuthor%5D)**,** [**Jeong DY**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jeong%20DY%22%5BAuthor%5D)**,** [**Bang IS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bang%20IS%22%5BAuthor%5D)**,** [**Nam MH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nam%20MH%22%5BAuthor%5D)**,** [**Chae KS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Chae%20KS%22%5BAuthor%5D)**. A 60-Hz sinusoidal magnetic field induces apoptosis of prostate cancer cells through reactive oxygen species.** [**Int J Radiat Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aRadiat%20Biol.');) **84(11):945-955, 2008.**

PURPOSE: To explore the effects of power frequency magnetic fields (MF) on cell growth in prostate cancer, DU145, PC3, and LNCaP cells were examined in vitro. MATERIALS AND METHODS: The cells were exposed to various intensities and durations of 60-Hz sinusoidal MF in combination with various serum concentrations in the media. To analyze MF effects on cell growth, cell counting, trypan blue exclusion assay, Western blot analysis, flow cytometry, enzyme-linked immunosorbent assay (ELISA), semi-quantitative reverse transcriptase-polymerase chain reaction (RT-PCR), fluorescence microscopy, and spectrofluorometry were used. RESULTS: MF exposure induced significant cell growth inhibition and apoptosis in an intensity- and time-dependent manner, in which cell cycle arrest, cleaved Caspase-3, and reactive oxygen species (ROS) increased. Pretreatment with a Caspase-3 inhibitor or antioxidant, N-acetyl-L-cysteine (NAC), significantly attenuated MF-induced cell growth inhibition and cell death. Media replacement experiments failed to show any notable change in the MF effects. CONCLUSIONS: These results demonstrate 60-Hz sinusoidal MF-activated cell growth inhibition of prostate cancer in vitro. Apoptosis together with cell cycle arrest were the dominant causes of the MF-elicited cell growth inhibition, mediated by MF-induced ROS. These results suggest that a possibility of using 60-Hz MF in radiation therapy of prostate cancer could usefully be investigated.

**(E)** **(VT, AE, IX)** [**Koyama S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Koyama%20S%22%5BAuthor%5D)**,** [**Nakahara T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nakahara%20T%22%5BAuthor%5D)**,** [**Hirose H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hirose%20H%22%5BAuthor%5D)**,** [**Ding GR**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ding%20GR%22%5BAuthor%5D)**,** [**Takashima Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Takashima%20Y%22%5BAuthor%5D)**,** [**Isozumi Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Isozumi%20Y%22%5BAuthor%5D)**,** [**Miyakoshi J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Miyakoshi%20J%22%5BAuthor%5D)**. ELF electromagnetic fields increase hydrogen peroxide (H2O2)-induced mutations in pTN89 plasmids.** [**Mutat Res.**](javascript:AL_get(this,%20'jour',%20'Mutat%20%0d%0aRes.');) **560(1):27-32, 2004.**

We have examined the mutational effects of hydrogen peroxide (H(2)O(2)) in the presence and absence of an extremely low-frequency magnetic field (ELFMF), using pTN89 plasmids. Mutations were detected in the supF gene carried by these plasmids in Escherichia coli. The plasmids were either treated with H(2)O(2) (1microM) alone at 37 degrees C for 4h, or were exposed to an ELFMF (60Hz, 5 millitesla (mT)) simultaneously with H(2)O(2) treatment. The mutation frequency was 2.28 x 10(-4) for H(2)O(2) treatment alone, and 5.81 x 10(-4) for ELFMF exposure with H(2)O(2) treatment. We did not observe any mutations using treatment with ELFMF exposure alone. This indicates that the ELFMF may potentiate H(2)O(2)-induced mutation. Sequence analysis of the supF mutant plasmids revealed that base substitutions, G: C-->A :T transitions and G:C-->T:A transversions were dominant in both treatment groups, and there was no difference in the mutation spectrum or the hotspots between the groups. Therefore, ELFMFs may interact and potentiate the damage induced by H(2)O(2), resulting in an increase in the number of mutations.

**(E) (VT, AE, IX)** [**Koyama S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Koyama%20S%22%5BAuthor%5D)**,** [**Sakurai T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sakurai%20T%22%5BAuthor%5D)**,** [**Nakahara T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nakahara%20T%22%5BAuthor%5D)**,** [**Miyakoshi J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Miyakoshi%20J%22%5BAuthor%5D)**. Extremely low frequency (ELF) magnetic fields enhance chemically induced formation of apurinic/apyrimidinic (AP)sites in A172 cells.** [**Int J Radiat Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aRadiat%20Biol.');) **84(1):53-59, 2008.**

PURPOSE: To detect the effects of extremely low frequency (ELF) magnetic fields, the number of apurinic/apyrimidinic (AP) sites in human glioma A172 cells was measured following exposure to ELF magnetic fields. MATERIALS AND METHODS: The cells were exposed to an ELF magnetic field alone, to genotoxic agents (methyl methane sulfonate (MMS) and hydrogen peroxide (H2O2)) alone, or to an ELF magnetic field with the genotoxic agents. After exposure, DNA was extracted, and the number of AP sites was measured. RESULTS: There was no difference in the number of AP sites between cells exposed to an ELF magnetic field and sham controls. With MMS or H2O2 alone, the number of AP sites increased with longer treatment times. Exposure to an ELF magnetic field in combination with the genotoxic agents increased AP-site levels compared with the genotoxic agents alone. CONCLUSIONS: Our results suggest that the number of AP sites induced by MMS or H2O2 is enhanced by exposure to ELF magnetic fields at 5 millitesla (mT). This may occur because such exposure can enhance the activity or lengthen the lifetime of radical pairs.

**(E) (HU, CE, IFR) Kunt H, Şentürk İ, Gönül Y, Korkmaz M, Ahsen A, Hazman Ö, Bal A, Genç A, Songur A. Effects of electromagnetic radiation exposure on bone mineral density, thyroid, and oxidative stress index in electrical workers. OncoTargets and Therapy. 2016(9):745-754, 2016.**   
  
Background: In the literature, some articles report that the incidence of numerous diseases increases among the individuals who live around high-voltage electric transmission lines (HVETL) or are exposed vocationally. However, it was not investigated whether HVETL affect bone metabolism, oxidative stress, and the prevalence of thyroid nodule. Methods: Dual-energy X-ray absorptiometry (DEXA) bone density measurements, serum free triiodothyronine (FT3), free thyroxine (FT4), RANK, RANKL, osteoprotegerin (OPG), alkaline phosphatase (ALP), phosphor, total antioxidant status (TAS), total oxidant status (TOS), and oxidative stress index (OSI) levels were analyzed to investigate this effect. Results: Bone mineral density levels of L1–L4 vertebrae and femur were observed significantly lower in the electrical workers. ALP, phosphor, RANK, RANKL, TOS, OSI, and anteroposterior diameter of the left thyroid lobe levels were significantly higher, and OPG, TAS, and FT4 levels were detected significantly lower in the study group when compared with the control group. Conclusion: Consequently, it was observed that the balance between construction and destruction in the bone metabolism of the electrical workers who were employed in HVETL replaced toward destruction and led to a decrease in OPG levels and an increase in RANK and RANKL levels. In line with the previous studies, long-term exposure to an electromagnetic field causes disorders in many organs and systems. Thus, it is considered that long-term exposure to an electromagnetic field affects bone and thyroid metabolism and also increases OSI by increasing the TOS and decreasing the antioxidant status

**(E) (VT, CE, IX)** [**Kurzeja E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kurzeja%20E%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**,** [**Synowiec-Wojtarowicz A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Synowiec-Wojtarowicz%20A%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**,** [**Stec M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Stec%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**,** [**Glinka M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Glinka%20M%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**,** [**Gawron S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Gawron%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**,** [**Pawłowska-Góral K**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Paw%C5%82owska-G%C3%B3ral%20K%5BAuthor%5D&cauthor=true&cauthor_uid=23873295)**. Effect of a static magnetic fields and fluoride ions on the antioxidant defense system of mice fibroblasts.** [**Int J Mol Sci.**](http://www.ncbi.nlm.nih.gov/pubmed/23873295)**14(7):15017-15028, 2013.**

The results of studies on the biological influence of magnetic fields are controversial and do not provide clear answers regarding their impact on cell functioning. Fluoride compounds are substances that influence free radical processes, which occur when the reactive forms of oxygen are present. It is not known whether static magnetic fields (SMF) cause any changes in fluoride assimilation or activity. Therefore, the aim of this work was to determine the potential relationship between magnetic field exposure to, and the antioxidant system of, fibroblasts cultured with fluoride ions. Three chambers with static magnetic fields of different intensities (0.4, 0.6, and 0.7 T) were used in this work. Fluoride ions were added at a concentration of 0.12 mM, which did not cause the precipitation of calcium or magnesium. The results of this study show that static magnetic fields reduce the oxidative stress caused by fluoride ions and normalize the activities of antioxidant enzymes, including superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT). Static magnetic fields modify the energy state of fibroblasts, causing an increase in the ATP concentration and a decrease in the MDA concentration. These results suggest that exposure to fluoride and an SMF improves the tolerance of cells to the oxidative stress induced by fluoride ions.

**(E) (VO, CE, IFR, IOD, DAO)** [**Kuzay D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Kuzay%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28516790)**,** [**Ozer C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ozer%20C%5BAuthor%5D&cauthor=true&cauthor_uid=28516790)**,** [**Sirav B**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sirav%20B%5BAuthor%5D&cauthor=true&cauthor_uid=28516790)**,** [**Canseven AG**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Canseven%20AG%5BAuthor%5D&cauthor=true&cauthor_uid=28516790)**,** [**Seyhan N**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Seyhan%20N%5BAuthor%5D&cauthor=true&cauthor_uid=28516790)**. Oxidative effects of extremely low frequency magnetic field and radio frequency radiation on testes tissues of diabetic and healthy rats.** [**Bratisl Lek Listy.**](https://www.ncbi.nlm.nih.gov/pubmed/28516790) **118(5):278-282, 2017.**

With the development of technology, people are increasingly under the exposure of electromagnetic fields. Individuals with chronic diseases such as diabetes are now long-term exposed to Radio Frequency-RF radiation and extremely low frequency (ELF) magnetic fields (MFs). The purpose of this present study is to investigate oxidative effects and antioxidant parameters of ELF MFs and RF radiation on testis tissue in diabetic and healthy rats. Wistar male rats were divided into 10 groups. Intraperitoneal single dose STZ (65 mg/kg) dissolved in citrate buffer (0.1M (pH 4.5)) was injected to diabetes groups. ELF MFs and RF radiation were used as an electromagnetic exposure for 20 min/day, 5 days/week for one month. Testis tissue oxidant malondialdehyde (MDA), and antioxidants glutathione (GSH), and total nitric oxide (NOx) levels were determined. The results of ANOVA and Mann-Whitney tests were compared; p < 0.05 was considered significant. ELF and RF radiation resulted in an increase in testicular tissue MDA and NOX levels (p < 0.05), and caused a decrease in GSH levels (p < 0.05) in both healthy and diabetic rats, yet more distinctively in diabetic rats. The most pronounced effect was recorded in D-RF + ELF group (p < 0.005). Both radiation practices increased the oxidative stress in testis tissue while causing a decrease in antioxidant level which was more distinctive in diabetic rats (Tab. 1, Fig. 3, Ref. 30).

[**Lahbib A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lahbib%20A%5BAuthor%5D&cauthor=true&cauthor_uid=24899393)**,** [**Ghodbane S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ghodbane%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24899393)**,** [**Sakly M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Sakly%20M%5BAuthor%5D&cauthor=true&cauthor_uid=24899393)**,** [**Abdelmelek H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Abdelmelek%20H%5BAuthor%5D&cauthor=true&cauthor_uid=24899393)**. Vitamins and glucose metabolism: The role of static magnetic fields.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/24899393) **90(12):1240-1245, 2014. (review)**

PURPOSE: This review focuses on our own data and other data from the literature of static magnetic fields (SMF) bioeffects and vitamins and glucose metabolism. Three main areas of investigation have been covered: Static magnetic field and glucose metabolism, static magnetic field and vitamins and the role of vitamins on glucose metabolism. CONCLUSION: Considering these articles comprehensively, the conclusions are as follows: The primary cause of changes in cells after incubation in external SMF is disruption of free radical metabolism and elevation of their concentration. Such disruption causes oxidative stress leading to an unsteadiness of glucose level and insulin release. Moreover, based on available data, it was concluded that exposure to SMF alters plasma levels of vitamin A, C, D and E; these parameters can take part in disorder of glucose homeostasis and insulin release.

**(E)** **(VO, AE, AO)** [**Lai H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lai%20H%22%5BAuthor%5D)**,** [**Singh NP**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Singh%20NP%22%5BAuthor%5D)**. Melatonin and N-tert-butyl-alpha-phenylnitrone block 60-Hz magnetic field-induced DNA single and double strand breaks in rat brain cells.** [**J Pineal Res.**](javascript:AL_get(this,%20'jour',%20'J%20Pineal%20%0d%0aRes.');) **22(3):152-162, 1997.**

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-phenylnitrone (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.

**(E)** **(VO, AE, AO)** [**Lai H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lai%20H%22%5BAuthor%5D)**,** [**Singh NP**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Singh%20NP%22%5BAuthor%5D)**. Magnetic-field-induced DNA strand breaks in brain cells of the rat.** [**Environ Health Perspect.**](javascript:AL_get(this,%20'jour',%20'Environ%20%0d%0aHealth%20Perspect.');) **112(6):687-694, 2004.**

In previous research, we found that rats acutely (2 hr) exposed to a 60-Hz sinusoidal magnetic field at intensities of 0.1-0.5 millitesla (mT) showed increases in DNA single- and double-strand breaks in their brain cells. Further research showed that these effects could be blocked by pretreating the rats with the free radical scavengers melatonin and N-tert-butyl-alpha-phenylnitrone, suggesting the involvement of free radicals. In the present study, effects of magnetic field exposure on brain cell DNA in the rat were further investigated. Exposure to a 60-Hz magnetic field at 0.01 mT for 24 hr caused a significant increase in DNA single- and double-strand breaks. Prolonging the exposure to 48 hr caused a larger increase. This indicates that the effect is cumulative. In addition, treatment with Trolox (a vitamin E analog) or 7-nitroindazole (a nitric oxide synthase inhibitor) blocked magnetic-field-induced DNA strand breaks. These data further support a role of free radicals on the effects of magnetic fields. Treatment with the iron chelator deferiprone also blocked the effects of magnetic fields on brain cell DNA, suggesting the involvement of iron. Acute magnetic field exposure increased apoptosis and necrosis of brain cells in the rat. We hypothesize that exposure to a 60-Hz magnetic field initiates an iron-mediated process (e.g., the Fenton reaction) that increases free radical formation in brain cells, leading to DNA strand breaks and cell death. This hypothesis could have an important implication for the possible health effects associated with exposure to extremely low-frequency magnetic fields in the public and occupational environments.

**(E) (VO, AE, IOD, IAO)** [**Lee BC**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lee%20BC%22%5BAuthor%5D)**,** [**Johng HM**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Johng%20HM%22%5BAuthor%5D)**,** [**Lim JK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lim%20JK%22%5BAuthor%5D)**,** [**Jeong JH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Jeong%20JH%22%5BAuthor%5D)**,** [**Baik KY**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Baik%20KY%22%5BAuthor%5D)**,** [**Nam TJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Nam%20TJ%22%5BAuthor%5D)**,** [**Lee JH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Lee%20JH%22%5BAuthor%5D)**,** [**Kim J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kim%20J%22%5BAuthor%5D)**,** [**Sohn UD**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sohn%20UD%22%5BAuthor%5D)**,** [**Yoon G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yoon%20G%22%5BAuthor%5D)**,** [**Shin S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Shin%20S%22%5BAuthor%5D)**,** [**Soh KS**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Soh%20KS%22%5BAuthor%5D)**. Effects of extremely low frequency magnetic field on the antioxidant defense system in mouse brain: a chemiluminescence study.** [**J Photochem Photobiol B.**](javascript:AL_get(this,%20'jour',%20'J%20%0d%0aPhotochem%20Photobiol%20B.');) **73(1-2):43-48, 2004.**

Among the putative mechanisms, by which extremely low frequency (ELF) magnetic field (MF) may affect biological systems is that of increasing free radical life span in organisms. To test this hypothesis, we investigated whether ELF (60 Hz) MF can modulate antioxidant system in mouse brain by detecting chemiluminescence and measuring superoxide dismutase (SOD) activity in homogenates of the organ. Compared to sham exposed control group, lucigenin-initiated chemiluminescence in exposed group was not significantly increased. However, lucigenin-amplified t-butyl hydroperoxide (TBHP)-initiated brain homogenates chemiluminescence, was significantly increased in mouse exposed to 60 Hz, MF, 12 G for 3 h compared to sham exposed group. We also measured SOD activity, that plays a critical role of the antioxidant defensive system in brain. In the group exposed to 60 Hz, MF, 12 G for 3 h, brain SOD activity was significantly increased. These results suggest that 60 Hz, MF could deteriorate antioxidant defensive system by reactive oxygen species (ROS), other than superoxide radicals. Further studies are needed to identify the kind of ROS generated by the exposure to 60 Hz, MF and elucidate how MF can affect biological system in connection with oxidative stress.

**(NE) (VT, AE)** [**Lee HJ**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20HJ%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Jin YB**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jin%20YB%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Choi JI**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Choi%20JI%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Lee JW**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20JW%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=21898471)**. Combined effects of 60 Hz electromagnetic field exposure with various stress factors on cellular transformation in NIH3T3 cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/21898471) **33(3):207-214, 2012**.

Epidemiological studies have suggested that extremely low-frequency magnetic fields (ELF-MF) are associated with an increased incidence of cancer. Studies using in vitro systems have reported mixed results for the effects of ELF-MF alone, and the World Health Organization (WHO) Research Agenda published in 2007 suggested that high priority research should include an evaluation of the co-carcinogenic effects of ELF-MF exposure using in vitro models. Here, the carcinogenic potential of ELF-MF exposure alone and in combination with various stress factors was investigated in NIH3T3 mouse fibroblasts using an in vitro cellular transformation assay. NIH3T3 cells were exposed to a 60 Hz ELF-MF (1 mT) alone or in combination with ionizing radiation (IR), hydrogen peroxide (H₂O₂), or c-Myc overexpression, and the resulting number of anchorage-independent colonies was counted. A 4 h exposure of NIH3T3 cells to ELF-MF alone produced no cell transformation. Moreover, ELF exposure did not influence the transformation activity of IR, H₂O₂, or activated c-Myc in our in vitro assay system, suggesting that 1 mT ELF-MF did not affect any additive or synergistic transformation activities in combination with stress factors such as IR, H₂O₂, or activated c-Myc in NIH3T3 cells.

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PURPOSE: The purpose of this study is to investigate the mechanism of cellular proliferation of electromagnetic field (EMF) on human intervertebral disc (IVD) cells. MATERIALS AND METHODS: Human IVD cells were cultured three-dimensionally in alginate beads. EMF was exposed to IVD cells with 650 Ω, 1.8 millitesla magnetic flux density, 60 Hz sinusoidal wave. Cultures were divided into a control and EMF group. Cytotoxicity, DNA synthesis and proteoglycan synthesis were measured by MTT assay, [(3)H]-thymidine, and [(35)S]-sulfate incorporation. To detect phenotypical expression, reverse transcription-polymerase chain reactions (RT-PCR) were performed for aggrecan, collagen type I, and type II mRNA expression. To assess action mechanism of EMF, IVD cells were exposed to EMF with N(G)-Monomethyl-L-arginine (NMMA) and acetylsalicylic acid (ASA). RESULTS: There was no cytotoxicity in IVD cells with the EMF group in MTT assay. Cellular proliferation was observed in the EMF group (p < 0.05). There was no difference in newly synthesized proteoglycan normalized by DNA synthesis between the EMF group and the control. Cultures with EMF showed no significant change in the expression of aggrecan, type I, and type II collagen mRNA compared to the control group. Cultures with NMMA (blocker of nitric oxide) or ASA (blocker of prostaglandin E2) exposed to EMF demonstrated decreased DNA synthesis compared to control cultures without NMMA or ASA (p < 0.05). CONCLUSION: EMF stimulated DNA synthesis in human IVD cells while no significant effect on proteoglycan synthesis and chondrogenic phenotype expressions. DNA synthesis was partially mediated by nitric oxide and prostaglandin E2. EMF can be utilized to stimulate proliferation of IVD cells, which may provide efficient cell amplification in cell therapy to degenerative disc disease.

**Lewczuk B, Redlarski G, Zak A, Ziółkowska N, Przybylska-Gornowicz B, Krawczuk M. Influence of electric, magnetic, and electromagnetic fields on the circadian system: current stage of knowledge. Biomed Res Int. 2014:169459. Epub 2014 Jul 22. (review)**

One of the side effects of each electrical device work is the electromagnetic field generated near its workplace. All organisms, including humans, are exposed daily to the influence of different types of this field, characterized by various physical parameters. Therefore, it is important to accurately determine the effects of an electromagnetic field on the physiological and pathological processes occurring in cells, tissues, and organs. Numerous epidemiological and experimental data suggest that the extremely low frequency magnetic field generated by electrical transmission lines and electrically powered devices and the high frequencies electromagnetic radiation emitted by electronic devices have a potentially negative impact on the circadian system. On the other hand, several studies have found no influence of these fields on chronobiological parameters. According to the current state of knowledge, some previously proposed hypotheses, including one concerning the key role of melatonin secretion disruption in pathogenesis of electromagnetic field induced diseases, need to be revised. This paper reviews the data on the effect of electric, magnetic, and electromagnetic fields on melatonin and cortisol rhythms-two major markers of the circadian system as well as on sleep. It also provides the basic information about the nature, classification, parameters, and sources of these fields.

**(E)** **(VT, AE, IOD, IAO)** [**Lewicka M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lewicka%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**,** [**Henrykowska GA**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Henrykowska%20GA%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**,** [**Pacholski K**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pacholski%20K%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**,** [**Szczęsny A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Szcz%C4%99sny%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**,** [**Dziedziczak-Buczyńska M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Dziedziczak-Buczy%C5%84ska%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**,** [**Buczyński A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Buczy%C5%84ski%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25923084)**. The impact of electromagnetic radiation of different parameters on platelet oxygen metabolism - in vitro studies.** [**Adv Clin Exp Med.**](http://www.ncbi.nlm.nih.gov/pubmed/25923084) **24(1):31-35, 2015.**

BACKGROUND: Electromagnetic radiation emitted by a variety of devices, e.g. cell phones, computers and microwaves, interacts with the human body in many ways. Research studies carried out in the last few decades have not yet resolved the issue of the effect of this factor on the human body and many questions are left without an unequivocal answer. Various biological and health-related effects have not been fully recognized. Thus further studies in this area are justified. OBJECTIVES: A comparison of changes within catalase enzymatic activity and malondialdehyde concentration arising under the influence of the electromagnetic radiation emitted by car electronics, equipment used in physiotherapy and LCD monitors. MATERIAL AND METHODS: The suspension of human blood platelets at a concentration of 1 × 109/0.001 dm 3, obtained from whole blood by manual apheresis, was the study material. Blood platelets were exposed to an electromagnetic field for 30 min in a laboratory stand designed for the reconstruction of the electromagnetic radiation generated by car electronics, physiotherapy equipment and LCD monitors. The changes in catalase activity and malondialdehyde concentration were investigated after the exposure and compared to the control values (unexposed material). RESULTS: An increase in catalase activity and malondialdehyde concentration was observed after 30 min exposure of platelets to EMF regardless of the radiation source. The most significant changes determining the degree of oxidative stress were observed after exposure to the EMF generated by car electronics. CONCLUSIONS: The low frequency electromagnetic fields generated by car electronics, physiotherapy equipment and LCD monitors may be a cause of oxidative stress in the human body and may lead to free radical diseases.

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PURPOSE: To investigate whether extremely low frequency electromagnetic field (ELF-EMF) exposure could induce oxidative stress in workers performing tour-inspection near transformers and distribution power lines. MATERIALS AND METHODS: Occupational short-term 'spot' measurements were performed. In total, 310 inspection workers exposed to ELF-EMF were selected as the exposure group and 300 logistical staff as the control group. Plasma total antioxidant capacity (T-AOC) and glutathione peroxidase (GPx) activity were tested by the colorimetric method. Superoxide dismutase (SOD) activity was tested using the xanthine oxidase method. Plasma malondialdehyde (MDA) concentration was determined with a thiobarbituric acid assay. The micronucleus cell frequency (MCF) and Micronuclei frequency (MN) were also tested for genotoxic assessment. RESULTS: No significant changes of enzyme activities or MDA concentration were found. Neither the frequency of micronucleus lymphocytes nor micronuclei frequency changes were statistically significant. CONCLUSION: Continual ELF-EMF exposure might not induce oxidative stress in workers from a power supply bureau.

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Extremely low frequency electromagnetic field (ELF-EMF) exposure is attracting increased attention as a possible disease-inducing factor. The in vivo effects of short-term and long-term ELF-EMF exposure on male Drosophila melanogaster were studied using transcriptomic analysis for preliminary screening and QRT-PCR for further verification. Transcriptomic analysis indicated that 439 genes were up-regulated and 874 genes were down-regulated following short-term exposures and that 514 genes were up-regulated and 1206 genes were down-regulated following long-term exposures (expression >2- or <0.5-fold, respectively). In addition, there are 238 up-regulated genes and 598 down-regulated genes in the intersection of short-term and long-term exposure (expression >2- or <0.5-fold). The DEGs (differentially expressed genes) in D. melanogaster following short-term exposures were involved in metabolic processes, cytoskeletal organization, mitotic spindle organization, cell death, protein modification and proteolysis. Long-term exposure let to changes in expression of genes involved in metabolic processes, response to stress, mitotic spindle organization, aging, cell death and cellular respiration. In the intersection of short-term and long-term exposure, a series of DEGs were related to apoptosis, aging, immunological stress and reproduction. To check the ELF-EMF effects on reproduction, some experiments on male reproduction ability were performed. Their results indicated that short-term ELF-EMF exposure may decrease the reproductive ability of males, but long-term exposures had no effect on reproductive ability. Down-regulation of ark gene in the exposed males suggests that the decrease in reproductive capacity may be induced by the effects of ELF-EMF exposure on spermatogenesis through the caspase pathway. QRT-PCR analysis confirmed that jra, ark and decay genes were down regulated in males exposed for 1 Generation (1G) and 72h, which suggests that apoptosis may be inhibited in vivo. ELF-EMF exposure may have accelerated cell senescence, as suggested by the down-regulation of both cat and jra genes and the up-regulation of hsp22 gene. Up-regulation of totA and hsp22 genes during exposure suggests that exposed flies might induce an in vivo immune response to counter the adverse effects encountered during ELF-EMF exposure. Down-regulation of cat genes suggests that the partial oxidative protection system might be restrained, especially during short-term exposures. This study demonstrates the bioeffects of ELF-EMF exposure and provides evidence for understanding the in vivo mechanisms of ELF-EMF exposure on male D. melanogaster.

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Although melatonin (MT) has been reported to protect cells against oxidative damage induced by electromagnetic radiation, few reports have addressed whether there are other protective mechanisms. Here, we investigated the effects of MT on extremely low-frequency electromagnetic field (ELF-EMF)-induced Nav activity in rat cerebellar granule cells (GCs). Exposing cerebellar GCs to ELF-EMF for 60 min. significantly increased the Nav current (INa ) densities by 62.5%. MT (5 μM) inhibited the ELF-EMF-induced INa increase. This inhibitory effect of MT is mimicked by an MT2 receptor agonist and was eliminated by an MT2 receptor antagonist. The Nav channel steady-state activation curve was significantly shifted towards hyperpolarization by ELF-EMF stimulation but remained unchanged by MT in cerebellar GC that were either exposed or not exposed to ELF-EMF. ELF-EMF exposure significantly increased the intracellular levels of phosphorylated PKA in cerebellar GCs, and both MT and IIK-7 did not reduce the ELF-EMF-induced increase in phosphorylated PKA. The inhibitory effects of MT on ELF-EMF-induced Nav activity was greatly reduced by the calmodulin inhibitor KN93. Calcium imaging showed that MT did not increase the basal intracellular Ca2+ level, but it significantly elevated the intracellular Ca2+ level evoked by the high K+ stimulation in cerebellar GC that were either exposed or not exposed to ELF-EMF. In the presence of ruthenium red, a ryanodine-sensitive receptor blocker, the MT-induced increase in intracellular calcium levels was reduced. Our data show for the first time that MT protects against neuronal INa that result from ELF-EMF exposure through Ca2+ influx-induced Ca2+ release.

**(E)** **(VO, CE, IOD, DAO)** [**Liu Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Liu%20Y%22%5BAuthor%5D)**,** [**Weng E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Weng%20E%22%5BAuthor%5D)**,** [**Zhang Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zhang%20Y%22%5BAuthor%5D)**,** [**Hong R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hong%20R%22%5BAuthor%5D)**. [Effects of extremely low frequency electromagnetic field and its combination with lead on the antioxidant system in mouse]** [**Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi.**](javascript:AL_get(this,%20'jour',%20'Zhonghua%20%0d%0aLao%20Dong%20Wei%20Sheng%20Zhi%20Ye%20Bing%20Za%20Zhi.');) **20(4):263-265, 2002. [Article in Chinese]**

OBJECTIVE: To study the effects of extremely low frequency electromagnetic field(ELF EMF) and its combination with lead on the antioxidant system in mouse brain and liver tissues. METHOD: Mice were exposed to a 50 Hz sinusoidal 0.2 mT or 6.0 mT EMF for 2 weeks. At the same time, some groups were exposed to lead(50 mg/kg). After the exposure, the antioxidant system and cell membrane fluidity in brain and liver were measured. RESULTS: Malondiadehyde(MDA) content in brain and liver increased from the control levels of (1.33 +/- 0.12) and (3.95 +/- 0.21) nmol/mg pro to (1.35 +/- 0.09) and (6.15 +/- 0.28) nmol/mg pro respectively following 0.2 mT exposure, and to (3.98 +/- 0.10) and (6.50 +/- 0.79) nmol/mg pro respectively following 6.0 mT exposure. Total antioxidant capability(T-AOC) in brain and liver decreased from the control levels of (4.39 +/- 0.48) and (2.45 +/- 0.21) U/mg pro to (3.99 +/- 0.39) and (1.92 +/- 0.32) U/mg pro respectively following 0.2 mT, and to (3.12 +/- 0.37) and (1.57 +/- 0.14) U/mg pro respectively following 6.0 mT. GSH content decreased only in liver tissue from the control level of (194.60 +/- 20.93) mg/g pro to (189.24 +/- 5.61) mg/g pro(0.2 mT) and (153.04 +/- 1.18) mg/g pro(6.0 mT). Cellular membrane fluidity decreased from the control levels of (1.396 +/- 0.040) and (2.899 +/- 0.552) to (1.224 +/- 0.190) and (1.894 +/- 0.0761) (0.2 mT), (1.159 +/- 0.179) and (1.516 +/- 0.204)(6.0 mT) respectively. Compared with single EMF exposure(6.0 mT), EMF combined with lead exposure induced remarkable increase in MDA, GSH content and T-AOC and decrease in cell membrane fluidity both in the brain and liver, and increase in SOD activity only in liver. CONCLUSION: ELF EMF might alter the metabolism of free radicals, decrease anti-oxidant capability and enhance lipid peroxidation. The combination of EMF with lead showed synergic effects on lipid peroxidation.

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With the increasing use of electromagnetic technology, the effects of extremely low-frequency electromagnetic fields (ELF-EMF) on biological systems, central neurotransmitter systems, and human health have attracted extensive attention worldwide. In this study, lotus seedpod procyanidins (LSPCs) were evaluated for their protective effects on ELF-EMF induced oxidative stress injury in mice. Sixty male ICR mice were used for the experiment. The mice were randomly divided into five equal groups. The control group did not receive LSPCs or ELF-EMF but orally received normal saline. The ELF-EMF group received ELF-EMF exposure plus normal saline orally. The other three groups received ELF-EMF exposure plus LSPCs orally (60, 90, or 120mg kg(-1).bw, respectively). Each group exposed to ELF-EMF at 8 mT, 4h day(-1) for 28 consecutive days after administration daily of LSPCs or normal saline to mice for 15 consecutive days with the exception of the control group. Thereafter, blood and cerebral cortex of the mice were analyzed for antioxidant indices, including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GSH-Px), glutathione reductase (GR), glutathione-S-transferase (GST) and malondialdehyde (MDA). LSPCs administration at different doses significantly inhibited oxidative stress damage of mice induced by ELF-EMF. LSPCs treatment augmented SOD, CAT, GSH-Px, GR and GST activity. Furthermore, administration significantly lowered MDA level in LSPCs treatment groups LSPCs. All results indicated LSPCs can effectively prevent oxidative stress injury induced by ELF-EMF exposure, which may be related to its ability of scavenging free radicals and stimulating antioxidant enzyme activity.

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The aim of this study was to investigate the mechanism of cell activation induced by extremely low frequency magnetic fields (ELF-MF) (50 Hz) in human cells. We examined the production of free radicals in human umbilical cord blood-derived monocytes and in human Mono Mac 6 cells. The release of superoxide radical anions was analyzed using nitroblue tetrazolium chloride and the total of reactive oxygen species (ROS) was detected using dihydrorhodamine 123. Our results show a significant increase of superoxide radical anion production up-to 1.4 fold as well as an increase in ROS release up-to 1.2 fold upon exposure of monocytes to 1 mT ELF-MF (45 min). Mono Mac 6 cells exhibit higher superoxide radical anion and ROS production up-to 1.4 and 1.5 fold, respectively. These results indicate that Mono Mac 6 cells are more sensitive to ELF-MF than monocytes. Using diphenyleneiodonium chloride (DPI) a specific inhibitor for the NADPH oxidase, the MF-effect was not inhibited in Mono Mac 6 cells. Therefore, we suggest that ELF-MF exposure induces the activation of NADH oxidase in these cells. However, the MF-effect was inhibited by DPI in monocytes, indicating the activation of the NADPH oxidase after exposure to ELF-MF.

**(E)** **(VT, AE, IFR)** [**Luukkonen J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Luukkonen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24374227)**,** [**Liimatainen A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Liimatainen%20A%5BAuthor%5D&cauthor=true&cauthor_uid=24374227)**,** [**Juutilainen J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Juutilainen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24374227)**,** [**Naarala J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Naarala%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24374227)**. Induction of genomic instability, oxidative processes, and mitochondrial activity by 50Hz magnetic fields in human SH-SY5Y neuroblastoma cells.** [**Mutat Res.**](http://www.ncbi.nlm.nih.gov/pubmed/24374227) **760:33-41, 2014.**

Epidemiological studies have suggested that exposure to 50Hz magnetic fields (MF) increases the risk of childhood leukemia, but there is no mechanistic explanation for carcinogenic effects. In two previous studies we have observed that a 24-h pre-exposure to MF alters cellular responses to menadione-induced DNA damage. The aim of this study was to investigate the cellular changes that must occur already during the first 24h of exposure to MF, and to explore whether the MF-induced changes in DNA damage response can lead to genomic instability in the progeny of the exposed cells. In order to answer these questions, human SH-SY5Y neuroblastoma cells were exposed to a 50-Hz, 100-μT MF for 24h, followed by 3-h exposure to menadione. The main finding was that MF exposure was associated with increased level of micronuclei, used as an indicator of induced genomic instability, at 8 and 15d after the exposures. Other delayed effects in MF-exposed cells included increased mitochondrial activity at 8d, and increased reactive oxygen species (ROS) production and lipid peroxidation at 15d after the exposures. Oxidative processes (ROS production, reduced glutathione level, and mitochondrial superoxide level) were affected by MF immediately after the exposure. In conclusion, the present results suggest that MF exposure disturbs oxidative balance immediately after the exposure, which might explain our previous findings on MF altered cellular responses to menadione-induced DNA damage. Persistently elevated levels of micronuclei were found in the progeny of MF-exposed cells, indicating induction of genomic instability.

**(E) (VT, AE, MC)** [**Mahmoudinasab H**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mahmoudinasab%20H%5BAuthor%5D&cauthor=true&cauthor_uid=28097161)**,** [**Sanie-Jahromi F**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sanie-Jahromi%20F%5BAuthor%5D&cauthor=true&cauthor_uid=28097161)**,** [**Saadat M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Saadat%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28097161)**. Effects of extremely low-frequency electromagnetic field on expression levels of some antioxidant genes in human MCF-7 cells.** [**Mol Biol Res Commun.**](https://www.ncbi.nlm.nih.gov/pubmed/28097161) **5(2):77-85. 2016.**

In the past three decades, study on the biological effects of extremely low-frequency electromagnetic fields (ELF-EMFs) has been of interest to scientists. Although the exact mechanism of its effect is not fully understood, free radical processes has been proposed as a possible mechanism. This study was designed to evaluate the effect of 50-Hz EMFs on the mRNA levels of seven antioxidant genes (*CAT*, *SOD1*, *SOD2*, *GSTO1*, *GSTM3*, *MSGT1*, and *MSGT3*) in human MCF-7 cells. The EMF exposure patterns were: 1) 5 min field-on/5 min filed-off, 2) 15 min field-on/15 min field-off, 3) 30 min field-on continuously. In all three exposure conditions we tried to have total exposure time of 30 minutes. Control cultures were located in the exposure apparatus when the power was off. The experiments were done at two field intensities; 0.25 mT and 0.50 mT. The RNA extraction was done at two times; immediately post exposure and two hours post exposure. The mRNA levels were determined using quantitative real-time polymerase chain reaction. MTT assay for three exposure conditions in the two field intensities represented no cytotoxic effect on MCF-7 cells. Statistical comparison showed a significant difference between 0.25 mT and 0.50 mT intensities for "the 15 min field-on/15 min field-off condition" (Fisher's exact test, P=0.041), indicating that at 0.50 mT intensity field, the number of down-regulated and/or up-regulated genes increased compared with the other ones. However, there is no statistical significant difference between the field intensities for the two others EMF exposure conditions.

**(E) (VO, CE, IFR, IOD)** [**Manikonda PK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Manikonda%20PK%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Rajendra P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rajendra%20P%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Devendranath D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Devendranath%20D%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Gunasekaran B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Gunasekaran%20B%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Channakeshava**](http://www.ncbi.nlm.nih.gov/pubmed?term=Channakeshava%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Aradhya SR**](http://www.ncbi.nlm.nih.gov/pubmed?term=Aradhya%20SR%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Sashidhar RB**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sashidhar%20RB%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**,** [**Subramanyam C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Subramanyam%20C%5BAuthor%5D&cauthor=true&cauthor_uid=24334533)**. Extremely low frequency magnetic fields induce oxidative stress in rat brain.** [**Gen Physiol Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/24334533) **33(1):81-90, 2014.**

The present investigation was conducted to understand the influence of long-term exposure of rats to extremely low frequency magnetic fields (ELF-MF), focusing on oxidative stress (OS) on different regions of rat's brain. Male Wistar rats (21-day-old) were exposed to ELF-MF (50 Hz; 50 and 100 µT) for 90 days continuously; hippocampal, cerebellar and cortical regions from rats were analyzed for (i) reactive oxygen species (ROS), (ii) metabolites indicative of OS and (iii) antioxidant enzymes. In comparison to control group rats, the rats that were continuously exposed to ELF-MF caused OS and altered glutathione (GSH/GSSG) levels in dose-dependent manner in all the regions of the brain. Accumulation of ROS, lipid peroxidation end products and activity of superoxide dismutase in different regions was in the descending order of cerebellum < hippocampus < cortex. Decrement in GSH/GSSG levels and increment in glutathione peroxidase activity were in the descending order of hippocampus < cerebellum < cortex. The continuous exposure to ELF-MF caused OS in all the examined regions of brain more significantly at 100 µT than at 50 µT. Varied influences observed in different regions of the brain, as documented in this study, may contribute to altered metabolic patterns in its related regions of the central nervous system, leading to aberrant neuronal functions.

**(E)** **(VT AE, IFR, AO)** [**Mannerling AC**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mannerling%20AC%22%5BAuthor%5D)**,** [**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Simk%C3%B3%20M%22%5BAuthor%5D)**,** [**Mild KH**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mild%20KH%22%5BAuthor%5D)**,** [**Mattsson MO**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mattsson%20MO%22%5BAuthor%5D)**. Effects of 50-Hz magnetic field exposure on superoxide radical anion formation and HSP70 induction in human K562 cells.** [**Radiat Environ Biophys.**](javascript:AL_get(this,%20'jour',%20'Radiat%20%0d%0aEnviron%20Biophys.');) **49(4):731-741, 2010.**

Epidemiological studies suggest a correlation between exposure to low-level extremely low-frequency (ELF) magnetic fields (MF) and certain cancers and neurodegenerative diseases. Experimental studies have not provided any mechanism for such effects, although at flux density levels significantly higher than the ones encountered in epidemiological studies, radical homoeostasis and levels of stress response proteins can be affected. Here, we report on the influence of MF exposure (50-Hz sine wave; 1 h; 0.025-0.10 mT; vertical or horizontal MF exposure direction) on different cellular parameters (proliferation, cell cycle distribution, superoxide radical anion, and HSP70 protein levels) in the human leukaemia cell line K562. The positive control heat treatment (42 degrees C, 1 h) did not affect either cell proliferation or superoxide radical anion production but caused accumulation of cells in the G2 phase and increased the stress protein HSP70. MF exposure (0.10 mT, 1 h) did not affect either cell cycle kinetics or proliferation. Both vertical and horizontal MF exposures for 1 h caused significantly and transiently increased HSP70 levels (>twofold), at several flux densities, compared to sham controls and also compared to heat treatment. This exposure also increased (30-40%) the levels of the superoxide radical anion, comparable to the positive control PMA. Addition of free radical scavengers (melatonin or 1,10-phenantroline) inhibited the MF-induced increase in HSP70. In conclusion, an early response to ELF MF in K562 cells seems to be an increased amount of oxygen radicals, leading to HSP70 induction. Furthermore, the results suggest that there is a flux density threshold where 50-Hz MF exerts its effects on K562 cells, at or below 0.025 mT, and also that it is the MF, and not the induced electric field, which is the active parameter.

**(NE)** **(VT, AE)** [**Markkanen A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Markkanen%20A%22%5BAuthor%5D)**,** [**Naarala J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Naarala%20J%22%5BAuthor%5D)**,** [**Juutilainen J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Juutilainen%20J%22%5BAuthor%5D)**. A Study on the effects of 50 Hz magnetic fields on UV-induced radical reactions in murine fibroblasts.** [**J Radiat Res (Tokyo).**](http://www.ncbi.nlm.nih.gov/pubmed/20921828##) **51(5):609-613, 2010.**

The aim of this study was to test the hypothesis that the "radical pair mechanism" (magnetic field effect on recombination rate of radical pairs) explains our previous findings indicating that 50 Hz magnetic fields (MF) of about 100 µT modify biological responses to ultraviolet (UV) radiation. In the present study, the effects of 50 Hz MF on cellular oxidative processes induced by UV radiation were investigated. Murine L929 fibroblast cells were exposed to 50 Hz MF of 100 or 300 µT during a 1-h UV exposure or for 24 h before it. The decay kinetics of oxidative reactions were analysed by measuring ultraweak chemiluminescence (photon emissions) of the exposed cells by scintillation counter in the out-of-coincidence mode. No significant MF effects were found. The results do not support the hypothesis that 100-300 µT MF modify biological responses to UV radiation by causing an overall change in oxidative reactions at cellular level.

**(E) (VT, AE, AO) Martínez MA, Úbeda A, Moreno J, Trillo MÁ. Power frequency magnetic fields affect the p38 MAPK-mediated regulation of NB69 cell proliferation implication of free radicals. Int J Mol Sci. 2016 Apr 6;17(4). pii: E510.**  
  
The proliferative response of the neuroblastoma line NB69 to a 100 µT, 50 Hz magnetic field (MF) has been shown mediated by activation of the MAPK-ERK1/2 pathway. This work investigates the MF effect on the cell cycle of NB69, the participation of p38 and c-Jun N-terminal (JNK) kinases in the field-induced proliferative response and the potential involvement of reactive oxygen species (ROS) in the activation of the MAPK-ERK1/2 and -p38 signaling pathways. NB69 cultures were exposed to the 100 µT MF, either intermittently for 24, 42 or 63 h, or continuously for periods of 15 to 120 min, in the presence or absence of p38 or JNK inhibitors: SB203580 and SP600125, respectively. Antioxidant N-acetylcysteine (NAC) was used as ROS scavenger. Field exposure induced transient activation of p38, JNK and ERK1/2. The MF proliferative effect, which was mediated by changes in the cell cycle, was blocked by the p38 inhibitor, but not by the JNK inhibitor. NAC blocked the field effects on cell proliferation and p38 activation, but not those on ERK1/2 activation. The MF-induced proliferative effects are exerted through sequential upregulation of MAPK-p38 and -ERK1/2 activation, and they are likely mediated by a ROS-dependent activation of p38.

**(E) (VO, AE, DAO)** [**Martínez-Sámano J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mart%C3%ADnez-S%C3%A1mano%20J%5BAuthor%5D&cauthor=true&cauthor_uid=20701462)**,** [**Torres-Durán PV**](http://www.ncbi.nlm.nih.gov/pubmed?term=Torres-Dur%C3%A1n%20PV%5BAuthor%5D&cauthor=true&cauthor_uid=20701462)**,** [**Juárez-Oropeza MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ju%C3%A1rez-Oropeza%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=20701462)**,** [**Elías-Viñas D**](http://www.ncbi.nlm.nih.gov/pubmed?term=El%C3%ADas-Vi%C3%B1as%20D%5BAuthor%5D&cauthor=true&cauthor_uid=20701462)**,** [**Verdugo-Díaz L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Verdugo-D%C3%ADaz%20L%5BAuthor%5D&cauthor=true&cauthor_uid=20701462)**. Effects of acute electromagnetic field exposure and movement restraint on antioxidant system in liver, heart, kidney and plasma of Wistar rats: a preliminary report.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/20701462) **86(12):1088-1094, 2010.**

PURPOSE: The aim of the present study was to evaluate the early effects of acute (2 h) exposure to extremely low frequency electromagnetic fields (ELF-EMF), as well as movement restraint (MR) and the combination of both on the antioxidant systems in the plasma, liver, kidney, and heart of rats. MATERIALS AND METHODS: Twenty-four adult male Wistar rats were divided in two groups, restrained and unrestrained. The restrained animals were confined into an acrylic tube for 120 min. Half of the animals of each group were exposed to ELF-EMF (60 Hz, 2.4 mT) during the period of restriction. Immediately after treatment, reduced glutathione (GSH), catalase (CAT), superoxide dismutase (SOD), and thiobarbituric acid reactive substances (TBARS) were measured in tissues. RESULTS: GSH concentration was significantly lower in the heart of all experimental animals when compared to the control group; furthermore, the decrease was higher in the liver of restrained animals. SOD activity was lower in the plasma of restrained and EMF exposed animals compared to unrestrained rats. There were no significant differences in CAT activity and TBARS levels among all the experimental groups vs. the control group. CONCLUSION: Two hours of 60 Hz EMF exposure might immediately alter the metabolism of free radicals, decreasing SOD activity in plasma and GSH content in heart and kidney, but does not induce immediate lipid peroxidation. Oxidative stress induced by movement restraint was stronger than that produced by EMF.

**(E)** **(VO, AE, DAO)** [**Martínez-Sámano J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mart%C3%ADnez-S%C3%A1mano%20J%5BAuthor%5D&cauthor=true&cauthor_uid=22560984)**,** [**Torres-Durán PV**](http://www.ncbi.nlm.nih.gov/pubmed?term=Torres-Dur%C3%A1n%20PV%5BAuthor%5D&cauthor=true&cauthor_uid=22560984)**,** [**Juárez-Oropeza MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ju%C3%A1rez-Oropeza%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=22560984)**,** [**Verdugo-Díaz L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Verdugo-D%C3%ADaz%20L%5BAuthor%5D&cauthor=true&cauthor_uid=22560984)**. Effect of acute extremely low frequency electromagnetic field exposure on the antioxidant status and lipid levels in rat brain.** [**Arch Med Res.**](http://www.ncbi.nlm.nih.gov/pubmed/22560984) **43(3):183-189, 2012.**

BACKGROUND AND AIMS: It is generally accepted that electromagnetic fields (EMF) can exert biological effects; however, the mechanisms by which EMF elicits responses are still unknown. The present study was designed to assess the immediate effects of acute EMF exposure, movement restriction, and the combination of both on the antioxidant systems and lipid content in the whole brain of rat. METHODS: Thirty two male Wistar rats were arranged in four groups: control, EMF exposed, movement restrained (MR), and EMF + MR for 2 h. Rats were then sacrificed and their brains analyzed for superoxide dismutase and catalase activities, reduced glutathione, nitric oxide, total cholesterol, and triacylglycerol levels, as well as plasma corticosterone concentrations. RESULTS: Acute exposure to EMF induces reduction in catalase and superoxide dismutase activities, whereas the combination of EMF + MR also decreases both reduced glutathione and nitric oxide levels. Our results show that the acute exposure to EMF does not induce elevation of stress-hormone corticosterone but impairs the antioxidant status in rat brain. CONCLUSIONS: Plasma corticosterone concentration and antioxidant data indicate that the acute exposure to EMF appears to be a mild stressor that leads to some adaptive responses due to the activation of systems controlling the brain oxidative balance.

**(E)** **(VT, AE, AO)** [**Martino CF**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Martino%20CF%22%5BAuthor%5D)**. Static magnetic field sensitivity of endothelial cells.** [**Bioelectromagnetics**.](http://www.ncbi.nlm.nih.gov/pubmed/21433034##) **32(6):506-508, 2011.**

In this manuscript, data demonstrating the magnetic sensitivity of human umbilical vein endothelial cells (HUVECs) is presented. The effects of low level fields (LLF; 0.2-1 µT), 30 and 120 µT magnetic fields on the proliferation of endothelial cells were investigated. Primary HUVECs were cultured and exposed to the distinct magnetic conditions in the same incubator. Although cell numbers were slightly affected between 30 and 120 µT magnetic fields, reducing the magnetic field to low levels clearly inhibited proliferation. The rationale of introducing LLF is to elucidate a possible mechanism of interaction. Small differences of 30 µT reduce endothelial cell numbers significantly. The addition of free radical scavenger superoxide dismutase suppressed the enhanced proliferation caused by 120 µT static magnetic fields. It is proposed that the static magnetic field interacts with endothelial cells via a free radical mechanism.

**(E) (VT, AE, IFR, LI)** [**Martino CF**](http://www.ncbi.nlm.nih.gov/pubmed?term=Martino%20CF%5BAuthor%5D&cauthor=true&cauthor_uid=21887222)**,** [**Castello PR**](http://www.ncbi.nlm.nih.gov/pubmed?term=Castello%20PR%5BAuthor%5D&cauthor=true&cauthor_uid=21887222)**. Modulation of hydrogen peroxide production in cellular systems by low level magnetic fields.** [**PLoS One.**](http://www.ncbi.nlm.nih.gov/pubmed/21887222) **2011;6(8):e22753.**

Increased generation of reactive oxygen species (ROS) and an altered redox status have long been observed in cancer cells, suggesting that ROS might be involved in the development of these cells. However, recent studies suggest that inducing an excess of ROS in cancer cells can be exploited for therapeutic benefits. Cancer cells in advanced stage tumors frequently exhibit multiple genetic alterations and high oxidative stress, suggesting that it might be possible to preferentially modulate the development of these cells by controlling their ROS production. Low levels of ROS are also important for the development and survival of normal cells. In this manuscript, we present data on the influence of the suppression of the Earth's magnetic field (low level magnetic fields or LLF) which magnitudes range from 0.2 µT to 2 µT on the modulation of hydrogen peroxide (H(2)O(2)) in human fibrosarcoma cancer cell line HT1080, pancreatic AsPC-1 cancer cell line, and bovine pulmonary artery endothelial cells (PAEC) exposed to geomagnetic field (control; 45 µT-60 µT). Reduction of the Earth's magnetic field suppressed H(2)O(2) production in cancer cells and PAEC. The addition of catalase and superoxide dismutase (SOD) mimetic MnTBAP inhibited the magnetic field effect. Modulating ROS production by magnetic fields may open new venues of biomedical research and therapeutic strategies.

**(NE) (VT, AE)** [**Messiha HL**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Messiha%20HL%5BAuthor%5D&cauthor=true&cauthor_uid=25505136)**,** [**Wongnate T**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Wongnate%20T%5BAuthor%5D&cauthor=true&cauthor_uid=25505136)**,** [**Chaiyen P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Chaiyen%20P%5BAuthor%5D&cauthor=true&cauthor_uid=25505136)**,** [**Jones AR**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jones%20AR%5BAuthor%5D&cauthor=true&cauthor_uid=25505136)**,** [**Scrutton NS**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Scrutton%20NS%5BAuthor%5D&cauthor=true&cauthor_uid=25505136)**. Magnetic field effects as a result of the radical pair mechanism are unlikely in redox enzymes.** [**J R Soc Interface.**](http://www.ncbi.nlm.nih.gov/pubmed/25505136) **2015 Feb 6;12(103). pii: 20141155. doi: 10.1098/rsif.2014.1155.**

Environmental exposure to electromagnetic fields is potentially carcinogenic. The radical pair mechanism is considered the most feasible mechanism of interaction between weak magnetic fields encountered in our environment and biochemical systems. Radicals are abundant in biology, both as free radicals and reaction intermediates in enzyme mechanisms. The catalytic cycles of some flavin-dependent enzymes are either known or potentially involve radical pairs. Here, we have investigated the magnetic field sensitivity of a number of flavoenzymes with important cellular roles. We also investigated the magnetic field sensitivity of a model system involving stepwise reduction of a flavin analogue by a nicotinamide analogue-a reaction known to proceed via a radical pair. Under the experimental conditions used, magnetic field sensitivity was not observed in the reaction kinetics from stopped-flow measurements in any of the systems studied. Although widely implicated in radical pair chemistry, we conclude that thermally driven, flavoenzyme-catalysed reactions are unlikely to be influenced by exposure to external magnetic fields.

**(E) (VT, AE, IFR, IAO)** [**Miliša M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mili%C5%A1a%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**,** [**Đikić D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=%C4%90iki%C4%87%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**,** [**Mandić T**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mandi%C4%87%20T%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**,** [**Grozić D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Grozi%C4%87%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**,** [**Čolić I**](https://www.ncbi.nlm.nih.gov/pubmed/?term=%C4%8Coli%C4%87%20I%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**,** [**Ostojić A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ostoji%C4%87%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28452248)**. Response of aquatic protists to electric field exposure.** [**Int J Radiat Biol.**](https://www.ncbi.nlm.nih.gov/pubmed/28452248) **2017 Apr 28:1-35. doi: 10.1080/09553002.2017.1321809. [Epub ahead of print]**

PURPOSE: To test the effects of short term exposure of aquatic organisms to electric field (EF) with negligible magnetic component. MATERIALS AND METHODS: We built a plate capacitor that served as a source of EF of strengths that can be found in nature near transmission lines. We exposed two cultured protist species Euglena viridis and Paramecium caudatum to EFs for 24 hours and monitored their abundance, morphology, intracellular superoxide anion (by DHE), hydrogen peroxide by (H2DCF) and MDA contents, catalase (CAT) and superoxide dismutase (SOD) activity. RESULTS: We found that even short term exposure to low strength EF causes changes in population abundance, morphology and oxidative stress response in both species. As the EF strength increased, abundance of both species decreased. However, at weaker EFs fission rates were seemingly promoted. We noted decrease in size in both organisms in directions perpendicular to their fission planes correlated with EF strength. DHE and H2DCF fluorescence intensity and SOD activity were higher in organisms exposed to the stronger EFs. CONCLUSIONS: We suggest that the electric component of the field, rather than the magnetic, is the main cause of all the noted effects. As a result, aquatic organisms should be given greater importance in studies assessing the effects of EMFs in spite of attenuating effects of water to EF strengths.

**(E) (VT, AE, CE, IFR, IAO, DAO)** [**Morabito C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Morabito%20C%5BAuthor%5D&cauthor=true&cauthor_uid=21220925)**,** [**Guarnieri S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Guarnieri%20S%5BAuthor%5D&cauthor=true&cauthor_uid=21220925)**,** [**Fanò G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Fan%C3%B2%20G%5BAuthor%5D&cauthor=true&cauthor_uid=21220925)**,** [**Mariggiò MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mariggi%C3%B2%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=21220925)**. Effects of acute and chronic low frequency electromagnetic field exposure on PC12 cells during neuronal differentiation.** [**Cell Physiol Biochem.**](http://www.ncbi.nlm.nih.gov/pubmed/21220925) **26(6):947-958, 2010a.**

BACKGROUND/AIMS: The purpose of this study was to provide information about the in vitro neuritogenesis during cell exposure to extremely low frequency electromagnetic fields (ELF-EMFs) of different intensities and durations using pheochromocytoma-derived cell line (PC12 cells) as neuronal model. METHODS: Proliferative rates and neuritogenesis were tested by colorimetric assay and morphological analysis, respectively; reactive oxygen species (ROS) levels and intracellular Ca(2+) variations monitored using single cell videomicroscopy. RESULTS: The long-lasting ELF-EMF exposure (0.1-1.0 mT) did not appear to significantly affect the biological response (proliferation and neuritogenesis). However, during the acute ELF-EMF exposure (30 min), in undifferentiated PC12 cells, there were increased ROS levels and decreased catalase activity, that, conversely, resulted increased after chronic exposure (7 days) at 1.0 mT. Acute exposure (0.1-1.0 mT) affected the spontaneous intracellular Ca(2+) variations in undifferentiated cells, in which basal intracellular Ca(2+) resulted increased after chronic exposure. In addition acute exposure affected cell response to a depolarizing agent, while basal membrane potential was not changed. CONCLUSION: Even if further studies remain necessary to identify the ROS/intracellular Ca(2+)cross-talking pathway activated by ELF-EMF exposure, we support the hypothesis that ROS and Ca(2+) could be the cellular "primum movens" of the ELF-EMF induced effects on biological systems.

**(E) (VT, AE, IFR, IAO)** [**Morabito C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Morabito%20C%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**,** [**Rovetta F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rovetta%20F%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**,** [**Bizzarri M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bizzarri%20M%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**,** [**Mazzoleni G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mazzoleni%20G%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**,** [**Fanò G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Fan%C3%B2%20G%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**,** [**Mariggiò MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mariggi%C3%B2%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=20005945)**. Modulation of redox status and calcium handling by extremely low frequency electromagnetic fields in C2C12 muscle cells: A real-time, single-cell approach.** [**Free Radic Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/20005945) **48(4):579-589, 2010b.**

The biological effects of electric and magnetic fields, which are ubiquitous in modern society, remain poorly understood. Here, we applied a single-cell approach to study the effects of short-term exposure to extremely low frequency electromagnetic fields (ELF-EMFs) on muscle cell differentiation and function using C2C12 cells as an in vitro model of the skeletal muscle phenotype. Our focus was on markers of oxidative stress and calcium (Ca(2+)) handling, two interrelated cellular processes previously shown to be affected by such radiation in other cell models. Collectively, our data reveal that ELF-EMFs (1) induced reactive oxygen species production in myoblasts and myotubes with a concomitant decrease in mitochondrial membrane potential; (2) activated the cellular detoxification system, increasing catalase and glutathione peroxidase activities; and (3) altered intracellular Ca(2+)homeostasis, increasing the spontaneous activity of myotubes and enhancing cellular reactivity to a depolarizing agent (KCl) or an agonist (caffeine) of intracellular store Ca(2+)channels. In conclusion, our data support a possible link between exposure to ELF-EMFs and modification of the cellular redox state, which could, in turn, increase the level of intracellular Ca(2+)and thus modulate the metabolic activity of C2C12 cells.

**(E) (VT, AE, IFR)** [**Naarala J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Naarala%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**,** [**Kesari KK**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Kesari%20KK%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**,** [**McClure I**](https://www.ncbi.nlm.nih.gov/pubmed/?term=McClure%20I%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**,** [**Chavarriaga C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chavarriaga%20C%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**,** [**Juutilainen J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Juutilainen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**,** [**Martino CF**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Martino%20CF%5BAuthor%5D&cauthor=true&cauthor_uid=28497056)**. Direction-dependent effects of combined static and ELF magnetic fields on cell proliferation and superoxide radical production.** [**Biomed Res Int.**](https://www.ncbi.nlm.nih.gov/pubmed/28497056) **2017;2017:5675086. doi: 10.1155/2017/5675086. Epub 2017 Apr 12.**

Proliferation of human umbilical vein endothelial cells was stimulated by a nearly vertical 60 or 120 *μ*T static magnetic field (MF) in comparison to cells that were shielded against MFs. When the static field was combined with an extremely low frequency (ELF) MF (18 Hz, 30 *μ*T), proliferation was suppressed by a horizontal but not by a vertical ELF field. As these results suggested that the effects of an ELF MF depend on its direction in relation to the static MF, independent experiments were carried out to confirm such dependence using 50 Hz MFs and a different experimental model. Cytosolic superoxide level in rat glioma C6 cells exposed in the presence of a nearly vertical 33 *μ*T static MF was increased by a horizontal 50 Hz, 30 *μ*T MF, but not affected by a vertical 50 Hz MF. The results suggest that a weak ELF MF may interact with the static geomagnetic field in producing biological effects, but the effect depends on the relative directions of the static and ELF MFs.

**(NE) (VT, AE)**  [**Nakayama M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Nakayama%20M%5BAuthor%5D&cauthor=true&cauthor_uid=27430265)**,** [**Nakamura A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Nakamura%20A%5BAuthor%5D&cauthor=true&cauthor_uid=27430265)**,** [**Hondou T**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hondou%20T%5BAuthor%5D&cauthor=true&cauthor_uid=27430265)**,** [**Miyata H**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Miyata%20H%5BAuthor%5D&cauthor=true&cauthor_uid=27430265)**. Evaluation of cell viability, DNA single-strand breaks, and nitric oxide production in LPS-stimulated macrophage RAW264 exposed to a 50-Hz magnetic field.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/27430265) **2016 Jul 19:1-7. [Epub ahead of print]**

PURPOSE: Synergistic effects between cellular oxidative stress and magnetic fields may explain the adverse biological effects of 50/60 Hz magnetic fields. To determine whether this hypothesis holds in macrophage RAW264 cells, we measured DNA single-strand breaks (SSB), cell viability, and nitric oxide (NO) production in cells with or without exposure to 0.5-mT, 50-Hz magnetic fields for 24 h and with or without simultaneous stimulation via the bacterial endotoxin, lipopolysaccharide (LPS). MATERIALS AND METHODS: Macrophages stimulated with 10 ng/ml LPS for 1 h were exposed to or not exposed to a magnetic field and were then subjected to (1) the alkaline comet assay to measure SSBs, (2) trypan-blue exclusion assay for cell viability, and (3) measurements of NO for evaluation of oxidative stress. RESULTS: The 50-Hz magnetic field enhanced DNA SSB and decreased cell viability only in the LPS-stimulated macrophages in which NO production was greatly enhanced. The magnetic field alone did not alter NO production. CONCLUSION: Co-stimulation of the cell with LPS and a 50-Hz magnetic field promoted SSB and lowered cell viability, but these were not mediated by LPS-induced NO production.

**(E) (VT, AE, IFR)** [**Noda Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Noda%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=10927192)**,** [**Mori A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mori%20A%5BAuthor%5D&cauthor=true&cauthor_uid=10927192)**,** [**Liburdy RP**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Liburdy%20RP%5BAuthor%5D&cauthor=true&cauthor_uid=10927192)**,** [**Packer L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Packer%20L%5BAuthor%5D&cauthor=true&cauthor_uid=10927192)**. Pulsed magnetic fields enhance nitric oxide synthase activity in rat cerebellum.** [**Pathophysiology.**](https://www.ncbi.nlm.nih.gov/pubmed/10927192) **7(2):127-130, 2000.**

The effect of pulsed magnetic fields on nitric oxide synthase (NOS) activity in the rat brain was investigated. Sprague-Dawley rats (male, 200-250 g body weight) brain were dissected regionally, and the crude enzyme solutions were treated with pulsed DC, AC or static DC magnetic fields at 0 degrees C for 1 h. After exposure, NOS activity was measured as nitrite and nitrate levels generated from incubation with arginine, CaCl(2) and beta-nicotinamide adenine dinucleotide phosphate. Under these experimental conditions, neither AC nor static DC field treatment showed any significant change in NOS activity. A significant increase in NOS activity was observed in the cerebellum (111.2+/-2.0%, P<0.05, five separate experiments) for a 1 Gauss (0.1 mT) pulsed DC field. Under the same experimental condition, only a slight change or no effect was observed in the hippocampus, cortex, medulla oblongata, hypothalamus, striatum and midbrain. These studies suggest that pulsed magnetic fields result in a different effect on NOS activity in the cerebellum of the rats.

[**Okano H**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Okano%20H%5BAuthor%5D&cauthor=true&cauthor_uid=18508647)**. Effects of static magnetic fields in biology: role of free radicals.** [**Front Biosci.**](https://www.ncbi.nlm.nih.gov/pubmed/18508647) **13:6106-6125, 2008. (Review)**

Biological systems can respond to a wide range of static magnetic fields (SMF). Some of these responses seem to be mediated partly through free radical reactions. For example, in magnetic sense and navigation using the geomagnetic field, one of the most promising mechanisms for explaining magnetic compass is "a radical pair mechanism". Biological free radicals are most commonly oxygen or nitrogen based with an unpaired electron, leading to the terms "reactive oxygen species (ROS)" or "reactive nitrogen species (RNS)". When applying SMF to medical treatment, coupling SMF exposure with possible chemotherapy of cancers is a novel fascinating area that SMF could enhance agent-induced ROS production against tumors. In addition, one of the potent mechanisms of SMF effects on hemodynamics and blood pressure has sometimes been linked to nitric oxide pathway. However, health and environmental concerns have been raised because the SMF effects on oxidative stress leading to genetic mutation and apoptosis/necrosis have been found. It seems to take place from free radical generation.

**(E)** **(VT, AE, IAO)** [**Osera C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Osera%20C%22%5BAuthor%5D)**,** [**Fassina L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fassina%20L%22%5BAuthor%5D)**,** [**Amadio M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Amadio%20M%22%5BAuthor%5D)**,** [**Venturini L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Venturini%20L%22%5BAuthor%5D)**,** [**Buoso E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Buoso%20E%22%5BAuthor%5D)**,** [**Magenes G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Magenes%20G%22%5BAuthor%5D)**,** [**Govoni S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Govoni%20S%22%5BAuthor%5D)**,** [**Ricevuti G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ricevuti%20G%22%5BAuthor%5D)**,** [**Pascale A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pascale%20A%22%5BAuthor%5D)**. Cytoprotective response induced by electromagnetic stimulation on SH-SY5Y human neuroblastoma cell line.** [**Tissue Eng Part A.**](http://www.ncbi.nlm.nih.gov/pubmed/21615217##) **17(19-20):2573-2582, 2011.**

It is well known that physiological functions and pathological conditions of cells and tissues can be influenced not only by chemical molecules, but also by physical stimuli such as electromagnetic waves. In particular, epidemiological studies suggest possible associations between exposure to electromagnetic fields and an increased risk of tumors and neurodegenerative disorders, such as Alzheimer's disease. However, depending on the dose and on the length of treatment, the electromagnetic stimuli can be harmful or induce a cytoprotective cellular response, suggesting a possible application in medical therapy. In this study, under a tissue engineering viewpoint, we investigated the effects of an electromagnetic wave (magnetic field intensity, 2 mT; frequency, 75 Hz) on a neuronal cellular model characterized by the overexpression of the amyloid precursor protein (APP). After a prolonged electromagnetic treatment, lower mitochondrial activity and proliferation rate, resulting in a higher cellular quiescence, were observed. Focusing on the stress and oxidative pathways, we detected an overall increase of two fundamental proteins, the chaperone heat shock protein HSP70 and the free radical scavenger superoxide dismutase-1 enzyme (SOD-1). Interestingly, we found that the electromagnetic stimulation promotes the nonamyloidogenic processing of APP through an increased expression of the α-secretase ADAM10 and an enhanced release of the soluble neurotrophic factor sAPPα (a product of the ADAM10-mediated cleavage of APP). In conclusion, these findings suggest that the electromagnetic stimulus, if properly administered in terms of dose and timing, is able to induce a cytoprotective response in the cell. Moreover, these results suggest a possible use of this particular physical stimulation to improve the functional capability of the cells to face noxae.

**(E) (VT, AE, IAO, IX)** [**Osera C**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Osera%20C%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Amadio M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Amadio%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Falone S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Falone%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Fassina L**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Fassina%20L%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Magenes G**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Magenes%20G%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Amicarelli F**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Amicarelli%20F%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Ricevuti G**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ricevuti%20G%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Govoni S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Govoni%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**,** [**Pascale A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pascale%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25708841)**. Pre-exposure of neuroblastoma cell line to pulsed electromagnetic field prevents H2 O2 -induced ROS production by increasing MnSOD activity.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/25708841) **36(3):219-232, 2015.**

Electromagnetic fields (EMFs) have been linked to increased risk of cancers and neurodegenerative diseases; however, EMFs can also elicit positive effects on biological systems, and redox status seems crucially involved in EMF biological effects. This study aimed to assess whether a short and repeated pulsed EMF (PEMF) could trigger adaptive responses against an oxidative insult in a neuronal cellular model. We found that a 40 min overall (four times a week, 10 min each) pre-exposure to PEMF did not affect major physiological parameters and led to a significant increase of Mn-dependent superoxide dismutase activity in the human neuroblastoma SH-SY5Y cell line. In addition, we found PEMF-pre-exposed cells exhibited decreased reactive oxygen species production following a 30 min H2 O2 challenge, with respect to non pre-exposed cells. Our findings might provide new insights on the role played by short and repeated PEMF stimulations in the enhancement of cellular defenses against oxidative insults. Although studies in normal neuronal cells would be useful to further confirm our hypothesis, we suggest that specific PEMF treatments may have potential biological repercussions in diseases where oxidative stress is implicated.

**(E) (VO, AE, IOD, DAO) Pandir D, Sahingoz R.** **Magnetic field-induced oxidative stress and DNA damage in Mediterranean flour moth Ephestia kuehniella Zeller (Lepidoptera: Pyralidae) larvae. J Pest Sci 87(1): 79-87, 2014.**

Ephestia kuehniella Zeller (Lepidoptera: Pyralidae) is cosmopolitan pest of stored products. The effect of strong magnetic fields (MFs) on DNA damage and oxidative stress on larvae stage of E. kuehniella was assessed. Antioxidant enzyme systems, which include superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione S-transferase (GST), and malondialdehyde (MDA), the end product of lipid peroxidation as a result of strong MF intoxication that might occur in the larvae tissue, were evaluated. A simple technique of single-cell gel electrophoresis (DNA comet assay) enabled a quick detection of MF treatment on larvae. The larvae were exposed in a 1.4-Tesla (T) MF from a DC power supply at 50 Hz for different time periods (3, 6, 12, 24, 48, and 72 h). MFs caused increasing DNA damage and demonstrated using the comet assay with its parameters including tail DNA%, tail length and tail moment. DNA damage at increasing exposure times were significantly larger than the control group ( p < 0.05). These parameters were detected using BS 200 ProP with image analysis software. SOD, CAT, GPx, and GST activities decreased and MDA level increased in the MF-treated group in larvae tissue compared to control group for increasing exposure times at 1.4 T ( p < 0.05). In our investigation, we showed that MFs caused oxidative stress and proved to be DNA damage as revealed by the comet assay. MFs may be used to determine potential toxic effects as a control agent against E. kuehniella larvae

**(E) (VT, AE, IFR)** [**Park JE**](http://www.ncbi.nlm.nih.gov/pubmed?term=Park%20JE%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**,** [**Seo YK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Seo%20YK%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**,** [**Yoon HH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yoon%20HH%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**,** [**Kim CW**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kim%20CW%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**,** [**Park JK**](http://www.ncbi.nlm.nih.gov/pubmed?term=Park%20JK%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**,** [**Jeon S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jeon%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23411410)**. Electromagnetic fields induce neural differentiation of human bone marrow derived mesenchymal stem cells via ROS mediated EGFR activation.** [**Neurochem Int.**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Park+JE+and+elelctromagnetic+field) **62(4):418-424, 2013.**

Even though the inducing effect of electromagnetic fields (EMF) on the neural differentiation of human bone marrow mesenchymal stem cells (hBM-MSCs) is a distinctive, the underlying mechanism of differentiation remains unclear. To find out the signaling pathways involved in the neural differentiation of BM-MSCs by EMF, we examined the CREB phosphorylation and Akt or ERK activation as an upstream of CREB. In hBM-MSCs treated with ELF-EMF (50 Hz, 1 mT), the expression of neural markers such as NF-L, MAP2, and NeuroD1 increased at 6 days and phosphorylation of Akt and CREB but not ERK increased at 90 min in BM-MSCs. Moreover, EMF increased phosphorylation of epidermal growth factor receptor (EGFR) as an upstream receptor tyrosine kinase of PI3K/Akt at 90 min. It has been well documented that ELF-MF exposure may alter cellular processes by increasing intracellular reactive oxygen species (ROS) concentrations. Thus, we examined EMF-induced ROS production in BM-MSCs. Moreover, pretreatment with a ROS scavenger, N-acetylcystein, and an EGFR inhibitor, AG-1478, prevented the phosphorylation of EGFR and downstream molecules. These results suggest that EMF induce neural differentiation through activation of EGFR signaling and mild generation of ROS.

**(E) (VT, AE, IFR, DFR, IAO, DAO)** [**Patruno A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Amerio P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Amerio%20P%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Pesce M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pesce%20M%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Vianale G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Vianale%20G%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Di Luzio S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Di%20Luzio%20S%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Tulli A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Tulli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Franceschelli S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Franceschelli%20S%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Grilli A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Grilli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Muraro R**](http://www.ncbi.nlm.nih.gov/pubmed?term=Muraro%20R%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**,** [**Reale M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=19799606)**. Extremely low frequency electromagnetic fields modulate expression of inducible nitric oxide synthase, endothelial nitric oxide synthase and cyclooxygenase-2 in the human keratinocyte cell line HaCat: potential therapeutic effects in wound healing.** [**Br J Dermatol.**](http://www.ncbi.nlm.nih.gov/pubmed/19799606) **162(2):258-266, 2010.**

BACKGROUND: Extremely low frequency (ELF) electromagnetic fields (EMF) are known to produce a variety of biological effects. Clinical studies are ongoing using EMF in healing of bone fractures and skin wounds. However, little is known about the mechanisms of action of ELF-EMF. Several studies have demonstrated that expression and regulation of nitric oxide synthase (NOS) and cyclooxygenase-2 (COX-2) are vital for wound healing; however, no reports have demonstrated a direct action of ELF-EMF in the modulation of these inflammatory molecules in human keratinocytes. OBJECTIVES: The present study analysed the effect of ELF-EMF on the human keratinocyte cell line HaCaT in order to assess the mechanisms of action of ELF-EMF and to provide further support for their therapeutic use in wound healing. METHODS: Exposed HaCaT cells were compared with unexposed control cells. At different exposure times, expression of inducible NOS (iNOS), endothelial NOS (eNOS) and COX-2 was evaluated by Western blot analysis. Modulation of iNOS and eNOS was monitored by evaluation of NOS activities, production of nitric oxide (NO) and O(2)(-) and expression of activator protein 1 (AP-1). In addition, catalase activity and prostaglandin (PG) E(2) production were determined. Effects of ELF-EMF on cell growth and viability were monitored. RESULTS: The exposure of HaCaT cells to ELF-EMF increased iNOS and eNOS expression levels. These ELF-EMF-dependent increased expression levels were paralled by increased NOS activities, and increased NO production. In addition, higher levels of AP-1 expression as well as a higher cell proliferation rate were associated with ELF-EMF exposure. In contrast, ELF-EMF decreased COX-2 expression, PGE(2) production, catalase activity and O(2)(-) production. CONCLUSIONS: Mediators of inflammation, such as reactive nitrogen and PGE(2), and keratinocyte proliferation are critical for the tissue regenerative processes. The ability of ELF-EMF to upmodulate NOS activities, thus nitrogen intermediates, as well as cell proliferation, and to downregulate COX-2 expression and the downstream intermediate PGE(2), highlights the potential therapeutic role of ELF-EMF in wound healing processes.

**(E) (VT, AE, IAO, IFR)** [**Patruno A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Tabrez S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Tabrez%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Amerio P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Amerio%20P%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Pesce M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pesce%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Vianale G**](http://www.ncbi.nlm.nih.gov/pubmed?term=Vianale%20G%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Franceschelli S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Franceschelli%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Grilli A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Grilli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Kamal MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kamal%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**,** [**Reale M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22229327)**. Kinetic study on the effects of extremely low frequency electromagnetic field on catalase, cytochrome P450 and inducible nitric oxide synthase in human HaCaT and THP-1 cell lines.** [**CNS Neurol Disord Drug Targets.**](http://www.ncbi.nlm.nih.gov/pubmed/22229327) **10(8):936-944, 2011.**

Extremely low frequency electromagnetic fields (ELF-EMF) have been found to produce a variety of biological effects. These effects of ELF-EMF depend upon frequency, amplitude, and length of exposure, and are also related to intrinsic susceptibility and responsiveness of different cell types. Although the mechanism of this interaction is still obscure, ELF-EMF can influence cell proliferation, differentiation, cell cycle, apoptosis, DNA replication and protein expression. The aim of this study was to estimate various kinetic constants of catalase, cytochrome P450 and inducible nitric oxide synthase in response to ELF-EMF exposure in human HaCaT and THP-1 cell lines. In order to evaluate the effect of ELF-EMF on the modulation of cellular responses to an inflammatory stimulus, both cell lines were treated with lipopolysaccharide. To the best of our knowledge there is no available report on such type of kinetic study of selected enzymes in response to ELF-EMF in these cell lines. Therefore, the current study may reveal novel mechanism of ELFEMF biological interaction with the enzymological and hormonal systems of living organisms. These new insights may be important for ELF-EMF application particularly for wound healing, tissue regeneration, Parkinson's and Alzheimer's diseases.

**(E) (VT, AE, IFR, DAO)** [**Patruno A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Pesce M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pesce%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Marrone A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Marrone%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Speranza L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Speranza%20L%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Grilli A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Grilli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**De Lutiis MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=De%20Lutiis%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Felaco M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Felaco%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**,** [**Reale M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21928345)**. Activity of matrix metallo proteinases (MMPs) and the tissue inhibitor of MMP (TIMP)-1 in electromagnetic field-exposed THP-1 cells.** [**J Cell Physiol.**](http://www.ncbi.nlm.nih.gov/pubmed/21928345) **227(6):2767-2774, 2012.**

Matrix metalloproteinases (MMPs) and tissue inhibitors of MMPs (TIMPs) are the main determinants of tissue remodeling in both physiological and pathological processes. Metabolic processes, which generate oxidants and antioxidants can be influenced by environmental factors such as electromagnetic fields (EMF). We analyzed the effects of EMF on the activity and expression of MMPs in THP-1 cells. Cells were exposed to a 50 Hz, 1 mT EMF for 24 h and incubated with or without LPS. Our data indicate that THP-1 cells exposed to EMF causes a reduction of anti-oxidant enzyme activity and an enhancement of nitrogen intermediates involving the iNOS pathway. We then analyzed the role of nitration of TIMP-1 in increasing the activity of MMPs in EMF exposed cells. Molecular modeling tools were employed to identify the most plausible sites in the active conformation of TIMP-1; at least two protein sites, Y120 and Y38 and/or Y72 were identified. Reactive nitrogen species (RNS) may affect protein targets, such as TIMP-1, which are crucial for the regulation of MMP activities by oxidation of sulfydryl groups, or by nitration of tyrosine residues. These results may suggest a pathway connecting an imbalance of MMPs and their cognate inhibitor TIMP-1.

**(E) (VT, AE, IAO)** [**Patruno A**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**,** [**Tabrez S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Tabrez%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**,** [**Pesce M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Pesce%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**,** [**Shakil S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Shakil%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**,** [**Kamal MA**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kamal%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**,** [**Reale M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25498893)**. Effects of extremely low frequency electromagnetic field (ELF-EMF) on catalase, cytochrome P450 and nitric oxide synthase in erythro-leukemic cells.** [**Life Sci.**](http://www.ncbi.nlm.nih.gov/pubmed/25498893) **121:117-123, 2015.**

AIMS: Extremely low frequency electromagnetic fields (ELF-EMFs) are widely employed in electrical appliances and different equipment such as television sets, mobile phones, computers and microwaves. The molecular mechanism through which ELF-EMFs can influence cellular behavior is still unclear. A hypothesis is that ELF-EMFs could interfere with chemical reactions involving free radical production. Under physiologic conditions, cells maintain redox balance through production of ROS/RNS and antioxidant molecules. The altered balance between ROS generation and elimination plays a critical role in a variety of pathologic conditions including neurodegenerative diseases, aging and cancer. Actually, there is a disagreement as to whether there is a causal or coincidental relationship between ELF-EMF exposure and leukemia development. Increased ROS levels have been observed in several hematopoietic malignancies including acute and chronic myeloid leukemias. MAIN METHODS: In our study, the effect of ELF-EMF exposure on catalase, cytochrome P450 and inducible nitric oxide synthase activity and expression by Western blot analysis in myelogenous leukemia cell line K562 was evaluated. KEY FINDINGS: A significant modulation of iNOS, CAT and Cyt P450 protein expression was recorded as a result of ELF-EMF exposure in both phorbol 12-myristate 13-acetate (PMA)-stimulated and non-stimulated cell lines. Modulation in kinetic parameters of CAT, CYP-450 and iNOS enzymes in response to ELF-EMF indicates an interaction between the ELF-EMF and the enzymological system. SIGNIFICANCE: These new insights might be important in establishing a mechanistic framework at the molecular level within which the possible effects of ELF-EMF on health can be understood.

**(E)** **(VO, CE, IAO, IX)** [**Politański P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Polita%C5%84ski%20P%22%5BAuthor%5D)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Rajkowska%20E%22%5BAuthor%5D)**,** [**Pawlaczyk-Łuszczyńska M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pawlaczyk-%C5%81uszczy%C5%84ska%20M%22%5BAuthor%5D)**,** [**Dudarewicz A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Dudarewicz%20A%22%5BAuthor%5D)**,** [**Wiktorek-Smagur A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wiktorek-Smagur%20A%22%5BAuthor%5D)**,** [**Sliwińska-Kowalska M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sliwi%C5%84ska-Kowalska%20M%22%5BAuthor%5D)**,** [**Zmyślony M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zmy%C5%9Blony%20M%22%5BAuthor%5D)**. Static magnetic field affects oxidative stress in mouse cochlea.** [**Int J Occup Med Environ Health.**](http://www.ncbi.nlm.nih.gov/pubmed/21306983##) **23(4):377-384, 2010.**

OBJECTIVE: It has been shown that oxidative stress plays an important role in development of noise induced hearing loss. Since static magnetic fields (SMF) exposure may alter dynamics of oxidative processes in the tissue, the aim of the study was to assess the influence of SMF on noise-induced alteration in the cochlear level of reactive oxygen species (ROS) and hearing thresholds. MATERIALS AND METHODS: Auditory brainstem response (ABR), lipid peroxidation (LPO) levels, super-oxide dismutase (SOD) activity and catalase activity were assessed in the cochlea prior to, and at five time-points over two weeks following exposure of C57BL/6 mice to 8h, 119 dB SPL, 4 kHz octave band noise. RESULTS: The ABR indicated no permanent functional damage due to noise exposure either for the 4 kHz and 8 kHz SMF-exposed group or for animals not exposed to SMF. However, significant differences in LPO level, catalase and SOD activity between animals exposed to noise and SMF and those exposed to noise only were observed. CONCLUSIONS: The results suggest that SMF causes an increase in ROS level in the cochlea after noise exposure and, at the same time, it speeds up activation of antioxidative enzymes.

**(E)** **(VT, AE, DFR, IFR)** [**Poniedzialek B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Poniedzialek%20B%5BAuthor%5D&cauthor=true&cauthor_uid=23137127)**,** [**Rzymski P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rzymski%20P%5BAuthor%5D&cauthor=true&cauthor_uid=23137127)**,** [**Nawrocka-Bogusz H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Nawrocka-Bogusz%20H%5BAuthor%5D&cauthor=true&cauthor_uid=23137127)**,** [**Jaroszyk F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jaroszyk%20F%5BAuthor%5D&cauthor=true&cauthor_uid=23137127)**,** [**Wiktorowicz K**](http://www.ncbi.nlm.nih.gov/pubmed?term=Wiktorowicz%20K%5BAuthor%5D&cauthor=true&cauthor_uid=23137127)**. The effect of electromagnetic field on reactive oxygen species production in human neutrophils in vitro.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23137127) **32(3):333-341, 2013a.**

The present study was undertaken in order to determine the effect of low frequency electromagnetic field (EMF) on reactive oxygen species (ROS) production in human neutrophils in peripheral blood in vitro. We investigated how differently generated EMF and several levels of magnetic induction affect ROS production. To evaluate the level of ROS production, two fluorescent dyes were used: 2'7'-dichlorofluorscein-diacetate and dihydrorhodamine. Phorbol 12-myristate 13-acetate (PMA), known as strong stimulator of the respiratory burst, was also used. Alternating magnetic field was generated by means of Viofor JPS apparatus. Three different levels of magnetic induction have been analyzed (10, 40 and 60 μT). Fluorescence of dichlorofluorescein and 123 rhodamine was measured by flow cytometry. The experiments demonstrated that only EMF tuned to the calcium ion cyclotron resonance frequency was able to affect ROS production in neutrophils. Statistical analysis showed that this effect depended on magnetic induction value of applied EMF. Incubation in EMF inhibited cell activity slightly in unstimulated neutrophils, whereas the activity of PMA-stimulated neutrophils has increased after incubation in EMF.

**(E)** **(VT, AE, DFR, IFR)** [**Poniedziałek B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Poniedzia%C5%82ek%20B%5BAuthor%5D&cauthor=true&cauthor_uid=23631724)**,** [**Rzymski P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rzymski%20P%5BAuthor%5D&cauthor=true&cauthor_uid=23631724)**,** [**Karczewski J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Karczewski%20J%5BAuthor%5D&cauthor=true&cauthor_uid=23631724)**,** [**Jaroszyk F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jaroszyk%20F%5BAuthor%5D&cauthor=true&cauthor_uid=23631724)**,** [**Wiktorowicz K**](http://www.ncbi.nlm.nih.gov/pubmed?term=Wiktorowicz%20K%5BAuthor%5D&cauthor=true&cauthor_uid=23631724)**. Reactive oxygen species (ROS) production in human peripheral blood neutrophils exposed in vitro to static magnetic field.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/23631724) **32(4):560-568, 2013b.**

The aim of this study was to determine the effect of gradient static magnetic field (SMF) on reactive oxygen species (ROS) production in human neutrophils in peripheral blood in vitro. Blood samples collected from healthy individuals were incubated in an inhomogeneous SMF (in a south or north pole of the field) for 15, 30 or 45 minutes. The maximum value of induction (B max) amounted to ≈ 60 mT. To determine the strength of the ROS production, dihydrorhodamine (123DHR) as fluorophore and phorbol 12-myristate 13-acetate (PMA) as respiratory burst stimulator were used. 123DHR oxidation by ROS was measured by flow cytometry. The exposure of blood samples to SMF induced statistically significant changes in ROS production in unstimulated and PMA-stimulated neutrophils. The observed effects were highly correlated with the exposure time and depended on the orientation of the field. Although intracellular mechanisms underlying such interactions are not thoroughly understood, it could be presumed that SMF affects ROS metabolic oscillations and their formation and inactivation. This study emphasizes the importance of proper adjustment of exposure time to SMF for any potential therapeutic applications.

**(E) (VT, AE, IFR) Pooam M, Nakayama M, Nishigaki C, Miyata H. Effect of 50-Hz sinusoidal magnetic field on the production of superoxide anion and the expression of heat-shock protein 70 in RAW264 cells. Int J Chem 9:23-36, 2017.**

There is a growing concern if the power-line frequency (50/60 Hz) magnetic field (termed in this paper ELF-MF) increases cancer risks. Since one of the major causes of cancer is cellular oxidative stress, whether the ELF-MF increases the oxidative stress is a central problem in the studies on the biological effect of the ELF-MF. Here, we have investigated the effect of 50-Hz sinusoidal magnetic field on the production of O2-, the expression of heat shock protein (HSP) 70 and the mitochondrial membrane potential in cell line macrophage RAW264 cells. Macrophages were exposed to or not exposed to 0.1-mT or 0.5-mT, 50-Hz sinusoidal magnetic field and were subjected to (1) assay for O2- (2) analysis of the expression of HSP70, and (3) measurement of the mitochondrial membrane potential with a fluorescent indicator. The 50-Hz magnetic field enhanced production of O2- and the expression of HSP70, both of which are consistent with previous studies. The exposure to 50-Hz magnetic field decreased mitochondrial membrane potential indicating the diminished activity of mitochondria. The uncoupler of mitochondrial function, carbonyl cyanide p-trifluoromethoxyphenylhydrazone diminished the membrane potential, as expected. On the other hand, it increased the production of O2-. The results collectively suggest that the 50-Hz magnetic field diminished the mitochondrial membrane potential, which led to the increase in the production of O2- and the expression of HSP70 protein.

**(E)** **(VT AE, IFR)** [**Potenza L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Potenza%20L%22%5BAuthor%5D)**,** [**Martinelli C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Martinelli%20C%22%5BAuthor%5D)**,** [**Polidori E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Polidori%20E%22%5BAuthor%5D)**,** [**Zeppa S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zeppa%20S%22%5BAuthor%5D)**,** [**Calcabrini C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Calcabrini%20C%22%5BAuthor%5D)**,** [**Stocchi L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Stocchi%20L%22%5BAuthor%5D)**,** [**Sestili P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sestili%20P%22%5BAuthor%5D)**,** [**Stocchi V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Stocchi%20V%22%5BAuthor%5D)**. Effects of a 300 mT static magnetic field on human umbilical vein endothelial cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/20623760##) **31(8):630-639, 2010.**

This study describes the effects of a static magnetic field (SMF) on cell growth and DNA integrity of human umbilical vein endothelial cells (HUVECs). Fast halo assay was used to investigate nuclear damage; quantitative polymerase chain reaction (QPCR), standard PCR, and real-time PCR were used to evaluate mitochondrial DNA integrity, content, and gene expression. HUVECs were continually exposed to a 300 mT SMF for 4, 24, 48, and 72 h. Compared to control samples (unexposed cultures) the SMF-exposed cells did not show a statistically significant change in their viability. Conversely, the static field was shown to be significant after 4 h of exposure, inducing damage on both the nuclear and mitochondrial levels, reducing mitochondrial content and increasing reactive oxygen species. Twenty-four hours of exposure increased mitochondrial DNA content as well as expression of one of the main genes related to mitochondrial biogenesis. No significant differences between exposed and sham cultures were found after 48 and 72 h of exposure. The results suggest that a 300 mT SMF does not cause permanent DNA damage in HUVECs and stimulates a transient mitochondrial biogenesis.

**(E)** **(VO, CE, IOD, IAO)** [**Rageh MM**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rageh%20MM%5BAuthor%5D&cauthor=true&cauthor_uid=23091355)**,** [**El-Gebaly RH**](http://www.ncbi.nlm.nih.gov/pubmed?term=El-Gebaly%20RH%5BAuthor%5D&cauthor=true&cauthor_uid=23091355)**,** [**El-Bialy NS**](http://www.ncbi.nlm.nih.gov/pubmed?term=El-Bialy%20NS%5BAuthor%5D&cauthor=true&cauthor_uid=23091355)**. Assessment of genotoxic and cytotoxic hazards in brain and bone marrow cells of newborn rats exposed to extremely low-frequency magnetic field.** [**J Biomed Biotechnol.**](http://www.ncbi.nlm.nih.gov/pubmed/23091355) **2012;2012:716023.**

The present study aimed to evaluate the association between whole body exposure to extremely low frequency magnetic field (ELF-MF) and genotoxic , cytotoxic hazards in brain and bone marrow cells of newborn rats. Newborn rats (10 days after delivery) were exposed continuously to 50 Hz, 0.5 mT for 30 days. The control group was treated as the exposed one with the sole difference that the rats were not exposed to magnetic field. Comet assay was used to quantify the level of DNA damage in isolated brain cells. Also bone marrow cells were flushed out to assess micronucleus induction and mitotic index. Spectrophotometric methods were used to measure the level of malondialdehyde (MDA) and the activity of glutathione (GSH) and superoxide dismutase (SOD). The results showed a significant increase in the mean tail moment indicating DNA damage in exposed group (P < 0.01, 0.001, 0.0001). Moreover ELF-MF exposure induced a significant (P < 0.01, 0.001) four folds increase in the induction of micronucleus and about three folds increase in mitotic index (P < 0.0001). Additionally newborn rats exposed to ELF-MF showed significant higher levels of MDA and SOD (P < 0.05). Meanwhile ELF-MF failed to alter the activity of GSH. In conclusion, the present study suggests an association between DNA damage and ELF-MF exposure in newborn rats.

**(E)** [**(HU, AE, DOD) Raggi F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Raggi%20F%22%5BAuthor%5D)**,** [**Vallesi G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Vallesi%20G%22%5BAuthor%5D)**,** [**Rufini S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Rufini%20S%22%5BAuthor%5D)**,** [**Gizzi S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Gizzi%20S%22%5BAuthor%5D)**,** [**Ercolani E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Ercolani%20E%22%5BAuthor%5D)**,** [**Rossi R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Rossi%20R%22%5BAuthor%5D)**. ELF magnetic therapy and oxidative balance.** [**Electromagn Biol Med.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Electromagn%20Biol%20Med.');) **27(4):325-339, 2008.**

Knowledge about the relationship between exposure to extremely low-frequency (ELF) EMF and formation (or neutralization) of free radicals in the living cells is limited. Studies performed on animals and plants have shown conflicting effects on the relation between EMF and oxidative stress. Very few experiments have been performed on humans. The present study reports on the effects of an ELF magnetic therapy device (Seqex) on oxidative scale in humans. This device supplies complex magnetic signals with specific choices of frequency, intensity, and shape that are based on Liboff's ion cyclotron resonance hypothesis. Thirty-two healthy volunteers were treated using the Seqex cycle. A quantitative determination of oxidative stress was obtained at three time points by measuring malondialdehyde (MDA) concentrations in peripheral blood before and after the cycle and one month following completion of the cycle. A highly significant reduction in mean MDA (53.8%, p = 0.0002) was found at the end of the treatment. One month later the mean MDA had again risen, but there was still a significant overall reduction of 15.6% (p = 0.010) compared to original values.

**(E)** **(VO, AE, IAO)** [**Rajabbeigi E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rajabbeigi%20E%5BAuthor%5D&cauthor=true&cauthor_uid=23323716)**,** [**Ghanati F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghanati%20F%5BAuthor%5D&cauthor=true&cauthor_uid=23323716)**,** [**Abdolmaleki P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Abdolmaleki%20P%5BAuthor%5D&cauthor=true&cauthor_uid=23323716)**,** [**Payez A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Payez%20A%5BAuthor%5D&cauthor=true&cauthor_uid=23323716)**. Antioxidant capacity of parsley cells (Petroselinum crispum L.) in relation to iron-induced ferritin levels and static magnetic field.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/?term=rajabbeigi+and+antioxidant) **32(4):430-441, 2013.**

This study was aimed to evaluate antioxidant response of parsley cells to 21 ppm iron and static magnetic field (SMF; 30 mT). The activity of catalase (CAT) and ascorbate peroxidase (APX) and the contents of malonyldialdehyde, iron and ferritin were measured at 6 and 12 h after treatments. Exposure to SMF increased the activity of CAT in treated cells, while combination of iron and SMF treatments as well as iron supply alone decreased CAT activity, compared to that of control cells. Combination of SMF with iron treatment reduced iron content of the cells and ameliorated mal effect of iron on CAT activity. All treatments reduced APX activity; however, the content of total ascorbate increased in response to iron and SMF+iron. The results showed that among the components of antioxidant system of parsley cells, enhanced activity of CAT in SMF-treated cells and increase of ascorbate in SMF+Fe-treated ones were responsible for the maintenance of membranes integrity. Ferritin contents of SMF- and SMF+Fe-treated cells also decreased significantly 12 h after treatments, compared to those of the control cells. These results cast doubt on the proposed functions of ferritin as a putative reactive oxygen species detoxifying molecule.

**(E) (VO, CE, IX)** [**Rauš Balind S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rau%C5%A1%20Balind%20S%5BAuthor%5D&cauthor=true&cauthor_uid=24586442)**,** [**Selaković V**](http://www.ncbi.nlm.nih.gov/pubmed?term=Selakovi%C4%87%20V%5BAuthor%5D&cauthor=true&cauthor_uid=24586442)**,** [**Radenović L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Radenovi%C4%87%20L%5BAuthor%5D&cauthor=true&cauthor_uid=24586442)**,** [**Prolić Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Proli%C4%87%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=24586442)**,** [**Janać B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jana%C4%87%20B%5BAuthor%5D&cauthor=true&cauthor_uid=24586442)**. Extremely low frequency magnetic field (50 Hz, 0.5 mT) reduces oxidative stress in the brain of gerbils submitted to global cerebral ischemia.** [**PLoS One.**](http://www.ncbi.nlm.nih.gov/pubmed/24586442) **2014 Feb 19;9(2):e88921. doi: 10.1371/journal.pone.0088921. eCollection 2014.**

Magnetic field as ecological factor has influence on all living beings. The aim of this study was to determine if extremely low frequency magnetic field (ELF-MF, 50 Hz, 0.5 mT) affects oxidative stress in the brain of gerbils submitted to 10-min global cerebral ischemia. After occlusion of both carotid arteries, 3-month-old gerbils were continuously exposed to ELF-MF for 7 days. Nitric oxide and superoxide anion production, superoxide dismutase activity and index of lipid peroxidation were examined in the forebrain cortex, striatum and hippocampus on the 7(th) (immediate effect of ELF-MF) and 14(th) day after reperfusion (delayed effect of ELF-MF). Ischemia per se increased oxidative stress in the brain on the 7(th) and 14(th) day after reperfusion. ELF-MF also increased oxidative stress, but to a greater extent than ischemia, only immediately after cessation of exposure. Ischemic gerbils exposed to ELF-MF had increased oxidative stress parameters on the 7(th) day after reperfusion, but to a lesser extent than ischemic or ELF-MF-exposed animals. On the 14(th) day after reperfusion, oxidative stress parameters in the brain of these gerbils were mostly at the control levels. Applied ELF-MF decreases oxidative stress induced by global cerebral ischemia and thereby reduces possible negative consequences which free radical species could have in the brain. The results presented here indicate a beneficial effect of ELF-MF (50 Hz, 0.5 mT) in the model of global cerebral ischemia.

**(E) (VT, AE, IAO)** [**Reale M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**De Lutiis MA**](https://www.ncbi.nlm.nih.gov/pubmed/?term=De%20Lutiis%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Patruno A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Speranza L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Speranza%20L%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Felaco M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Felaco%20M%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Grilli A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Grilli%20A%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Macrì MA**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Macr%C3%AC%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Comani S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Comani%20S%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Conti P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Conti%20P%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**,** [**Di Luzio S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Di%20Luzio%20S%5BAuthor%5D&cauthor=true&cauthor_uid=16455275)**. Modulation of MCP-1 and iNOS by 50-Hz sinusoidal electromagnetic field.** [**Nitric Oxide.**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Reale+M+and+De+Lutiis+and+2006) **15(1):50-57, 2006.**

The purpose of this study was to investigate whether overnight exposure to 1 mT-50 Hz extremely low-frequency sinusoidal electromagnetic field (EMF) affects the expression and production of inducible nitric oxide synthase (iNOS) and monocyte chemotactic protein-1 (MCP-1) in human monocytes. RT-PCR and Western blot analysis demonstrate that EMF exposure affects the expression of iNOS and MCP-1 in cultured human mononuclear cells at the mRNA level and protein synthesis. Interestingly, the effects of EMF exposure clearly differed with respect to the potentiation and inhibition of iNOS and MCP-1 expression. Whereas iNOS was down-regulated both at the mRNA level and at the protein level, MCP-1 was up-regulated. These results provide helpful information regarding the EMF-mediated modulation of the inflammatory response in vivo. However, additional studies are necessary to demonstrate that EMF acts as a nonpharmacological inhibitor of NO and inducer of MCP-1 in some diseases where the balance of MCP-1 and NO may be important.

**(E) (VT, AE, IFR)**  [**Reale M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Reale%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**Kamal MA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kamal%20MA%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**Patruno A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Patruno%20A%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**Costantini E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Costantini%20E%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**D'Angelo C**](http://www.ncbi.nlm.nih.gov/pubmed?term=D'Angelo%20C%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**Pesce M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pesce%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**,** [**Greig NH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Greig%20NH%5BAuthor%5D&cauthor=true&cauthor_uid=25127118)**. Neuronal cellular responses to extremely low frequency electromagnetic field exposure: implications regarding oxidative stress and neurodegeneration.** [**PLoS One.**](http://www.ncbi.nlm.nih.gov/pubmed/25127118) **2014 Aug 15;9(8):e104973. doi: 10.1371/journal.pone.0104973. eCollection 2014.**

Neurodegenerative diseases comprise both hereditary and sporadic conditions characterized by an identifying progressive nervous system dysfunction and distinctive neuopathophysiology. The majority are of non-familial etiology and hence environmental factors and lifestyle play key roles in their pathogenesis. The extensive use of and ever increasing worldwide demand for electricity has stimulated societal and scientific interest on the environmental exposure to low frequency electromagnetic fields (EMFs) on human health. Epidemiological studies suggest a positive association between 50/60-Hz power transmission fields and leukemia or lymphoma development. Consequent to the association between EMFs and induction of oxidative stress, concerns relating to development of neurodegenerative diseases, such as Alzheimer disease (AD), have been voiced as the brain consumes the greatest fraction of oxygen and is particularly vulnerable to oxidative stress. Exposure to extremely low frequency (ELF)-EMFs are reported to alter animal behavior and modulate biological variables, including gene expression, regulation of cell survival, promotion of cellular differentiation, and changes in cerebral blood flow in aged AD transgenic mice. Alterations in inflammatory responses have also been reported, but how these actions impact human health remains unknown. We hence evaluated the effects of an electromagnetic wave (magnetic field intensity 1mT; frequency, 50-Hz) on a well-characterized immortalized neuronal cell model, human SH-SY5Y cells. ELF-EMF exposure elevated the expession of NOS and O2-, which were countered by compensatory changes in antioxidant catylase (CAT) activity and enzymatic kinetic parameters related to CYP-450 and CAT activity. Actions of ELF-EMFs on cytokine gene expression were additionally evaluated and found rapidly modified. Confronted with co-exposure to H2O2-induced oxidative stress, ELF-EMF proved not as well counteracted and resulted in a decline in CAT activity and a rise in O2- levels. Together these studies support the further evaluation of ELF-EMF exposure in cellular and in vivo preclinical models to define mechanisms potentially impacted in humans.

**(E)** **(VO, CE, LI, IAO, DAO)** [**Regoli F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Regoli%20F%22%5BAuthor%5D)**,** [**Gorbi S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Gorbi%20S%22%5BAuthor%5D)**,** [**Machella N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Machella%20N%22%5BAuthor%5D)**,** [**Tedesco S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tedesco%20S%22%5BAuthor%5D)**,** [**Benedetti M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Benedetti%20M%22%5BAuthor%5D)**,** [**Bocchetti R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Bocchetti%20R%22%5BAuthor%5D)**,** [**Notti A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Notti%20A%22%5BAuthor%5D)**,** [**Fattorini D**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fattorini%20D%22%5BAuthor%5D)**,** [**Piva F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Piva%20F%22%5BAuthor%5D)**,** [**Principato G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Principato%20G%22%5BAuthor%5D)**. Pro-oxidant effects of extremely low frequency electromagnetic fields in the land snail Helix aspersa.** [**Free Radic Biol Med.**](javascript:AL_get(this,%20'jour',%20'Free%20Radic%0d%0a%20Biol%20Med.');) **39(12):1620-1628, 2005.**

Pro-oxidant effects of extremely low frequency (ELF) 50-Hz magnetic fields were investigated in the land snail Helix aspersa exposed both in short-term laboratory treatments and under field conditions by maintaining the organisms in the proximity of a power line for up to 2 months. Oxidative perturbations were investigated as individual antioxidants (catalase, glutathione reductase, glutathione S-transferases, and total glutathione) and total scavenging capacity toward peroxyl radicals and hydroxyl radicals. Accumulation of lipid peroxidation products, destabilization of lysosomal membranes, and loss of DNA integrity were also evaluated as markers of cell damage. The overall results indicated an oxidative challenge caused by ELF magnetic fields with particularly prompt and sensitive responses for catalase, glutathione reductase, and the overall capability to neutralize peroxyl radicals. Cell injuries occurred to different extents according to duration and intensity of electromagnetic exposure and confirmed complex cause-effect relationships between pro-oxidant factors, efficiency of antioxidant defenses, and the onset of oxidative toxicity. This study highlights the importance of a multimarker approach for detecting a wide panel of biological responses, the necessity of investigating the long-term effects of early oxidative responses, and the role of ELF in enhancing susceptibility to other forms of pathologies or diseases.

**(E)** **(VT, AE, IFR)** [**Rollwitz J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rollwitz%20J%5BAuthor%5D&cauthor=true&cauthor_uid=15541292)**,** [**Lupke M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Lupke%20M%5BAuthor%5D&cauthor=true&cauthor_uid=15541292)**,** [**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Simk%C3%B3%20M%5BAuthor%5D&cauthor=true&cauthor_uid=15541292)**. Fifty-hertz magnetic fields induce free radical formation in mouse bone marrow-derived promonocytes and macrophages.** [**Biochim Biophys Acta.**](http://www.ncbi.nlm.nih.gov/pubmed/15541292) **1674(3):231-238, 2004.**

Our findings show a significant increase of free radical production after exposure to 50 Hz electromagnetic fields at a flux density of 1 mT to mouse bone marrow-derived (MBM) promonocytes and macrophages, indicating the cell-activating capacity of extremely low frequency magnetic fields (ELF-MF). We demonstrate that after exposure to ELF-MF mainly superoxide anion radicals were produced, both in MBM macrophages (33%) and also in their precursor cells (24%). To elucidate whether NADPH- or NADH-oxidase functions are target proteins for MF interaction, the flavoprotein inhibitor diphenyleneiodonium chloride (DPI) was used. MF-induced free radical production was not inhibited by DPI, whereas tetradecanoylphorbolacetate (TPA)-induced free radical production was diminished by about 70%. TPA is known to induce a direct activation of NADPH-oxidase through the PKC pathway. Since DPI lacks an inhibitory effect in MF-exposed MBM cells, we suggest that 50 Hz MF stimulates the NADH-oxidase pathway to produce superoxide anion radicals, but not the NADPH pathway. Furthermore, we showed an oscillation (1-10 days) in superoxide anion radical release in mouse macrophages, indicating a cyclic pattern of NADH-oxidase activity.

**(E)** **(VT, AE, IFR)** [**Roy S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Roy%20S%22%5BAuthor%5D)**,** [**Noda Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Noda%20Y%22%5BAuthor%5D)**,** [**Eckert V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Eckert%20V%22%5BAuthor%5D)**,** [**Traber MG**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Traber%20MG%22%5BAuthor%5D)**,** [**Mori A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mori%20A%22%5BAuthor%5D)**,** [**Liburdy R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Liburdy%20R%22%5BAuthor%5D)**,** [**Packer L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Packer%20L%22%5BAuthor%5D)**. The phorbol 12-myristate 13-acetate (PMA)-induced oxidative burst in rat peritoneal neutrophils is increased by a 0.1 mT (60 Hz) magnetic field.** [**FEBS Lett.**](javascript:AL_get(this,%20'jour',%20'FEBS%20%0d%0aLett.');) **376(3):164-166, 1995.**

Magnetic fields (MF) may affect biological systems by increasing free radical concentrations. To test this, we have investigated whether low frequency (60 Hz) low intensity (0.1 mT) MF can modulate the phorbol 12-myristate 13- acetate (PMA) induced respiratory burst in primed rat peritoneal neutrophils, followed in real time using the dye 2',7'-dichlorofluorescin (DCFH), which reacts with free radical-derived oxidants such as H2O2 (which is formed from the dismutation of superoxide) to become 2',7'-dichlorofluorecein (DCF), a highly fluorescent compound. In the presence of the MF, a 12.4% increase in the fluorescence signal was observed in PMA-stimulated neutrophils (n = 5, P < 0.02, 18 pairs of measurements). We believe this represents the first experimental observation of MF influencing events involving free radical species generated during signal transduction in living cells.

**(E)** **(VT, AE, IFR)** [**Sadeghipour R**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sadeghipour%20R%5BAuthor%5D&cauthor=true&cauthor_uid=22676212)**,** [**Ahmadian S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ahmadian%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22676212)**,** [**Bolouri B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bolouri%20B%5BAuthor%5D&cauthor=true&cauthor_uid=22676212)**,** [**Pazhang Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pazhang%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=22676212)**,** [**Shafiezadeh M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Shafiezadeh%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22676212)**. Effects of extremely low-frequency pulsed electromagnetic fields on morphological and biochemical properties of human breast carcinoma cells (T47D).** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/22676212) **31(4):425-435, 2012.**

This study was carried out to investigate the effects of 100 and 217 Hz extremely low-frequency pulsed electromagnetic fields (ELF-PEMF) on cell proliferation, actin reorganization, and ROS generation in a human breast carcinoma cells (T47D). Cells were exposed for 24-72 h, at 100 and 217 Hz, 0.1 mT. The treatment induced a time dependent decrease in cell growth after 72 h and revealed an increase in fluorescence intensity in cytoplasm and actin aggregations around the nucleus as detected by fluorescence microscopy. The amount of actin in T47D cells increased after 48 h exposure to 100 Hz and 24 h to 217 Hz while no changes in nuclear morphology were detected. Exposing the cells to 217 Hz for 72 h caused a dramatically increase of intracellular ROS generation while with exposure to 100 Hz it remained nearly unchanged. These results suggest that exposure to ELF-PEMF (100, 217 Hz, 0.1 mT) are able inducing an increase of actin level, its migration toward nucleus but despite of these changes and dramatically increase in ROS generation the symptoms of apoptosis were not observed. Our results support the hypothesis that cell response to EMF may only be observed at certain window effects; such as frequency and intensity of EMF parameters.

**(E)** **(VT, CE, IOD, IAO, DAO)** [**Sahebjamei H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Sahebjamei%20H%5BAuthor%5D&cauthor=true&cauthor_uid=16988990)**,** [**Abdolmaleki P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Abdolmaleki%20P%5BAuthor%5D&cauthor=true&cauthor_uid=16988990)**,** [**Ghanati F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ghanati%20F%5BAuthor%5D&cauthor=true&cauthor_uid=16988990)**. Effects of magnetic field on the antioxidant enzyme activities of suspension-cultured tobacco cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/16988990) **28(1):42-47, 2007.**

Effects of magnetic fields (MFs) on the activities of antioxidant enzymes of suspension-cultured tobacco cells were investigated. Compared with the control cells, exposure of the cells to static MF with the magnitudes of 10 and 30 mT for 5 days, 5 h each day, increased the activity of superoxide dismutase (SOD). In contrast, the activity of the catalase (CAT) and ascorbate peroxidase (APX) was decreased by MF, compared with those of the control cells. Level of lipid peroxidation was also increased by MF. It suggests that MF could deteriorate antioxidant defense system of plant cells.

**(E) (VO, CE, IFR)** [**Salunke BP**](http://www.ncbi.nlm.nih.gov/pubmed?term=Salunke%20BP%5BAuthor%5D&cauthor=true&cauthor_uid=24780504)**,** [**Umathe SN**](http://www.ncbi.nlm.nih.gov/pubmed?term=Umathe%20SN%5BAuthor%5D&cauthor=true&cauthor_uid=24780504)**,** [**Chavan JG**](http://www.ncbi.nlm.nih.gov/pubmed?term=Chavan%20JG%5BAuthor%5D&cauthor=true&cauthor_uid=24780504)**. Experimental evidence for involvement of nitric oxide in low frequency magnetic field induced obsessive compulsive disorder-like behavior.** [**Pharmacol Biochem Behav.**](http://www.ncbi.nlm.nih.gov/pubmed/24780504) **122:273-278, 2014.**

It is well documented that extremely low frequency magnetic field (ELF MF) produced effects on the function of nervous system in humans and laboratory animals. Dopaminergic and serotonergic pathways have been implicated in obsessive compulsive disorder (OCD). Recently involvement of nitric oxide (NO) in OCD-like behavior is suggested. Hence, the present study was carried out to understand the involvement of dopamine, serotonin and NO in ELF MF induced OCD-like behavior. Swiss albino mice were exposed to ELF MF (50Hz, 10G) for 8h/day for 7, 30, 60, 90 and 120days by subjecting them to Helmholtz coils. OCD-like behavior was assessed in terms of marble burying behavior (MBB) test. Results revealed that ELF MF induced time dependant MBB, on 7th, 30th, 60th, 90th, and 120th exposure day. Further, levels of dopamine, serotonin and NO after 120days of ELF MF exposure were determined in regions of the brain. The neurohumoral studies revealed that exposure to ELF MF increased NO levels in cortex, hippocampus and hypothalamus, and levels of dopamine and serotonin remain unaffected. As OCD-like behavior after ELF MF exposure was associated with higher levels of NO with no significant change in serotonin and dopamine, the effect of such exposure was studied in groups concurrently treated with NO modulators, NO precursor, L-ARG (400mg/kg) or NOS inhibitor, L-NAME (15.0mg/kg) or 7-NI (10.0mg/kg). These treatments revealed that NO precursor exacerbated and NOS inhibitors attenuated ELF MF induced OCD-like behavior with corresponding changes in the levels of NO.

**(E) (VO, IOD, IAO, DAO)** [**Seifirad S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Seifirad%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**,** [**Farzampour S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Farzampour%20S%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**,** [**Nourbakhsh M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Nourbakhsh%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**,** [**Amoli MM**](http://www.ncbi.nlm.nih.gov/pubmed?term=Amoli%20MM%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**,** [**Razzaghy-Azar M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Razzaghy-Azar%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**,** [**Larijani B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Larijani%20B%5BAuthor%5D&cauthor=true&cauthor_uid=25152870)**Effects of extremely low frequency electromagnetic fields on paraoxonase serum activity and lipid peroxidation metabolites in rat.** [**J Diabetes Metab Disord.**](http://www.ncbi.nlm.nih.gov/pubmed/25152870) **13(1):85, 2014.**

BACKGROUND: Atherogenic effects of ELF-MF exposure have not been studied well so far. Therefore we have hypothesized that ELF-MF exposure might have atherogenic effect by impairing antioxidant function and increasing lipid peroxidation. This study was therefore undertaken to examine the effects of ELF-MF on paraoxonase (PON) activity, antioxidant capacity and lipid peroxidation metabolites. Effects of time on remodeling of antioxidant system were also investigated in this study. METHODS: Seventy five Wistar rats were randomly allocated into five groups as follows: 1) Sham exposure, 2) Single exposure to 60 Hz, sacrificed immediately after exposure, 3) Single exposure to 60 Hz, sacrificed 72 hours after exposure, 4) Fourteen days of exposure to 60 Hz, sacrificed immediately after exposure, and 5) Fourteen days of exposure to 60 Hz, sacrificed 72 hours after exposure. Blood samples were collected and analyzed. The results were compared using ANOVA and post hoc Tukey HSD for multiple caparisons. RESULTS: Single ELF-MF exposure significantly increased lipid peroxidation (CD and MDA) and increased antioxidant serum activity (HDL, paraoxonase activity, and serum total antioxidant capacity). Chronic ELF-MF exposure increased lipid peroxidation and affected antioxidant system. Free fatty acids levels were significantly increased after both single and two weeks exposure. Chronic exposure led to irreversible changes while acute exposure tended to reversible alterations on above mentioned parameters. CONCLUSIONS: According to the results of this study, ELF-MF exposure could impair oxidant-antioxidant function and might increase oxidative stress and lipid peroxidation. Antioxidant capability was dependent on the duration and continuity of ELF-MF exposure.

**(E) (VO, CE, IFR, IOD, IAO)** [**Selaković V**](http://www.ncbi.nlm.nih.gov/pubmed?term=Selakovi%C4%87%20V%5BAuthor%5D&cauthor=true&cauthor_uid=23292355)**,** [**Rauš Balind S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Rau%C5%A1%20Balind%20S%5BAuthor%5D&cauthor=true&cauthor_uid=23292355)**,** [**Radenović L**](http://www.ncbi.nlm.nih.gov/pubmed?term=Radenovi%C4%87%20L%5BAuthor%5D&cauthor=true&cauthor_uid=23292355)**,** [**Prolić Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=Proli%C4%87%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=23292355)**,** [**Janać B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jana%C4%87%20B%5BAuthor%5D&cauthor=true&cauthor_uid=23292355)**. Age-dependent effects of ELF-MF on oxidative stress in the brain of mongolian gerbils.** [**Cell Biochem Biophys.**](http://www.ncbi.nlm.nih.gov/pubmed/23292355) **66(3):513-521, 2013.**

The aim of study was to investigate the effects of extremely low frequency magnetic field (ELF-MF; 50 Hz; 0.1, 0.25 and 0.5 mT) on oxidative stress in the brain of 3- (adult) and 10-month-old (middle-aged) gerbils. Nitric oxide (NO) level, superoxide (O(2) (-)) production, superoxide dismutase (SOD) activity, and index of lipid peroxidation (ILP) were measured in the forebrain cortex, striatum, hippocampus, and cerebellum immediately and 3 days after cessation of 7-day exposure. In all gerbils, ELF-MF significantly increased oxidative stress in all tested brain regions. This effect was correlated with the value of magnetic induction and was higher in middle-aged gerbils. Three days after cessation of exposure, the values of examined parameters were closer to control levels. In adult gerbils, the effect of ELF-MF of 0.1 mT on NO level, O(2) (-) production and SOD activity was almost fully disappeared, and ILP was at the control level regardless of the value of magnetic induction. In middle-aged gerbils, the effect of ELF-MF was still present but to a lesser degree than those observed immediately after cessation of exposure. These findings pointed out the ability of ELF-MF to induce age- and magnetic induction-dependent modification of oxidative stress in the brain.

[**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**,** [**Güler G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22G%C3%BCler%20G%22%5BAuthor%5D)**. Review of in vivo static and ELF electric fields studies performed at Gazi Biophysics Department.** [**Electromagn Biol Med.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Electromagn%20Biol%20Med.');) **25(4):307-323, 2006. (review)**

In vivo effects of Static Electric and ELF Magnetic and Electric fields have been carried out for more than 20 years in the Bioelectromagnetic Laboratory at the Biophysics Department of the Medical Faculty of Gazi University. In this article, the results of in vivo ELF Electric field studies are presented as a review. Static and 50 Hz ELF (Extremely Low Frequency) Electric (E) fields effects on free radical synthesis, antioxidant enzyme level, and collagen synthesis were analyzed on tissues of guinea pigs, such as brain, liver, lung, kidney, spleen, testis, and plasma. Animals were exposed to static and ELF electric fields with intensities ranging from 0.3 kV/m to 1.9 kV/m in vertical and horizontal directions. Exposure periods were 1, 3, 5, 7, and 10 days. Electric fields were generated from a specially designed parallel plate capacitor system. The results indicate that the effects of electric fields on the tissues studied depend significantly on the type and magnitude of electric field and exposure period.

**(E) (VO, CE, DAO)** [**Sharifian A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sharifian%20A%22%5BAuthor%5D)**,** [**Gharavi M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Gharavi%20M%22%5BAuthor%5D)**,** [**Pasalar P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Pasalar%20P%22%5BAuthor%5D)**,** [**Aminian O**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Aminian%20O%22%5BAuthor%5D)**. Effect of extremely low frequency magnetic field on antioxidant activity in plasma and red blood cells in spot welders.** [**Int Arch Occup Environ Health.**](javascript:AL_get(this,%20'jour',%20'Int%20Arch%20%0d%0aOccup%20Environ%20Health.');) **82(2):259-266, 2009.**

OBJECTIVE: The purpose of this study was to determine a possible relation between exposure to extremely low frequency magnetic field (ELF-MF) and the human antioxidant activity. METHODS: The total serum antioxidant status (TAS), red blood cells (RBCs) glutathione peroxidase (GPX) and superoxide dismutase (SOD) were measured in 46 spot welders who were occupationally exposed to ELF-MF (magnetic field strength = 8.8-84 microTesla (microT), frequency = 50 Hertz (Hz) and electric field strength = 20-133 V/m). The results were compared with a nonexposed ELF-MF control group. The correlation between magnetic field strength and antioxidant activity in RBCs and plasma was then assessed. RESULTS: No significant differences in TAS levels were observed (P value = 0.065). However, in RBCs of exposed group, a significant decrease in SOD and GPX activities was observed (P value = 0.001 and 0.003, respectively). This decrease was measured as 22 and 12.3%, respectively. Furthermore, a significant negative correlation between SOD/GPX activities and magnetic field intensity was observed (coefficients of SOD: -0.625, significance: 0.0001 and coefficients of GPX: -0.348, significance: 0.018). CONCLUSION: The results of this study indicate that ELF-MF could influence the RBC antioxidant activity and might act as an oxidative stressor. Intracellular antioxidant enzymes such as SOD and GPX were found to be the most important markers involving in this process. The influence of magnetic field on the antioxidant activity of RBCs might occur even at the recommended levels of exposure.

**(E) (VO, AE, IFR, IAO, DAO)** [**Shine MB**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Shine%20MB%5BAuthor%5D&cauthor=true&cauthor_uid=22253132)**,** [**Guruprasad KN**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Guruprasad%20KN%5BAuthor%5D&cauthor=true&cauthor_uid=22253132)**,** [**Anand A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Anand%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22253132)**. Effect of stationary magnetic field strengths of 150 and 200 mT on reactive oxygen species production in soybean.** [**Bioelectromagnetics.**](https://www.ncbi.nlm.nih.gov/pubmed/22253132) **33(5):428-437, 2012.**

Our previous investigation reported the beneficial effect of pre-sowing magnetic treatment for improving germination parameters and biomass accumulation in soybean. In this study, soybean seeds treated with static magnetic fields of 150 and 200 mT for 1 h were evaluated for reactive oxygen species (ROS) and activity of antioxidant enzymes. Superoxide and hydroxyl radicals were measured in embryos and hypocotyls of germinating seeds by electron paramagnetic resonance spectroscopy and kinetics of superoxide production; hydrogen peroxide and antioxidant activities were estimated spectrophotometrically. Magnetic field treatment resulted in enhanced production of ROS mediated by cell wall peroxidase while ascorbic acid content, superoxide dismutase and ascorbate peroxidase activity decreased in the hypocotyl of germinating seeds. An increase in the cytosolic peroxidase activity indicated that this antioxidant enzyme had a vital role in scavenging the increased H(2)O(2) produced in seedlings from the magnetically treated seeds. Hence, these studies contribute to our first report on the biochemical basis of enhanced germination and seedling growth in magnetically treated seeds of soybean in relation to increased production of ROS.

**(E)** **(VT, AE, IFR)** [**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Simk%C3%B3%20M%22%5BAuthor%5D)**,** [**Droste S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Droste%20S%22%5BAuthor%5D)**,** [**Kriehuber R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kriehuber%20R%22%5BAuthor%5D)**,** [**Weiss DG**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Weiss%20DG%22%5BAuthor%5D)**. Stimulation of phagocytosis and free radical production in murine macrophages by 50 Hz electromagnetic fields.** [**Eur J Cell Biol.**](javascript:AL_get(this,%20'jour',%20'Eur%20J%20Cell%0d%0a%20Biol.');) **80(8):562-566, 2001.**

Effects of 50 Hz electromagnetic fields on phagocytosis and free radical production were examined in mouse bone marrow-derived macrophages. Macrophages were in vitro exposed to electromagnetic fields using different magnetic field densities (0.5-1.5 mT). Short-time exposure (45 min) to electromagnetic fields resulted in significantly increased phagocytic uptake (36.3% +/- 15.1%) as quantified by measuring the internalization rate of latex beads. Stimulation with 1 nM 12-0-tetradecanoylphorbol-13-acetate (TPA) showed the same increased phagocytic activity as 1 mT electromagnetic fields. However, co-exposure to electromagnetic fields and TPA showed no further increase of bead uptake, and therefore we concluded that because of the absence of additive effects, the electromagnetic fields-induced stimulation of mouse bone marrow-derived macrophages does not involve the protein kinase C signal transduction pathway. Furthermore, a significant increased superoxide production after exposure to electromagnetic fields was detected.

[**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Simk%C3%B3%20M%22%5BAuthor%5D)**. Cell type specific redox status is responsible for diverse electromagnetic field effects.** [**Curr Med Chem.**](javascript:AL_get(this,%20'jour',%20'Curr%20Med%20%0d%0aChem.');) **14(10):1141-1152, 2007. (review)**

Epidemiologic and experimental research on the potential carcinogenic effects of extremely low frequency electromagnetic fields (ELF-EMF) has been performed for a long time. Epidemiologic studies regarding ELF-EMF-exposure have focused primarily on leukaemia development due to residential sources in children and adults, and from occupational exposure in adults, but also on other kinds of cancer. Genotoxic investigations of EMF have shown contradictory results, a biological mechanism is still lacking that can explain the link between cancer development and ELF-EMF-exposure. Recent laboratory research has attempted to show general biological effects, and such that could be related to cancer development and/or promotion. Metabolic processes which generate oxidants and antioxidants can be influenced by environmental factors, such as ELF-EMF. Increased ELF-EMF exposure can modify the activity of the organism by reactive oxygen species leading to oxidative stress. It is well established that free radicals can interact with DNA resulting in single strand breaks. DNA damage could become a site of mutation, a key step to carcinogenesis. Furthermore, different cell types react differently to the same stimulus, because of their cell type specific redox status. The modulation of cellular redox balance by the enhancement of oxidative intermediates, or the inhibition or reduction of antioxidants, is discussed in this review. An additional aspect of free radicals is their function to influence other illnesses such as Parkinson's and Alzheimer's diseases. On the other hand, modulation of antioxidants by ELF-EMF can lower the intracellular defence activity promoting the development of DNA damage. It has also been demonstrated that low levels of reactive oxygen species trigger intracellular signals that involve the transcription of genes and leading to responses including cell proliferation and apoptosis. In this review, a general overview is given about oxidative stress, as well as experimental studies are reviewed as they are related to changes in oxidant and antioxidant content after ELF-EMF exposure inducing different biological effects. Finally, we conclude from our review that modulations on the oxidant and antioxidant level through ELF-EMF exposure can play a causal role in cancer development.

[**Simkó M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Simk%C3%B3%20M%22%5BAuthor%5D)**,** [**Mattsson MO**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mattsson%20MO%22%5BAuthor%5D)**. Extremely low frequency electromagnetic fields as effectors of cellular responses in vitro: possible immune cell activation.** [**J Cell Biochem.**](javascript:AL_get(this,%20'jour',%20'J%20Cell%20%0d%0aBiochem.');) **93(1):83-92, 2004. (review)**

There is presently an intense discussion if electromagnetic field (EMF) exposure has consequences for human health. This include exposure to structures and appliances that emit in the extremely low frequency (ELF) range of the electromagnetic spectrum, as well as emission coming from communication devices using the radiofrequency part of the spectrum. Biological effects of such exposures have been noted frequently, although the implication for specific health effects is not that clear. The basic interaction mechanism(s) between such fields and living matter is unknown. Numerous hypotheses have been suggested, although none is convincingly supported by experimental data. Various cellular components, processes, and systems can be affected by EMF exposure. Since it is unlikely that EMF can induce DNA damage directly, most studies have examined EMF effects on the cell membrane level, general and specific gene expression, and signal transduction pathways. In addition, a large number of studies have been performed regarding cell proliferation, cell cycle regulation, cell differentiation, metabolism, and various physiological characteristics of cells. Although 50/60 Hz EMF do not directly lead to genotoxic effects, it is possible that certain cellular processes altered by exposure to EMF indirectly affect the structure of DNA causing strand breaks and other chromosomal aberrations. The aim of this article is to present a hypothesis of a possible initial cellular event affected by exposure to ELF EMF, an event which is compatible with the multitude of effects observed after exposure. Based on an extensive literature review, we suggest that ELF EMF exposure is able to perform such activation by means of increasing levels of free radicals. Such a general activation is compatible with the diverse nature of observed effects. Free radicals are intermediates in natural processes like mitochondrial metabolism and are also a key feature of phagocytosis. Free radical release is inducible by ionizing radiation or phorbol ester treatment, both leading to genomic instability. EMF might be a stimulus to induce an "activated state" of the cell such as phagocytosis, which then enhances the release of free radicals, in turn leading to genotoxic events. We envisage that EMF exposure can cause both acute and chronic effects that are mediated by increased free radical levels: (1) Direct activation of, for example macrophages (or other cells) by short-term exposure to EMF leads to phagocytosis (or other cell specific responses) and consequently, free radical production. This pathway may be utilized to positively influence certain aspects of the immune response, and could be useful for specific therapeutic applications. (2) EMF-induced macrophage (cell) activation includes direct stimulation of free radical production. (3) An increase in the lifetime of free radicals by EMF leads to persistently elevated free radical concentrations. In general, reactions in which radicals are involved become more frequent, increasing the possibility of DNA damage. (4) Long-term EMF exposure leads to a chronically increased level of free radicals, subsequently causing an inhibition of the effects of the pineal gland hormone melatonin. Taken together, these EMF induced reactions could lead to a higher incidence of DNA damage and therefore, to an increased risk of tumour development. While the effects on melatonin and the extension of the lifetime of radicals can explain the link between EMF exposure and the incidence of for example leukaemia, the two additional mechanisms described here specifically for mouse macrophages, can explain the possible correlation between immune cell system stimulation and EMF exposure.

**(E) (HU, AE, IAO, DFR)** [**Sirmatel O**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sirmatel%20O%5BAuthor%5D&cauthor=true&cauthor_uid=17660581)**,** [**Sert C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sert%20C%5BAuthor%5D&cauthor=true&cauthor_uid=17660581)**,** [**Sirmatel F**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sirmatel%20F%5BAuthor%5D&cauthor=true&cauthor_uid=17660581)**,** [**Selek S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Selek%20S%5BAuthor%5D&cauthor=true&cauthor_uid=17660581)**,** [**Yokus B**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Yokus%20B%5BAuthor%5D&cauthor=true&cauthor_uid=17660581)**. Total antioxidant capacity, total oxidant status and oxidative stress index in the men exposed to 1.5 T static magnetic field.** [**Gen Physiol Biophys.**](https://www.ncbi.nlm.nih.gov/pubmed/17660581) **26(2):86-90, 2007a.**

The aim of this study was to investigate the effects of a high-strength magnetic field produced by a magnetic resonance imaging (MRI) apparatus on oxidative stress. The effects of a 1.5 T static magnetic field on the total antioxidant capacity (TAC), total oxidant status (TOS) and oxidative stress index (OSI) in male subjects were investigated. In this study, 33 male volunteers were exposed to a 1.5 T static magnetic field for a short time and the TAC, TOS and OSI of each subject were determined. Magnetic field exposure was provided using a magnetic resonance apparatus; radiofrequency was not applied. Blood samples were taken from subjects and TAC, TOS and OSI values were measured using the methods of Erel. TAC showed a significant increase in post-exposures compared to pre-exposures to the magnetic field (p < 0.05). OSI and TOS showed a significant decrease in post-exposures compared to pre-exposures to a 1.5 T magnetic field (for each of two, p < 0.01). The 1.5 T static magnetic field used in the MRI apparatus did not yield a negative effect; on the contrary, it produced the positive effect of decreasing oxidative stress in men following short-term exposure.

**(E) (HU, AE, IFR)** [**Sirmatel O**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sirmatel%20O%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**,** [**Sert C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sert%20C%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**,** [**Tümer C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=T%C3%BCmer%20C%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**,** [**Oztürk A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ozt%C3%BCrk%20A%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**,** [**Bilgin M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Bilgin%20M%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**,** [**Ziylan Z**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ziylan%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=17080452)**. Change of nitric oxide concentration in men exposed to a 1.5 T constant magnetic field.** [**Bioelectromagnetics.**](https://www.ncbi.nlm.nih.gov/pubmed/17080452) **28(2):152-154, 2007b.**

This study was carried out in order to determine nitric oxide (NO) production immediately after a 1.5 T magnetic field 30 min exposure to an experimental group, comprising 33 healthy young male volunteers aged 18-26 years old. In addition, a control group, comprising 30 healthy male volunteers aged 19-26 years old, was not exposed to the magnetic field and their NO levels were also measured. The experimental group was exposed using a magnetic resonance imaging (MRI) apparatus. Nitrite and nitrate concentrations were determined by UV-VIS spectrophotometer. The results, related to the parameters measured in this study, were analyzed by one-way ANOVA. Total nitrite concentration in post-magnetic field samples was found to be higher than in pre-magnetic field samples (P < .05).

**(E) (VT, AE, IFR)** [**Solek P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Solek%20P%5BAuthor%5D&cauthor=true&cauthor_uid=28323003)**,** [**Majchrowicz L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Majchrowicz%20L%5BAuthor%5D&cauthor=true&cauthor_uid=28323003)**,** [**Bloniarz D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Bloniarz%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28323003)**,** [**Krotoszynska E**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Krotoszynska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=28323003)**,** [**Koziorowski M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Koziorowski%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28323003)**. Pulsed or continuous electromagnetic field induce p53/p21-mediated apoptotic signaling pathway in mouse spermatogenic cells in vitro and thus may affect male fertility.** [**Toxicology.**](https://www.ncbi.nlm.nih.gov/pubmed/28323003) **382:84-92, 2017.**

The impact of electromagnetic field (EMF) on the human health and surrounding environment is a common topic investigated over the years. A significant increase in the electromagnetic field concentration arouses public concern about the long-term effects of EMF on living organisms associated with many aspects. In the present study, we investigated the effects of pulsed and continuous electromagnetic field (PEMF/CEMF) on mouse spermatogenic cell lines (GC-1 spg and GC-2 spd) in terms of cellular and biochemical features in vitro. We evaluated the effect of EMF on mitochondrial metabolism, morphology, proliferation rate, viability, cell cycle progression, oxidative stress balance and regulatory proteins. Our results strongly suggest that EMF induces oxidative and nitrosative stress-mediated DNA damage, resulting in p53/p21-dependent cell cycle arrest and apoptosis. Therefore, spermatogenic cells due to the lack of antioxidant enzymes undergo oxidative and nitrosative stress-mediated cytotoxic and genotoxic events, which contribute to infertility by reduction in healthy sperm cells pool. In conclusion, electromagnetic field present in surrounding environment impairs male fertility by inducing p53/p21-mediated cell cycle arrest and apoptosis.

**(E)** **(VT, AE, IFR)** [**Sullivan K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sullivan%20K%22%5BAuthor%5D)**,** [**Balin AK**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Balin%20AK%22%5BAuthor%5D)**,** [**Allen RG**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Allen%20RG%22%5BAuthor%5D)**. Effects of static magnetic fields on the growth of various types of human cells.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/21225891##) **32(2):140-147, 2011.**

The effects of a static magnetic field (SMF) on the proliferation of various types of human cells were determined. All cultures were maintained at 37 °C throughout the experiment. SMF was generated by placing two magnets oppositely oriented on either side of a T25 flask. The flux density in the flask ranged from 35 to 120 mT. Growth curves were constructed by plotting cell number at 18 h and 4, 7, 11, and 14 days after seeding, with the 18-h point being a measure of attachment efficiency. Exposure to SMF significantly decreased initial attachment of fibroblasts and decreased subsequent growth compared to sham-exposed control. Significant effects were observed in both fetal lung (WI-38) and adult skin fibroblasts, but they were generally larger in the fetal lung fibroblast line. SMF did not affect attachment of human melanoma cells, but inhibited their growth by 20% on day 7. SMF produced no effects in a human adult stem cell line. Oxidant production increased 37% in WI-38 cells exposed to SMF (230-250 mT) during the first 18 h after seeding, when cell attachment occurs. Conversely, no elevation in oxidant levels was observed after a prolonged 5-day exposure. These results indicate that exposure to SMF has significant biological effects in some, but not all types of human cells.

**(E) (VT, AE, DFR)** [**Sun YL**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20YL%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Chen ZH**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20ZH%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Chen XH**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20XH%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Yin C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Yin%20C%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Li DJ**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Li%20DJ%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Ma XL**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ma%20XL%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Zhao F**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhao%20F%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Zhang G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20G%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Shang P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Shang%20P%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**,** [**Qian AR**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Qian%20AR%5BAuthor%5D&cauthor=true&cauthor_uid=25398175)**. Diamagnetic levitation promotes osteoclast differentiation from RAW264.7 cells.** [**IEEE Trans Biomed Eng.**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sun+Y-L+and+Chen+ZH+and+2015) **62(3):900-908, 2015.**

The superconducting magnet with a high magnetic force field can levitate diamagnetic materials. In this study, a specially designed superconducting magnet with large gradient high magnetic field (LGHMF), which provides three apparent gravity levels (μg, 1 g, and 2 g), was used to study its influence on receptor activator of nuclear factor-κB ligand (RANKL)-induced osteoclast differentiation from preosteoclast cell line RAW264.7. The effects of LGHMF on the viability, nitric oxide (NO) production, morphology in RAW264.7 cells were detected by the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) method, the Griess method, and the immunofluorescence staining, respectively. The changes induced by LGHMF in osteoclast formation, mRNA expression, and bone resorption were determined by tartrate-resistant acid phosphatase staining, semiquantity PCR, and bone resorption test, respectively. The results showed that: 1) LGHMF had no lethal effect on osteoclast precursors but attenuated NO release in RAW264.7 cells. 2) Diamagnetic levitation (μg) enhanced both the formation and bone resorption capacity of osteoclast. Moreover, diamagnetic levitation up-regulated mRNA expression of RANK, Cathepsin K, MMP-9, and NFATc1, while down-regulated RunX2 in comparison with controls. Furthermore, diamagnetic levitation induced obvious morphological alterations in osteoclast, including active cytoplasmic peripheral pseudopodial expansion, formation of pedosome belt, and aggregation of actin ring. 3) Magnetic field produced by LGHMF attenuated osteoclast resorption activity. Collectively, LGHMF with combined effects has multiple effects on osteoclast, which attenuated osteoclast resorption with magnetic field, whereas promoted osteoclast differentiation with diamagnetic levitation. Therefore, these findings indicate that diamagnetic levitation could be used as a novel ground-based microgravity simulator, which facilitates bone cell research of weightlessness condition.

**(E) (VT, AE, IFR, AO)** [**Tang R**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Tang%20R%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Xu Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Xu%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Ma F**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ma%20F%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Ren J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Ren%20J%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Shen S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Shen%20S%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Du Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Du%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Hou Y**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Hou%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**,** [**Wang T**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Wang%20T%5BAuthor%5D&cauthor=true&cauthor_uid=26807660)**. Extremely low frequency magnetic fields regulate differentiation of regulatory T cells: Potential role for ROS-mediated inhibition on AKT.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/26807660) **2016 Jan 25. doi: 10.1002/bem.21954. [Epub ahead of print]**

Our previous studies showed that extremely low frequency magnetic fields (ELF-MFs) inhibited tumor growth and change proportion of splenic regulatory T cells (Treg cells). Here, we focus on the effect of ELF-MFs on lung metastatic melanoma mouse model and the regulatory mechanism of ELF-MFs on the differentiation of Treg cells. Tumor-bearing mice were exposed to sham ELF-MFs and ELF-MFs (0.4 T, 7.5 Hz) 2 h/day for 27 days. Metastatic tumor burden of lung was significantly decreased after ELF-MF treatment. Compared to the control group, expressions of matrix metalloproteinase (MMP2, MMP9) and forkhead box P3 (Foxp3) in lung nodules significantly decreased in the ELF-MF group. Moreover, in vitro, after stimulated with anti-CD3, anti-CD28 antibodies and transforming growth factor-β (TGF-β) and treated with ELF-MFs for 2 h, expression of Foxp3 in total T cells was significantly decreased. Differentiation rate of Treg cells was inhibited from 32.0% to 22.1% by ELF-MFs. Furthermore, reactive oxygen species (ROS) was increased and phospho-serine/threonine protein kinase (p-AKT) was inhibited in both T cells and Jurkat cells. ROS scavenger N-acetyl-l-cysteine reversed inhibition of AKT pathway and expression of Foxp3 from 18.6% to 26.6% in T cells. Taken together, our data show that ELF-MF exposure promoted the inhibitory effect of ROS on AKT pathway and decreased Foxp3 expression, which provides an explanation for why ELF-MF exposure can inhibit differentiation of Treg cells and enhance antitumor effect in metastatic melanoma mouse model.

**(E) (VO, CE, IX)**[**Tasset I**](http://www.ncbi.nlm.nih.gov/pubmed?term=Tasset%20I%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Medina FJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Medina%20FJ%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Jimena I**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jimena%20I%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Agüera E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ag%C3%BCera%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Gascón F**](http://www.ncbi.nlm.nih.gov/pubmed?term=Gasc%C3%B3n%20F%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Feijóo M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Feij%C3%B3o%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Sánchez-López F**](http://www.ncbi.nlm.nih.gov/pubmed?term=S%C3%A1nchez-L%C3%B3pez%20F%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Luque E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Luque%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Peña J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pe%C3%B1a%20J%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Drucker-Colín R**](http://www.ncbi.nlm.nih.gov/pubmed?term=Drucker-Col%C3%ADn%20R%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**,** [**Túnez I**](http://www.ncbi.nlm.nih.gov/pubmed?term=T%C3%BAnez%20I%5BAuthor%5D&cauthor=true&cauthor_uid=22406415)**. Neuroprotective effects of extremely low-frequency electromagnetic fields on a Huntington's disease rat model: effects on neurotrophic factors and neuronal density.** [**Neuroscience.**](http://www.ncbi.nlm.nih.gov/pubmed/22406415) **209:54-63, 2012.**

There is evidence to suggest that the neuroprotective effect of exposure of extremely low-frequency electromagnetic fields (ELF-EMF) may be due, at least in part, to the effect of these fields on neurotrophic factors levels and cell survival, leading to an improvement in behavior. This study was undertaken to investigate the neuroprotective effects of ELFEF in a rat model of 3-nitropropionic acid (3NP)-induced Huntington's disease. Behavior patterns were evaluated, and changes in neurotrophic factor, cell damage, and oxidative stress biomarker levels were monitored in Wistar rats. Rats were given 3NP over four consecutive days (20 mg/kg body weight), whereas ELFEF (60 Hz and 0.7 mT) was applied over 21 days, starting after the last injection of 3NP. Rats treated with 3NP exhibited significantly different behavior in the open field test (OFT) and the forced swim test (FST), and displayed significant differences in neurotrophic factor levels and oxidative stress biomarkers levels, together with a neuronal damage and diminished neuronal density, with respect neuronal controls. ELFEF improved neurological scores, enhanced neurotrophic factor levels, and reduced both oxidative damage and neuronal loss in 3NP-treated rats. ELFEF alleviates 3NP-induced brain injury and prevents loss of neurons in rat striatum, thus showing considerable potential as a therapeutic tool.

**(E) (VO, CE, IOD, DAO)** [**Tayefi H**](http://www.ncbi.nlm.nih.gov/pubmed?term=Tayefi%20H%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Kiray A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kiray%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Kiray M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kiray%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Ergur BU**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ergur%20BU%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Bagriyanik HA**](http://www.ncbi.nlm.nih.gov/pubmed?term=Bagriyanik%20HA%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Pekcetin C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pekcetin%20C%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Fidan M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Fidan%20M%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**,** [**Ozogul C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ozogul%20C%5BAuthor%5D&cauthor=true&cauthor_uid=22427754)**. The effects of prenatal and neonatal exposure to electromagnetic fields on infant rat myocardium.** [**Arch Med Sci.**](http://www.ncbi.nlm.nih.gov/pubmed/22427754) **6(6):837-842, 2010.**

**INTRODUCTION:** Electromagnetic fields (EMF) have adverse effects as a result of widespread use of electromagnetic energy on biological systems. The aim of this study was to investigate the effects of prenatal exposure to EMF on rat myocardium by biochemical and histopathological evaluations. **MATERIAL AND METHODS:** In this study, 10 pregnant Wistar rats were used. Half of the pregnant rats were exposed to EMF of 3 mT, and the other half to sham conditions during gestation. After parturition, rat pups in the 5 EMF-exposed litters from birth until postnatal day 20 were exposed to EMF of 3 mT for 4 h/day (EMF-exposed group, n = 30). Rat pups in sham litters from birth until postnatal day 20 were exposed to sham conditions (sham group, n= 20). **RESULTS:** In the EMF-exposed group, lipid peroxidation levels significantly increased compared to sham. Superoxide dismutase activities decreased significantly in the EMF-exposed group compared to sham. TUNEL staining showed that the number of TUNEL-positive cells increased significantly in EMF-exposed rats compared with sham. Under electron microscopy, there were mitochondrial degeneration, reduction in myofibrils, dilated sarcoplasmic reticulum and perinuclear vacuolization in EMF-exposed rats. **CONCLUSIONS:** In conclusion, the results show that prenatal exposure to EMF causes oxidative stress, apoptosis and morphological pathology in myocardium of rat pups. The results of our study indicate a probable role of free radicals in the adverse effects of prenatal exposure to EMF. Further studies are needed to demonstrate whether the EMF exposure can induce adverse effects on the myocardium.

**(E) (VO, CE, IAO)**[**Todorović D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Todorovi%C4%87%20D%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Mirčić D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mir%C4%8Di%C4%87%20D%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Ilijin L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ilijin%20L%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Mrdaković M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mrdakovi%C4%87%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Vlahović M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Vlahovi%C4%87%20M%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Prolić Z**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Proli%C4%87%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**,** [**Mataruga VP**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mataruga%20VP%5BAuthor%5D&cauthor=true&cauthor_uid=21953292)**. Effect of magnetic fields on antioxidative defense and fitness-related traits of Baculum extradentatum (insecta, phasmatodea).** [**Bioelectromagnetics.**](https://www.ncbi.nlm.nih.gov/pubmed/21953292) **33(3):265-273, 2012.**

This study aimed to determine the effect of magnetic fields on the antioxidative defense and fitness-related traits of Baculum extradentatum. Following exposure to magnetic fields, antioxidative defense (superoxide dismutase (SOD), catalase (CAT) activities, and total glutathione (GSH) content) and fitness-related traits (egg mortality, development dynamics, and mass of nymphs) were monitored in nymphs. The experimental groups were: control (kept out of influence of the magnets), a group exposed to a constant magnetic field (CMF) of 50 mT, and a group exposed to an alternating magnetic field (AMF) of 50 Hz, 6 mT. We found increased SOD and CAT activities in animals exposed to constant and AMFs, whereas GSH activity was not influenced by experimental magnetic fields. No differences were found in egg mortality between control and experimental groups. Significant differences in the time of development between the control and the CMF group were observed, as well as between the CMF and the AMF group. No differences were found in the mass of the nymphs between the three experimental groups. In conclusion, CMF and AMF have the possibility to modulate the antioxidative defense and some of the fitness-related traits in B. extradentatum.

**(NE) (VO, CE, IX)** [**Túnez I**](http://www.ncbi.nlm.nih.gov/pubmed?term=T%C3%BAnez%20I%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Drucker-Colín R**](http://www.ncbi.nlm.nih.gov/pubmed?term=Drucker-Col%C3%ADn%20R%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Jimena I**](http://www.ncbi.nlm.nih.gov/pubmed?term=Jimena%20I%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Medina FJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Medina%20FJ%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Muñoz Mdel C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Mu%C3%B1oz%20Mdel%20C%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Peña J**](http://www.ncbi.nlm.nih.gov/pubmed?term=Pe%C3%B1a%20J%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**,** [**Montilla P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Montilla%20P%5BAuthor%5D&cauthor=true&cauthor_uid=16524377)**. Transcranial magnetic stimulation attenuates cell loss and oxidative damage in the striatum induced in the 3-nitropropionic model of Huntington's disease.** [**J Neurochem.**](http://www.ncbi.nlm.nih.gov/pubmed/16524377) **97(3):619-630, 2006.**

An investigation was conducted on the effect of transcranial magnetic field stimulation (TMS) on the free radical production and neuronal cell loss produced by 3-nitropropionic acid in rats. The effects of 3-nitropropionic acid were evaluated by examining the following changes in: the quantity of hydroperoxides and total radical-trapping antioxidant potential (TRAP), lipid peroxidation products, protein carbonyl groups, reduced glutathione (GSH) content, glutathione peroxidase (GSH-Px), catalase and succinate dehydrogenase (SDH) activities; total nitrite and cell death [morphological changes, quantification of neuronal loss and lactate dehydrogenase (LDH) levels]. Our results reveal that 3-nitropropionic acid induces oxidative and nitrosative stress in the striatum, prompts cell loss and also shows that TMS prevents the harmful effects induced by the acid. In conclusion, the results show the ability of TMS to modify neuronal response to 3-nitropropionic acid.

**(NE)** **(VO, CE)** [**Türközer Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22T%C3%BCrk%C3%B6zer%20Z%22%5BAuthor%5D)**,** [**Güler G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22G%C3%BCler%20G%22%5BAuthor%5D)**,** [**Seyhan N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Seyhan%20N%22%5BAuthor%5D)**. Effects of exposure to 50 Hz electric field at different strengths on oxidative stress and antioxidant enzyme activities in the brain tissue of guinea pigs.** [**Int J Radiat Biol.**](javascript:AL_get(this,%20'jour',%20'Int%20J%20%0d%0aRadiat%20Biol.');) **84(7):581-590, 2008.**

PURPOSE: The aim of this study was to evaluate the possible effects of varied exposure to 50 Hz extremely low frequency (ELF) electric field (EF) on the lipid peroxidation levels and antioxidant enzyme activities in the brain homogenates of guinea pigs. Subjects were exposed to 2 kV/m, 2.5 kV/m, 3 kV/m, 3.5 kV/m, 4 kV/m, 4.5 kV/m and 5 kV/m electric fields for three days, 8 h a day in both vertical and horizontal directions. MATERIALS AND METHODS: Malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were measured in order to identify possible alterations in lipid peroxidation levels and antioxidant status due to electric field exposure. Xanthine oxidase (XO), myeloperoxidase (MPO) and adenosine deaminase (ADA) activities were also evaluated in the same samples. RESULTS: Although the study showed several positive but non-significant findings (p > 0.05), we did not find significant differences among all of the exposed groups and sham groups in lipid peroxidation levels and enzyme activities (p > 0.05) at all strengths and in both directions. Furthermore, the result was the same when the comparison was made between the groups in vertical directions and horizontal directions (p > 0.05). CONCLUSION: The present study observed effects of 50 Hz EF exposure on lipid peroxidation levels and antioxidant defense mechanisms but these were not statistically significant at the 95% confidence level. Further research on the effects ELF-EF exposure on lipid peroxidation levels and antioxidant defence mechanisms are warranted.

**(NE) (VT, AE)** [**Vannoni D**](http://www.ncbi.nlm.nih.gov/pubmed?term=Vannoni%20D%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Albanese A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Albanese%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Battisti E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Battisti%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Aceto E**](http://www.ncbi.nlm.nih.gov/pubmed?term=Aceto%20E%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Giglioni S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Giglioni%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Corallo C**](http://www.ncbi.nlm.nih.gov/pubmed?term=Corallo%20C%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Carta S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Carta%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Ferrata P**](http://www.ncbi.nlm.nih.gov/pubmed?term=Ferrata%20P%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Fioravanti A**](http://www.ncbi.nlm.nih.gov/pubmed?term=Fioravanti%20A%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**,** [**Giordano N**](http://www.ncbi.nlm.nih.gov/pubmed?term=Giordano%20N%5BAuthor%5D&cauthor=true&cauthor_uid=22475096)**. In vitro exposure of human osteoarthritic chondrocytes to ELF fields and new therapeutic application of musically modulated electromagnetic fields: biological evidence.** [**J Biol Regul Homeost Agents.**](http://www.ncbi.nlm.nih.gov/pubmed/22475096) **26(1):39-49, 2012.**

Osteoarthritis (OA) is the most frequently occurring rheumatic disease, caused by metabolic changes in chondrocytes, the cells that maintain cartilage. Treatment with electromagnetic fields (MF) produces benefits in patients affected by this pathology. Isolated human osteoarthritic (OA) chondrocytes were cultured in vitro under standard conditions or stimulated with IL-1beta or IGF-1, to mimic the imbalance between chondroformation and chondroresorption processes observed in OA cartilage in vivo. The cells were exposed for a specific time to extremely low frequency (ELF; 100-Hz) electromagnetic fields and to the Therapeutic Application of Musically Modulated Electromagnetic Fields (TAMMEF), which are characterized by variable frequencies, intensities, and waveforms. Using flow cytometry, we tested the effects of the different types of exposure on chondrocyte metabolism. The exposure of the cells to both systems enhances cell proliferation, does not generate reactive oxygen species, does not cause glutathione depletion or changes in mitochondrial transmembrane potential and does not induce apoptosis. This study presents scientific support to the fact that MF could influence OA chondrocytes from different points of view (viability, ROS production and apoptosis). We can conclude that both ELF and TAMMEF systems could be recommended for OA therapy and represent a valid non-pharmacological approach to the treatment of this pathology.

**(E)** **(VO, CE, DFR, DAO)** [**Vignola MB**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Vignola%20MB%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**,** [**Dávila S**](http://www.ncbi.nlm.nih.gov/pubmed/?term=D%C3%A1vila%20S%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**,** [**Cremonezzi D**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Cremonezzi%20D%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**,** [**Simes JC**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Simes%20JC%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**,** [**Palma JA**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Palma%20JA%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**,** [**Campana VR**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Campana%20VR%5BAuthor%5D&cauthor=true&cauthor_uid=22812423)**. Evaluation of inflammatory biomarkers associated with oxidative stress and histological assessment of magnetic therapy on experimental myopathy in rats.** [**Electromagn Biol Med.**](http://www.ncbi.nlm.nih.gov/pubmed/22812423) **31(4):320-332. 2012.**

The effect of pulsed electromagnetic field (PEMF) therapy, also called magnetic therapy, upon inflammatory biomarkers associated with oxidative stress plasma fibrinogen, nitric oxide (NO), L-citrulline, carbonyl groups, and superoxide dismutase (SOD) was evaluated through histological assessment, in rats with experimental myopathy. The groups studied were: (A) control (intact rats that received PEMF sham exposures); (B) rats with myopathy and sacrificed 24 h later; (C) rats with myopathy; (D) rats with myopathy and treated with PEMF; and (E) intact rats treated with PEMF. Groups A, C, D, and E were sacrificed 8 days later. Myopathy was induced by injecting 50 μl of 1% carrageenan λ (type IV) once sub-plantar. Treatment was carried out with PEMF emitting equipment with two flat solenoid disks for 8 consecutive days in groups D and E, at 20 mT and 50 Hz for 30 min/day/rat. The biomarkers were determined by spectrophotometry. The muscles (5/8) were stained with Hematoxylin-Eosin and examined by optic microscopy. Quantitative variables were statistically analyzed by the Fisher test, and categorical applying Pearson's Chi Squared test at p < 0.05 for all cases. In Groups B and C, the biomarkers were significantly increased compared to A, D, and E groups: fibrinogen (p < 0.001); NO, L-citrulline and carbonyl groups (p < 0.05); SOD (p < 0.01) as well as the percentage of area with inflammatory infiltration (p < 0.001). PEMF caused decreased levels of fibrinogen, L-citrulline, NO, SOD, and carbonyl groups and significant muscle recovery in rats with experimental myopathies.

**(NE) (VT, AE, IAO)** [**Villarini M**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Villarini%20M%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Gambelunghe A**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Gambelunghe%20A%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Giustarini D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Giustarini%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Ambrosini MV**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ambrosini%20MV%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Fatigoni C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Fatigoni%20C%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Rossi R**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Rossi%20R%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Dominici L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Dominici%20L%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Levorato S**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Levorato%20S%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Muzi G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Muzi%20G%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Piobbico D**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Piobbico%20D%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**,** [**Mariucci G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Mariucci%20G%5BAuthor%5D&cauthor=true&cauthor_uid=28985943)**. No evidence of DNA damage by co-exposure to extremely low frequency magnetic fields and aluminum on neuroblastoma cell lines.** [**Mutat Res.**](https://www.ncbi.nlm.nih.gov/pubmed/28985943) **823:11-21, 2017.**

Whether exposure to 50-60Hz extremely low frequency magnetic fields (ELF-MF) exerts neurotoxic effects is a debated issue. Analogously, the potential role of Aluminum (Al) in neurodegeneration is a matter of controversial debate. As all living organisms are exposed to ELF-MF and/or Al daily, we found investigating the early effects of co-exposure to ELF-MF and Al in SH-SY5Y and SK-N-BE-2 human neuroblastoma (NB) cells intriguing. SH-SY5Y5 and SK-N-BE-2 cells underwent exposure to 50Hz ELF-MF (0.01, 0.1 or 1mT) or AlCl3 (4 or 40μM) or co-exposure to 50Hz ELF-MF and AlCl3 for 1h continuously or 5h intermittently. The effects of the treatment were evaluated in terms of DNA damage, redox status changes and Hsp70 expression. The DNA damage was assessed by Comet assay; the cellular redox status was investigated by measuring the amount of reduced glutathione (GSH) and glutathione disulfide (GSSG) while the inducible Hsp70 expression was evaluated by western blot analysis and real-time RT-PCR. Neither exposure to ELF-MF or AlCl3 alone induced DNA damage, changes in GSH/GSSG ratio or variations in Hsp70 expression with respect to the controls in both NB cell lines. Similarly, co-exposure to ELF-MF and AlCl3 did not have any synergic toxic effects. The results of this in vitro study, which deals with the effects of co-exposure to 50Hz MF and Aluminum, seem to exclude that short-term exposure to ELF-MF in combination with Al can have harmful effects on human SH-SY5Y and SK-N-BE-2 cells.

[**Vojtísek M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Vojt%C3%ADsek%20M%22%5BAuthor%5D)**,** [**Knotková J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Knotkov%C3%A1%20J%22%5BAuthor%5D)**,** [**Kasparová L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kasparov%C3%A1%20L%22%5BAuthor%5D)**,** [**Svandová E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Svandov%C3%A1%20E%22%5BAuthor%5D)**,** [**Markvartová V**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Markvartov%C3%A1%20V%22%5BAuthor%5D)**,** [**Tůma J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22T%C5%AFma%20J%22%5BAuthor%5D)**,** [**Vozeh F**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Vozeh%20F%22%5BAuthor%5D)**,** [**Patková J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Patkov%C3%A1%20J%22%5BAuthor%5D)**. Metal, EMF, and brain energy metabolism.** [**Electromagn Biol Med.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Electromagn%20Biol%20Med.');) **28(2):188-193, 2009. (Review)**

Some implications of cooperative potential of metal ions and electromagnetic fields' radiation (EMF) in carcinogenic processes are discussed. It is known that these factors, chemical and physical individually have connections with processes of oxidative stress. Special attention was paid to possible manifestation within the brain. Therefore, the entry of a few potentially neurotoxic metals into the brain is discussed.

[**Wang H**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Wang%20H%5BAuthor%5D&cauthor=true&cauthor_uid=29057846)**,** [**Zhang X**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20X%5BAuthor%5D&cauthor=true&cauthor_uid=29057846)**. Magnetic Fields and Reactive Oxygen Species.** [**Int J Mol Sci.**](https://www.ncbi.nlm.nih.gov/pubmed/29057846) **18;18(10), 2017. (Review)**

Reactive oxygen species (ROS) ubiquitously exist in mammalian cells to participate in various cellular signaling pathways. The intracellular ROS levels are dependent on the dynamic balance between ROS generation and elimination. In this review, we summarize reported studies about the influences of magnetic fields (MFs) on ROS levels. Although in most cases, MFs increased ROS levels in human, mouse, rat cells, and tissues, there are also studies showing that ROS levels were decreased or not affected by MFs. Multiple factors could cause these discrepancies, including but not limited to MF type/intensity/frequency, exposure time and assay time-point, as well as different biological samples examined. It will be necessary to investigate the influences of different MFs on ROS in various biological samples systematically and mechanistically, which will be helpful for people to get a more complete understanding about MF-induced biological effects. In addition, reviewing the roles of MFs in ROS modulation may open up new scenarios of MF application, which could be further and more widely adopted into clinical applications, particularly in diseases that ROS have documented pathophysiological roles.

**(E)** **(VT, AE, AO, IAO, AO)** [**Wartenberg M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wartenberg%20M%22%5BAuthor%5D)**,** [**Wirtz N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wirtz%20N%22%5BAuthor%5D)**,** [**Grob A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Grob%20A%22%5BAuthor%5D)**,** [**Niedermeier W**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Niedermeier%20W%22%5BAuthor%5D)**,** [**Hescheler J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hescheler%20J%22%5BAuthor%5D)**,** [**Peters SC**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Peters%20SC%22%5BAuthor%5D)**,** [**Sauer H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sauer%20H%22%5BAuthor%5D)**. Direct current electrical fields induce apoptosis in oral mucosa cancer cells by NADPH oxidase-derived reactive oxygen species.** [**Bioelectromagnetics.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Bioelectromagnetics.');) **29(1):47-54, 2008.**

The presence of more than one dental alloy in the oral cavity often causes pathological galvanic currents and voltage resulting in superficial erosions of the oral mucosa and eventually in the emergence of oral cancer. In the present study the mechanisms of apoptosis of oral mucosa cancer cells in response to electromagnetic fields was investigated. Direct current (DC) electrical fields with field strengths between 2 and 16 V/m, applied for 24 h to UM-SCC-14-C oral mucosa cancer cells, dose-dependently resulted in decreased cell proliferation as evaluated by Ki-67 immunohistochemistry and upregulation of the cyclin-dependent kinase (CDK) inhibitors p21(cip1/waf1) and p27(kip1), which are associated with cell cycle arrest. Electrical field treatment (4 V/m, 24 h) increased apoptosis as evaluated by immunohistochemical analysis of cleaved caspase-3 and poly-(ADP-ribose)-polymerase-1 (PARP-1). Furthermore, robust reactive oxygen species (ROS) generation, increased expression of NADPH oxidase subunits as well as Hsp70 was observed. Electrical field treatment (4 V/m, 24 h) resulted in increased expression of Cu/Zn superoxide dismutase and decreased intracellular concentration of reduced glutathione (GSH), whereas the expression of catalase remained unchanged. Pre-treatment with the free radical scavenger N-acetyl cysteine (NAC) and the superoxide dismutase mimetic EUK-8 abolished caspase-3 and PARP-1 induction, suggesting that apoptosis in oral mucosa cancer cells is initated by ROS generation in response to DC electrical field treatment.

**(E)** **(VT, AE, IOD, IFR, AO)** [**Wolf FI**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wolf%20FI%22%5BAuthor%5D)**,** [**Torsello A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Torsello%20A%22%5BAuthor%5D)**,** [**Tedesco B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tedesco%20B%22%5BAuthor%5D)**,** [**Fasanella S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Fasanella%20S%22%5BAuthor%5D)**,** [**Boninsegna A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Boninsegna%20A%22%5BAuthor%5D)**,** [**D'Ascenzo M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22D%27Ascenzo%20M%22%5BAuthor%5D)**,** [**Grassi C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Grassi%20C%22%5BAuthor%5D)**,** [**Azzena GB**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Azzena%20GB%22%5BAuthor%5D)**,** [**Cittadini A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Cittadini%20A%22%5BAuthor%5D)**. 50-Hz extremely low frequency electromagnetic fields enhance cell proliferation and DNA damage: possible involvement of a redox mechanism.** [**Biochim Biophys Acta.**](javascript:AL_get(this,%20'jour',%20'Biochim%20%0d%0aBiophys%20Acta.');) **1743(1-2):120-129, 2005.**

HL-60 leukemia cells, Rat-1 fibroblasts and WI-38 diploid fibroblasts were exposed for 24-72 h to 0.5-1.0-mT 50-Hz extremely low frequency electromagnetic field (ELF-EMF). This treatment induced a dose-dependent increase in the proliferation rate of all cell types, namely about 30% increase of cell proliferation after 72-h exposure to 1.0 mT. This was accompanied by increased percentage of cells in the S-phase after 12- and 48-h exposure. The ability of ELF-EMF to induce DNA damage was also investigated by measuring DNA strand breaks. A dose-dependent increase in DNA damage was observed in all cell lines, with two peaks occurring at 24 and 72 h. A similar pattern of DNA damage was observed by measuring formation of 8-OHdG adducts. The effects of ELF-EMF on cell proliferation and DNA damage were prevented by pretreatment of cells with an antioxidant like alpha-tocopherol, suggesting that redox reactions were involved. Accordingly, Rat-1 fibroblasts that had been exposed to ELF-EMF for 3 or 24 h exhibited a significant increase in dichlorofluorescein-detectable reactive oxygen species, which was blunted by alpha-tocopherol pretreatment. Cells exposed to ELF-EMF and examined as early as 6 h after treatment initiation also exhibited modifications of NF kappa B-related proteins (p65-p50 and I kappa B alpha), which were suggestive of increased formation of p65-p50 or p65-p65 active forms, a process usually attributed to redox reactions. These results suggest that ELF-EMF influence proliferation and DNA damage in both normal and tumor cells through the action of free radical species. This information may be of value for appraising the pathophysiologic consequences of an exposure to ELF-EMF.

**(E) (VO, CE, IAO) Wu SX, Xu YQ, Di GQ, Jiang JH, Xin L, Wu TY. Influence of environmental static electric field on antioxidant enzymes activities in hepatocytes of mice. Genet Mol Res. 2016 Jul 25;15(3). doi: 10.4238/gmr.15038800.**

With the increasing voltage of direct current transmission line, the intensity of the environmental static electric field has also increased. Thus, whether static electric fields cause biological injury is an important question. In this study, the effects of chronic exposure to environmental static electric fields on some antioxidant enzymes activities in the hepatocytes of mice were investigated. Male Institute of Cancer Research mice were exposed for 35 days to environmental static electric fields of different electric field intensities of 9.2-21.85 kV/m (experiment group I, EG-I), 2.3-15.4 kV/m (experiment group II, EG-II), and 0 kV/m (control group, CG). On days 7, 14, 21, and 35 of the exposure cycle, liver homogenates were obtained and the activities of antioxidant enzymes like superoxide dismutase, glutathione S-transferase, and glutathione peroxidase were determined, as well as the concentration of malonaldehyde. The results revealed a significant increase in superoxide dismutase activity in both EG-I and EG-II on the 7th (P < 0.05) and 35th days (P < 0.01) of the exposure cycle compared to that in the control group. However, the other test indices such as glutathione S-transferase, glutathione peroxidase, and malonaldehyde showed only minimal changes during the exposure cycle. These results revealed a weak relationship between the exposure to environmental static electric fields and hepatic oxidative stress in living organisms.

**(E) (VT, AE, IFR, AO)**[**Yin C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Yin%20C%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Luo X**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Luo%20X%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Duan Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Duan%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Duan W**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Duan%20W%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Zhang H**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20H%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**He Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=He%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Sun G**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20G%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**,** [**Sun X**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sun%20X%5BAuthor%5D&cauthor=true&cauthor_uid=27470406)**. Neuroprotective effects of lotus seedpod procyanidins on extremely low frequency electromagnetic field-induced neurotoxicity in primary cultured hippocampal neurons.** [**Biomed Pharmacother.**](https://www.ncbi.nlm.nih.gov/pubmed/27470406) **82:628-639, 2016.**

The present study investigated the protective effects of lotus seedpod procyanidins (LSPCs) on extremely low frequency electromagnetic field (ELF-EMF)-induced neurotoxicity in primary cultured rat hippocampal neurons and the underlying molecular mechanism. The results of MTT, morphological observation, superoxide dismutase (SOD) and malondialdehyde (MDA) assays showed that compared with control, incubating neurons under ELF-EMF exposure significantly decreased cell viability and increased the number of apoptotic cells, whereas LSPCs evidently protected the hippocampal neurons against ELF-EMF-induced cell damage. Moreover, a certain concentration of LSPCs inhibited the elevation of intracellular reactive oxygen species (ROS) and Ca(2+) level, as well as prevented the disruption of mitochondrial membrane potential induced by ELF-EMF exposure. In addition, supplementation with LSPCs could alleviate DNA damage, block cell cycle arrest at S phase, and inhibit apoptosis and necrosis of hippocampal neurons under ELF-EMF exposure. Further study demonstrated that LSPCs up-regulated the activations of Bcl-2, Bcl-xl proteins and suppressed the expressions of Bad, Bax proteins caused by ELF-EMF exposure. In conclusion, these findings revealed that LSPCs protected against ELF-EMF-induced neurotoxicity through inhibiting oxidative stress and mitochondrial apoptotic pathway.

**(E) (VO, CE, IOD)** [**Yokus B**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yokus%20B%22%5BAuthor%5D)**,** [**Cakir DU**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Cakir%20DU%22%5BAuthor%5D)**,** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Akdag%20MZ%22%5BAuthor%5D)**,** [**Sert C**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sert%20C%22%5BAuthor%5D)**,** [**Mete N**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Mete%20N%22%5BAuthor%5D)**. Oxidative DNA damage in rats exposed to extremely low frequency electromagnetic fields. F**[**ree Radic Res.**](javascript:AL_get(this,%20'jour',%20'Free%20Radic%0d%0a%20Res.');) **39(3):317-323, 2005.**

Extremely low frequency (ELF) electromagnetic field (EMF) is thought to prolong the life of free radicals and can act as a promoter or co-promoter of cancer. 8-hydroxy-2'-deoxyguanosine (8OHdG) is one of the predominant forms of radical-induced lesions to DNA and is a potential tool to asses the cancer risk. We examined the effects of extremely low frequency electro magnetic field (ELF-EMF) (50 Hz, 0.97 mT) on 8OHdG levels in DNA and thiobarbituric acid reactive substances (TBARS) in plasma. To examine the possible time-dependent changes resulting from magnetic field, 8OHdG and TBARS were quantitated at 50 and 100 days. Our results showed that the exposure to ELF-EMF induced oxidative DNA damage and lipid peroxidation (LPO). The 8OHdG levels of exposed group (4.39+/-0.88 and 5.29+/-1.16 8OHdG/dG.10(5), respectively) were significantly higher than sham group at 50 and 100 days (3.02+/-0.63 and 3.46+/-0.38 8OHdG/dG.10(5)) (p<0.001, p<0.001). The higher TBARS levels were also detected in the exposure group both on 50 and 100 days (p<0.001, p<0.001). In addition, the extent of DNA damage and LPO would depend on the exposure time (p<0.05 and p<0.05). Our data may have important implications for the long-term exposure to ELF-EMF which may cause oxidative DNA damage.

**(E) (VO, CE, IOD)** [**Yokus B**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yokus%20B%5BAuthor%5D&cauthor=true&cauthor_uid=18979312)**,** [**Akdag MZ**](http://www.ncbi.nlm.nih.gov/pubmed?term=Akdag%20MZ%5BAuthor%5D&cauthor=true&cauthor_uid=18979312)**,** [**Dasdag S**](http://www.ncbi.nlm.nih.gov/pubmed?term=Dasdag%20S%5BAuthor%5D&cauthor=true&cauthor_uid=18979312)**,** [**Cakir DU**](http://www.ncbi.nlm.nih.gov/pubmed?term=Cakir%20DU%5BAuthor%5D&cauthor=true&cauthor_uid=18979312)**,** [**Kizil M**](http://www.ncbi.nlm.nih.gov/pubmed?term=Kizil%20M%5BAuthor%5D&cauthor=true&cauthor_uid=18979312)**. Extremely low frequency magnetic fields cause oxidative DNA damage in rats.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/18979312) **84(10):789-795, 2008.**

PURPOSE: To detect the genotoxic effects of extremely low frequency (ELF) -magnetic fields (MF) on oxidative DNA base modifications [8-hydroxyguanine (8-OH-Gua), 2,6-diamino-4-hydroxy-5-formamidopyrimidine (FapyGua) and 4,6-diamino-5-formamidopyrimidine (FapyAde)] in rat leucocytes, measured following exposure to ELF-MF. MATERIALS AND METHODS: After exposure to ELF-MF (50 Hz, 100 and 500 microT, for 2 hours/day during 10 months), DNA was extracted, and measurement of DNA lesions was achieved by gas chromatography/mass spectrometry (GC/MS) and liquid chromatography/mass spectrometry (LC/MS). RESULTS: Levels of FapyAde, FapyGua and 8OHdG in DNA were increased by both 100 microT and 500 microT ELF-MF as compared to a cage-control and a sham group; however, statistical significance was observed only in the group exposed to 100 microT. CONCLUSION: This is the first study to report that ELF-MF exposure generates oxidatively induced DNA base modifications which are mutagenic in mammalian cells, such as FapyGua, FapyAde and 8-OH-Gua, in vivo. This may explain previous studies showing DNA damage and genomic instability. These findings support the hypothesis that chronic exposure to 50-Hz MF may be potentially genotoxic. However, the intensity of ELF-MF has an important influence on the extent of DNA damage.

**(NE)** **(VT, AE)** [**Yoon HE**](http://www.ncbi.nlm.nih.gov/pubmed?term=Yoon%20HE%5BAuthor%5D&cauthor=true&cauthor_uid=24467330)**,** [**Lee JS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20JS%5BAuthor%5D&cauthor=true&cauthor_uid=24467330)**,** [**Myung SH**](http://www.ncbi.nlm.nih.gov/pubmed?term=Myung%20SH%5BAuthor%5D&cauthor=true&cauthor_uid=24467330)**,** [**Lee YS**](http://www.ncbi.nlm.nih.gov/pubmed?term=Lee%20YS%5BAuthor%5D&cauthor=true&cauthor_uid=24467330)**. Increased γ-H2AX by exposure to a 60-Hz magnetic fields combined with ionizing radiation, but not hydrogen peroxide, in non-tumorigenic human cell lines.** [**Int J Radiat Biol.**](http://www.ncbi.nlm.nih.gov/pubmed/24467330) **90(4):291-298, 2014.**

Abstract Purpose: Genotoxic effects have been considered the gold standard to determine if an environmental factor is a carcinogen, but the currently available data for extremely low frequency time-varying magnetic fields (ELF-MFs) remain controversial. As an environmental stimulus, the effect of ELF-MF on cellular DNA may be subtle. Therefore, a more sensitive method and systematic research strategy are warranted to evaluate genotoxicity. Materials and methods: We investigated the effect of ELF-MFs in combination with ionizing radiation (IR) or H2O2 on the DNA damage response of expression of phosphorylated H2AX (γ-H2AX) and production of γ-H2AX foci in non-tumorigenic human cell systems consisting of human lung fibroblast WI38 cells and human lung epithelial L132 cells. Results: Exposure to a 60-Hz, 2 mT ELF-MFs for 6 h produced increased γ-H2AX expression, as well as γ-H2AX foci production, a common DNA double-strand break (DSB) marker. However, exposure to a 1 mT ELF-MFs did not have the same effect. Moreover, 2 mT ELF-MFs exposure potentiated the expression of γ-H2AX and γ-H2AX foci production when combined with IR, but not when combined with H2O2. Conclusions: ELF-MFs could affect the DNA damage response and, in combination with different stimuli, provide different effects on γ-H2AX.

**(NE)** **(VO, AE, IX)** [**Yoshikawa T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Yoshikawa%20T%22%5BAuthor%5D)**,** [**Tanigawa M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tanigawa%20M%22%5BAuthor%5D)**,** [**Tanigawa T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Tanigawa%20T%22%5BAuthor%5D)**,** [**Imai A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Imai%20A%22%5BAuthor%5D)**,** [**Hongo H**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Hongo%20H%22%5BAuthor%5D)**,** [**Kondo M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Kondo%20M%22%5BAuthor%5D)**. Enhancement of nitric oxide generation by low frequency electromagnetic field.** [**Pathophysiology.**](javascript:AL_get(this,%20'jour',%20%0d%0a'Pathophysiology.');) **7(2):131-135, 2000.**

Oxidative stress is implicated in the intracellular signal transduction pathways for nitric oxide synthase (NOS) induction. The electromagnetic field (EMF) is believed to increase the free radical lifespan [S. Roy, Y. Noda, V. Eckert, M.G. Traber, A. Mori, R. Liburdy, L. Packer, The phorbol 12-myristate 13-acetate (PMA)-induced oxidative burst in rat peritoneal neutrophils is increased by a 0.1 mT (60 Hz) magnetic field, FEBS Lett. 376 (1995) 164-6; F.S. Prato, M. Kavaliers, J.J. Carson, Behavioural evidence that magnetic field effects in the land snail, Cepaea nemoralis, might not depend on magnetite or induced electric currents, Bioelectromagnetics 17 (1996) 123-30; A.L. Hulbert, J. Metcalfe, R. Hesketh, Biological response to electromagnetic fields, FASEB 12 (1998) 395-420]. We tested the effects of EMF on endotoxin induced nitric oxide (NO) generation in vivo. Male BALB/C mice were injected with lipopolysaccharide (LPS) intraperitoneously (i.p.), followed by the exposure to EMF (0.1 mT, 60 Hz). Five hours and 30 min after the LPS administration, mice were administered with a NO spin trap, ferrous N-methyl-D-glucaminedithiocarbamate (MGD-Fe). Thirty minutes later, mice were sacrificed, and their livers were removed. The results were compared to three control groups: group A (LPS (-) EMF(-)); group B (LPS(-) EMF(+)); group C (LPS(+) EMF(-)). The ESR spectra of obtained livers were examined at room temperature. Three-line spectra of NO adducts were observed in the livers of all groups. In groups A and B very weak signals were observed, but in groups C and D strong spectra were observed. The signal intensity of the NO adducts in Group D was also significantly stronger than that in Group C. EMF itself did not induce NO generation, however, it enhanced LPS induced NO generation in vivo.

[**Zhang J**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhang%20J%5BAuthor%5D&cauthor=true&cauthor_uid=24556024)**,** [**Ding C**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ding%20C%5BAuthor%5D&cauthor=true&cauthor_uid=24556024)**,** [**Ren L**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Ren%20L%5BAuthor%5D&cauthor=true&cauthor_uid=24556024)**,** [**Zhou Y**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Zhou%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=24556024)**,** [**Shang P**](https://www.ncbi.nlm.nih.gov/pubmed/?term=Shang%20P%5BAuthor%5D&cauthor=true&cauthor_uid=24556024)**. The effects of static magnetic fields on bone.** [**Prog Biophys Mol Biol.**](https://www.ncbi.nlm.nih.gov/pubmed/24556024) **114(3):146-152, 2014. (review)**

All the living beings live and evolve under geomagnetic field (25-65 μT). Besides, opportunities for human exposed to different intensities of static magnetic fields (SMF) in the workplace have increased progressively, such SMF range from weak magnetic field (<1 mT), moderate SMF (1 mT-1 T) to high SMF (>1 T). Given this, numerous scientific studies focus on the health effects and have demonstrated that certain magnetic fields have positive influence on our skeleton systems. Therefore, SMF is considered as a potential physical therapy to improve bone healing and keep bones healthy nowadays. Here, we review the mechanisms of effects of SMF on bone tissue, ranging from physical interactions, animal studies to cellular studies.

**(E)** **(VT, AE, IFR)** [**Zhao G**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zhao%20G%22%5BAuthor%5D)**,** [**Chen S**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Chen%20S%22%5BAuthor%5D)**,** [**Wang L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wang%20L%22%5BAuthor%5D)**,** [**Zhao Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zhao%20Y%22%5BAuthor%5D)**,** [**Wang J**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wang%20J%22%5BAuthor%5D)**,** [**Wang X**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wang%20X%22%5BAuthor%5D)**,** [**Zhang W**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zhang%20W%22%5BAuthor%5D)**,** [**Wu R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wu%20R%22%5BAuthor%5D)**,** [**Wu L**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wu%20L%22%5BAuthor%5D)**,** [**Wu Y**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Wu%20Y%22%5BAuthor%5D)**,** [**Xu A**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Xu%20A%22%5BAuthor%5D)**. Cellular ATP content was decreased by a homogeneous 8.5 T static magnetic field exposure: role of reactive oxygen species.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/21225886##) **32(2):94-101, 2011.**

The literature on the impact of strong static magnetic fields (SMF) on human health is vast and contradictory. The present study focused on the cellular effects of strong homogeneous SMF in human-hamster hybrid (A(L) ) cells, mitochondria-deficient (ρ(0) A(L) ) cells, and double-strand break (DSB) repair-deficient (XRS-5) cells. Adenosine triphosphate (ATP) content was significantly decreased in A(L) cells exposed to 8.5 Tesla (T) but not 1 or 4 T SMF for either 3 or 5 h. In addition, ATP content significantly decreased in the two deficient cell lines exposed to 8.5 T SMF for 3 h. With further incubation of 12 or 24 h without SMF exposure, ATP content could retrieve to the control level in the A(L) cells but not ρ(0) A(L) and XRS-5 cells. Under a fluorescence reader, the levels of reactive oxygen species (ROS) in the three cell lines were significantly increased by exposure to 8.5 T SMF for 3 h. Concurrent treatment with ROS inhibitor, DMSO, dramatically suppressed the ATP content in exposed A(L) cells. However, the CD59 mutation frequency and the cell cycle distribution were not significantly affected by exposure to 8.5 T SMF for 3 h. Our results indicated that the cellular ATP content was reduced by 8.5 T SMF for 3 h exposure, which was partially mediated by mitochondria and the DNA DSB repair process. Moreover, ROS were involved in the process of the cellular perturbations from the SMF.

**(E)** **(VT, AE, IOD, IAO)** [**Zwirska-Korczala K**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Zwirska-Korczala%20K%22%5BAuthor%5D)**,** [**Adamczyk-Sowa M**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Adamczyk-Sowa%20M%22%5BAuthor%5D)**,** [**Polaniak R**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Polaniak%20R%22%5BAuthor%5D)**,** [**Sowa P**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Sowa%20P%22%5BAuthor%5D)**,** [**Birkner E**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Birkner%20E%22%5BAuthor%5D)**,** [**Drzazga Z**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Drzazga%20Z%22%5BAuthor%5D)**,** [**Brzozowski T**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Brzozowski%20T%22%5BAuthor%5D)**,** [**Konturek SJ**](http://www.ncbi.nlm.nih.gov/pubmed?term=%22Konturek%20SJ%22%5BAuthor%5D)**. Influence of extremely-low-frequency magnetic field on antioxidative melatonin properties in AT478 murine squamous cell carcinoma culture.** [**Biol Trace Elem Res.**](javascript:AL_get(this,%20'jour',%20'Biol%20Trace%0d%0a%20Elem%20Res.');) **102(1-3):227-243, 2004.**

Effects of melatonin, extremely-low-frequency magnetic field (ELF-MF), and their combination on AT478 murine squamous cell carcinoma line were studied. Manganese superoxide dismutase (MnSOD), copper-zinc superoxide dismutase (Cu/ZnSOD), and glutathione peroxidase (GSH-Px) were used as markers of cells antioxidative status, and malondialdehyde (MDA) level was used as a marker of lipid peroxidation. After melatonin treatment, antioxidative enzyme activities were increased and MDA level was decreased. Application of ELF-MF on treated cells caused an increase of both superoxide dismutases activity and MDA level, but influence of ELF-MF on GSH-Px activity was negligible. All enzyme activity in culture medium containing melatonin (10(-3), 10(-4), 10(-5) M) after exposure to ELF-MF were significantly diminished compared to cells treated only with melatonin. Also MDA levels after combined treatment with melatonin and ELF-MF were significantly decreased. Observed changes were statistically significant (p<0.05). These results strongly suggest that ELF-MF attenuates antioxidative actions of melatonin on cellular level.

**(E) (VT, AE, IX, LI)** [**Zmyslony M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zmyslony%20M%5BAuthor%5D&cauthor=true&cauthor_uid=15515035)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rajkowska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=15515035)**,** [**Mamrot P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Mamrot%20P%5BAuthor%5D&cauthor=true&cauthor_uid=15515035)**,** [**Politanski P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Politanski%20P%5BAuthor%5D&cauthor=true&cauthor_uid=15515035)**,** [**Jajte J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Jajte%20J%5BAuthor%5D&cauthor=true&cauthor_uid=15515035)**. The effect of weak 50 Hz magnetic fields on the number of free oxygen radicals in rat lymphocytes in vitro.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/15515035) **25(8):607-612, 2004a.**

The aim of the work was verification of the hypothesis that weak power frequency (50 Hz) magnetic fields (MF) affected the number of free oxygen radicals in living biological cells and that these changes could be qualitatively explained by the radical pair mechanism. The experiments were performed on rat lymphocytes. One-hour exposure to 50 Hz MF at 20, 40, or 200 microT flux densities was performed inside a pair of Helmholtz coils with axis along or crosswise to the Earth's static MF. Iron ions (FeCl2) were used as a stimulator of the oxidation processes. Oxygen radicals were measured by fluorimetry using a DCF-DA fluorescent probe. Only in the lymphocytes exposed at 40 microT MF directed along the Earth's static MF there was a decrease of fluorescence in relation to non-exposed samples. Our observation seems to confirm the hypothesis that low level power frequency MF affects oxidative processes which occur in living biological cells and that this effect can be explained by the radical pair mechanism.[See comment in PubMed Commons below](http://www.ncbi.nlm.nih.gov/pubmed/15376237#comments)



**(E) (VT, AE, IX, LI)** [**Zmyślony M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Zmy%C5%9Blony%20M%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Palus J**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Palus%20J%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Dziubałtowska E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Dziuba%C5%82towska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Politański P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Polita%C5%84ski%20P%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Mamrot P**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Mamrot%20P%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Rajkowska E**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Rajkowska%20E%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**,** [**Kameduła M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Kamedu%C5%82a%20M%5BAuthor%5D&cauthor=true&cauthor_uid=15376237)**. Effects of in vitro exposure to power frequency magnetic fields on UV-induced DNA damage of rat lymphocytes.** [**Bioelectromagnetics.**](http://www.ncbi.nlm.nih.gov/pubmed/15376237) **25(7):560-562, 2004b.**

The mechanisms of biological effects of 50/60 Hz (power frequency) magnetic fields (MF) are still poorly understood. There are a number of studies indicating that MF affect biochemical processes in which free radicals are involved, such as the biological objects' response to ultraviolet radiation (UVA). Therefore, the present study was aimed to assess the effect of 50 Hz MFs on the oxidative deterioration of DNA in rat lymphocytes irradiated in vitro by UVA. UVA radiation (150 J/m2) was applied for 5 min for all groups and 50 Hz MF (40 microT rms) exposure was applied for some of the groups for 5 or 60 min. The level of DNA damage was assessed using the alkaline comet assay, the fluorescence microscope, and image analysis. It has been found that the 1 h exposure to MF caused an evident increase in all parameters consistent with damaged DNA. This suggest that MF affects the radical pairs generated during the oxidative or enzymatic processes of DNA repair.

Appendix A: a brief description of cellular oxidative processes

Activity in the mitochondrial electron transport chain leads to the production of superoxide (O2**.**-) which can be converted to hydrogen peroxide (H2O2) by the enzyme superoxide dismutate (SOD). H2O2 can be further converted by the iron-dependent Fenton reaction into the potent hydroxyl radical (OH**.**). In the cytoplasm, nitric oxide (NO**.**) is generated by various forms of nitric oxide synthase (NOS) by conversion of L-arginine to L-citrulline. NO**.** reacts with O2**.**- to generate the potent oxidant peroxynitrite (ONOO-). O2**.**- can also be produced by NOS by transfer of electron from NADPH to O2. Other enzymatic processes, such as cytochrome P450,also generate ROS in normal cellular activities.

Major anti-oxidative processes in cells include catalase/peroxidase that converts O2**.**- to H2O and O2. In the process, glutathione (GSH) is oxidized to glutathione disulfide (GSSG). GSSG is reduced back to GSH by the enzyme glutathione reductase with the conversion of NADPH to NADP. GSH and NADPH are the most common electron donors participated in cellular anti-oxidation processes. ONOO- is decomposed by peroxiredoxin and glutathione peroxidase into less potent nitrogen free radicals (NO3**.**/NO2**.**).

ROS react with cellular macromolecules. The most common form of DNA oxidative damage is the formation of hydroxylated bases. 8-hydroxy-2’-deoxyguanosine (8-OHdG) is generally used an index of oxidative DNA damage. ROS react with lipids to produce lipid peroxyl radicals and lipid hydroperoxides. Lipid peroxyl can subsequently form malondialdehyde (MDA), which is commonly used as an index of oxidative lipid damage. Lipid radicals can diffuse through membrane leading to protein oxidation and formation of DNA-MDA adduct. Oxidative lipid damages affect the structure and function of cell membrane. ROS attack proteins directly and indirectly. Protein carbonyl is a form of protein oxidative damage. Changes in protein structure lead to alteration in enzymatic activities, particularly, damage to membrane transport proteins leads to ionic imbalance such as intracellular concentrations of calcium and potassium. Oxidative stress could also cause changes in regulation of transcription factors in cells, e.g., the Nrf2 antioxidant pathway.