http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=15729859&dopt=Abstract

J Dermatol. 2004 Nov;31(11):878-83.

Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin.

Ayata A, **Mollaoglu H**, **Yilmaz HR**, **Akturk O**, **Ozguner F**, **Altuntas I**.

Department of Pediatrics, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey.

Most mobile phones emit 900 MHz of radiation that is mainly absorbed by the external organs. The effects of 900 MHz of radiation on fibrosis, lipid peroxidation, and anti-oxidant enzymes and the ameliorating effects of melatonin (Mel) were evaluated in rat skin. Thirty Wistar-Albino rats were used in the study. The experimental groups were the control group, the irradiated group (IR), and the irradiated+Mel treated group (IR+Mel). A dose of 900 MHz, 2 W radiation was applied to the IR group every day for 10 days (30 min/day). The IR+Mel group received 10 mg/kg/day melatonin in tap water for 10 days before the irradiation. At the end of the 10th day, a skin specimen was excised from the thoracoabdominal area. The levels of malondialdehyde (MDA) and hydroxypyroline and the activities of superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), and catalase (CAT) were studied in the skin samples. MDA and hydroxyproline levels and activities of CAT and GSH-Px were increased significantly in the IR group compared to the control group ($p<0.05$) and decreased significantly in the IR+Mel group ($p<0.05$). SOD activity was decreased significantly in the IR group and this decrease was not prevented by the Mel treatment. These results suggest that rats irradiated with 900 MHz suffer from increased fibrosis and lipid peroxidation (LPO). Mel treatment can reduce the fibrosis and LPO caused by radiation.

PMID: 15729859 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=9816579&query_hl=35&itool=pubmed_docsum

Med Pr. 1998;49(4):333-9.

[Platelets oxygen metabolism in women and men in different age groups]

Article in Polish]

Buczynski A, **Kocur J**, **Stopczyk D**, **Dziedziczak-Buczynska M**, **Kowalski W**.

Zakladu Medycyny Zapobiegawczej, Instytutu OOZW Wojskowej Akademii Medycznej, Lodzi.

The aim of the study as to assess the influence of the physiological aging process on the platelet cell metabolism in middle-aged people. A group of 17 healthy women (aged 47-59 years), and a group of healthy men (aged 45-60 years) were examined. The control group was composed of healthy women aged 19-25 years, and healthy men aged 19-27 years. The activity of hyperoxide dismutase, catalase, glutathione peroxidase and the concentration of malonyldialdehyde were determined in platelets. In comparison to the control group, a significant decrease in the activity of hyperoxide dismutase and glutathione peroxidase as well as an enhanced concentration of malonyldialdehyde were observed in the group studied. Moreover, a diminished catalase activity was noted in platelets of men, while in women there were no significant changes. The study indicated that disorders in the function of thrombocytes, an excessive generation of oxygen free radicals, and impaired mechanisms of cellular antioxidative defence accelerate atherosclerosis and aging process. Therefore, it is necessary to cover middle-aged people, particularly those occupationally exposed to factors affecting defence mechanisms, with adequate preventive programmes.

PMID: 9816579 [PubMed - indexed for MEDLINE]

Dun-Xian Tan, Lucien C. Manchester, Maria P. Terron, Luis J. Flores and Russel J. Reiter.
« MINI REVIEW - One molecule, many derivatives : A never-ending interaction of melatonin with reactive oxygen and nitrogen species ? ». J. Pineal Res. 2006
Doi:10.1111/j.1600-079X.2006.00407.x

Full study in pdf format.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=11989597&query_hl=19&itool=pubmed_docsum

Tumori. 2001 Nov-Dec;87(6):417-22.

Ginkgo biloba extract (EGb 761) modulates bleomycin-induced acute lung injury in rats.

El-Khatib AS, **Moustafa AM**, **Abdel-Aziz AA**, **Al-Shabanah OA**, **El-Kashef HA**.

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The effect of Ginkgo biloba extract (EGb 761) on bleomycin (BLM)-induced acute lung injury was studied in rats. The responsiveness of isolated pulmonary arterial rings to 5-hydroxytryptamine (5-HT) as well as the levels of some relevant biochemical markers in the lung tissue were taken as evidence for the acute lung injury. BLM was given intraperitoneally at a dose of 15 mg/kg/day for five consecutive days. It was found that BLM treatment attenuated the vasoconstrictor effect of 5-HT on the isolated pulmonary arteries. In lung tissues BLM also elevated the level of lipid peroxides and enhanced the activity of glutathione peroxidase. On the other hand, the level of glutathione and the activity of alkaline phosphatase were reduced. Body weight, lung weight and tissue glutathione-S-transferase activity were, however, not altered. Oral administration of EGb 761 at a dose of 100 mg/kg/day for five consecutive days did not alter any of the chosen biochemical parameters in the lung tissue except for a slight reduction in alkaline phosphatase activity. However, treatment with EGb 761 reduced the responsiveness of the pulmonary artery to 5-HT. Administration of EGb 761 (100 mg/kg/day; po) two hours prior to BLM (15 mg/kg/day; ip), for five consecutive days blunted the occurrence of further reduction in the vasoconstrictor response of the pulmonary artery to 5-HT. Furthermore, EGb 761 tended to normalize BLM-induced alterations in the measured biochemical markers in the lung tissue. The apparent modulatory influence of EGb 761 on BLM-induced acute lung injury stems, at least in part, from its beneficial free radical scavenging properties that provide the extract with antioxidant activity.

PMID: 11989597 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16715528&dopt=Abstract

Bioelectromagnetics. 2006 Sep;27(6):487-93.

Oxidative stress effects on the central nervous system of rats after acute exposure to ultra high frequency electromagnetic fields.

Ferreira AR, **Bonatto F**, **de Bittencourt Pasquali MA**, **Polydoro M**, **Dal-Pizzol F**, **Fernandez C**, **de Salles AA**, **Moreira JC**.

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Mobile telephones and their base stations are an important source of ultra high frequency electromagnetic fields (UHF-EMFs; 800-1800 MHz) and their utilization is increasing all over the world. Epidemiological studies have suggested that low energy UHF-EMFs may have biological effects, such as changes in oxidative metabolism after exposure. Therefore, we have investigated the effect of acute UHF-EMF exposure on non-enzymatic antioxidant defense and lipid and protein oxidative damage in the rat frontal cortex and hippocampus. We have used malondialdehyde (MDA) and carbonyl assays to assess lipid and protein oxidative damages, respectively. No changes in lipid and protein damage, and also in non-enzymatic defense were found in frontal cortex or hippocampus. These results suggest that acute UHF-EMF exposure is not able to produce detectable oxidative stress in rats from any age tested. However, more tests using a longer period of exposure and evaluating other tissues are necessary to ensure that there is no health risk associated with the use of mobile phones.

PMID: 16715528 [PubMed - in process]

Aim of study (according to author)

To study the effect of acute ultrahigh frequency electromagnetic field exposure on non-enzymatic antioxidant defense and lipid and protein oxidative damage in the rat frontal cortex and hippocampus.

Background/further details:

A total of 31 rats were divided by age into three groups (30, 80, and 210 days) and exposed.

Endpoint

- effects on the neurological system: oxidative metabolism in the brain

Exposure

General: analog mobile phone

Field characteristics	Parameters
834 MHz continuous wave (CW) exposure duration: repeated daily exposure, 7.5 h per day for 6 days	electric field strength: 23 V/m effective value (to 35.7 V/m) magnetic field strength: 65 mA/m (to 90 mA/m) power flux density: 0.15 mW/cm² mean value (to 0.35 mW/cm²) SAR: 0.41 W/kg (to 0.98 W/kg; for 30 day old rats) SAR: 0.36 W/kg (to 0.86 W/kg; for 70 day old rats)

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16037959&query_hl=24&itool=pubmed_docsum

Bioelectromagnetics. 2005 Oct;26(7):589-94.

Effects of a 50 Hz electric field on plasma lipid peroxide level and antioxidant activity in rats.

Harakawa S, **Inoue N**, **Hori T**, **Tochio K**, **Kariya T**, **Takahashi K**, **Doge F**, **Suzuki H**, **Nagasawa H**.

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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. (c) 2005 Wiley-Liss, Inc.

PMID: 16037959 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=14734207&dopt=Abstract

Clin Chim Acta. 2004 Feb;340(1-2):153-62.

Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain.

Ilhan A, **Gurel A**, **Armutcu F**, **Kamisli S**, **Iraz M**, **Akyol O**, **Ozen S**.

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BACKGROUND: The widespread use of mobile phones (MP) in recent years has raised the research activities in many countries to determine the consequences of exposure to the low-intensity electromagnetic radiation (EMR) of mobile phones. Since several experimental studies suggest a role of reactive oxygen species (ROS) in EMR-induced oxidative damage in tissues, in this study, we investigated the effect of Ginkgo biloba (Gb) on MP-induced oxidative damage in brain tissue of rats.

METHODS: Rats (EMR+) were exposed to 900 MHz EMR from MP for 7 days (1 h/day). In the EMR+Gb groups, rats were exposed to EMR and pretreated with Gb. Control and Gb-administrated groups were produced by turning off the mobile phone while the animals were in the same exposure conditions. Subsequently, oxidative stress markers and pathological changes in brain tissue were examined for each groups.

RESULTS: Oxidative damage was evident by the: (i) increase in malondialdehyde (MDA) and nitric oxide (NO) levels in brain tissue, (ii) decrease in brain superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) activities and (iii) increase in brain xanthine oxidase (XO) and adenosine deaminase (ADA) activities. These alterations were prevented by Gb treatment. Furthermore, Gb prevented the MP-induced cellular injury in brain tissue histopathologically.

CONCLUSION: Reactive oxygen species may play a role in the mechanism that has been proposed to explain the biological side effects of MP, and Gb prevents the MP-induced oxidative stress to preserve antioxidant enzymes activity in brain tissue.

PMID: 14734207 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=12415560&dopt=Abstract

Cell Biochem Funct. 2002 Dec;20(4):279-83.

Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits.

Irmak MK, **Fadillioglu E**, **Gulec M**, **Erdogan H**, **Yagmurca M**, **Akyol O**.

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The number of reports on the effects induced by electromagnetic radiation (EMR) in various cellular systems is still increasing. Until now no satisfactory mechanism has been proposed to explain the biological effects of this radiation. Oxygen free radicals may play a role in mechanisms of adverse effects of EMR. This study was undertaken to investigate the influence of electromagnetic radiation of a digital GSM mobile telephone (900 MHz) on oxidant and antioxidant levels in rabbits. Adenosine deaminase, xanthine oxidase, catalase, myeloperoxidase, superoxide dismutase (SOD) and glutathione peroxidase activities as well as nitric oxide (NO) and malondialdehyde levels were measured in sera and brains of EMR-exposed and sham-exposed rabbits. Serum SOD activity increased, and serum NO levels decreased in EMR-exposed animals compared to the sham group. Other parameters were not changed in either group. This finding may indicate the possible role of increased oxidative stress in the pathophysiology of adverse effect of EMR. Decreased NO levels may also suggest a probable role of NO in the adverse effect. Copyright 2002 John Wiley & Sons, Ltd.

PMID: 12415560 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=10437415&query_hl=7&itool=pubmed_docsum

Vojnosanit Pregl. 1999 Mar-Apr;56(2):113-7.

Oxidative stress in the thalamus of Wistar rats treated with 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine.

Jovanovic MD, **Ninkovic M**, **Malicevic Z**, **Mihajlovic R**, **Dukic M**, **Vasiljevic I**, **Jelenkovic A**, **Jovicic A**.

Military Medical Academy, Institute for Medical Research, Belgrade.

Experimental parkinsonism was induced in adult Wistar rats by selective nigrostriatal neurotoxine, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) in a single dose of 0.09 g/kg, by unilateral intrastriatal application using stereotaxic instrument. Control group included rats treated with 0.9% saline solution in the same manner. Animals were sacrificed by decapitation seven days after the treatment. Total glutathione was measured in the crude mitochondrial fraction of thalamus and striatum. Total glutathione content, as a measure of reduced cell atmosphere, was mutually decreased in the thalamus and striatum of MPTP-treated animals, compared to controls: thalamus ipsi- = 24.8 +/- 3.11, contralateral = 26.81 +/- 5.31; striatum ipsi- = 19.96 +/- 4.13, contralateral = 17.3 +/- 4.09 nmol/mg prot. Mutually depleted glutathione content in the thalamus and contralateral striatum, the structures distant from ipsilateral treated striatum, could indicate on spatial propagation of oxidative stress, not only in the selective vulnerable dopaminergic nigrostriatal neurons, but in the structures included in the motor and cognitive loops of basal ganglia.

PMID: 10437415 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16898263&query_hl=2&itool=pubmed_docsum

Toxicol Ind Health. 2006 Jun;22(5):211-6.

Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain.

Koylu H, **Mollaoglu H**, **Ozguner F**, **Nazyroglu M**, **Delibab N**.

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Microwaves (MW) from cellular phones may affect biological systems by increasing free radicals, which may enhance lipid peroxidation levels of the brain, thus leading to oxidative damage. Melatonin is synthesized in and secreted by the pineal gland at night and exhibits anti-oxidant properties. Several studies suggest that supplementation with anti-oxidant can influence MW-induced brain damage. The present study was designed to determine the effects of MW on the brain lipid peroxidation system, and the possible protective effects of melatonin on brain degeneration induced by MW. Twenty-eight Sprague-Dawley male rats were randomly divided into three groups as follows: (1) sham-operated control group (N = 8); (2) study 900-MHz MW-exposed group (N = 8); and (3) 900-MHz MW-exposed+melatonin (100 microg/kg sc before daily MW exposure treated group) (N = 10). Cortex brain and hippocampus tissues were removed to study the levels of lipid peroxidation as malonyl dialdehyde. The levels of lipid peroxidation in the brain cortex and hippocampus increased in the MW group compared with the control group, although the levels in the hippocampus were decreased by MW+melatonin administration. The brain cortex lipid peroxidation levels were unaffected by melatonin treatment. We conclude that melatonin may prevent MW-induced oxidative changes in the hippocampus by strengthening the anti-oxidant defense system, by reducing oxidative stress products.

PMID: 16898263 [PubMed - in process]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=14732250&dopt=Abstract

J Photochem Photobiol B. 2004 Jan 23;73(1-2):43-8.

Effects of extremely low frequency magnetic field on the antioxidant defense system in mouse brain: a chemiluminescence study.

Lee BC, **Johng HM**, **Lim JK**, **Jeong JH**, **Baik KY**, **Nam TJ**, **Lee JH**, **Kim J**, **Sohn UD**, **Yoon G**, **Shin S**, **Soh KS**.

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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.

PMID: 14732250 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=11516912&query_hl=19&itool=pubmed_docsum

J Pharm Biomed Anal. 2001 Nov;26(4):605-8.

Effects of acute exposure to the radiofrequency fields of cellular phones on plasma lipid peroxide and antioxidase activities in human erythrocytes.

Moustafa YM, **Moustafa RM**, **Belacy A**, **Abou-El-Ela SH**, **Ali FM**.

Department of Pharmacology and Toxicology, Faculty of Pharmacy, Suez Canal University, Ismailia 41522, Egypt.

Radiofrequency fields of cellular phones may affect biological systems by increasing free radicals, which appear mainly to enhance lipid peroxidation, and by changing the antioxidase activities of human blood thus leading to oxidative stress. To test this, we have investigated the effect of acute exposure to radiofrequency fields of commercially available cellular phones on some parameters indicative of oxidative stress in 12 healthy adult male volunteers. Each volunteer put the phone in his pocket in standby position with the keypad facing the body. The parameters measured were lipid peroxide and the activities of superoxide dismutase (SOD), total glutathione peroxidase (GSH-Px) and catalase. The results obtained showed that the plasma level of lipid peroxide was significantly increased after 1, 2 and 4 h of exposure to radiofrequency fields of the cellular phone in standby position. Moreover, the activities of SOD and GSH-Px in human erythrocytes showed significant reduction while the activity of catalase in human erythrocytes did not decrease significantly. These results indicate that acute exposure to radiofrequency fields of commercially available cellular phones may modulate the oxidative stress of free radicals by enhancing lipid peroxidation and reducing the activation of SOD and GSH-Px, which are free radical scavengers. Therefore, these results support the interaction of radiofrequency fields of cellular phones with biological systems.

PMID: 11516912 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16318001&query_hl=1

Vopr Kurortol Fizioter Lech Fiz Kult. 2005 Sep-Oct;(5):17-20.

[Influence of (460 MHz) electromagnetic fields on the induced lipid peroxidation in the structures of visual analyzer and hypothalamus in experimental animals]

[Article in Russian]

Musaev AV, **Ismailova LF**, **Gadzhiev AM**.

Changes in the intensity of ascorbate- and NADPN2-dependent induced lipid peroxidation (LPO) were studied in exposure of the visual analyzer and hypothalamus of 3- and 12-month-old rats to radiation with microwaves of high and low intensity. The exposure to microwaves of high intensity stimulated basal LPO but suppressed activity of LPO-inducing systems. This suggests disturbances in the activity of different sources of active oxygen forms. Microwaves of low intensity activated systems of induced LPO. This is accompanied with synchronous activity of the antioxidant defense system maintaining a normal oxidation-reduction balance of the cell. The conclusion is that, depending on their intensity, microwaves can be either beneficial to health or be a factor of oxidative stress.

PMID: 16318001 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=11039304&query_hl=7&itool=pubmed_docsum

Vojnosanit Pregl. 2000 May-Jun;57(3):257-63.

Effects of nerve growth factor on antioxidative system in the thalamus of MPTP treated Wistar rats.

Ninkovic MB, **Jovanovic MD**, **Malicevic Z**, **Dukic M**, **Jelenkovic A**, **Mihajlovic R**, **Vasiljevic I**, **Jovicic A**.

Military Medical Academy, Institute for Medical Research, Belgrade.

1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-induced parkinsonism is one of the most useful models for the study of that disease. It has been suggested that MPTP-induced neurotoxicity may involve the production of reactive oxygen species. MPTP was applied intracerebrally, unilaterally, in the striatum in single dose of 0.09 g/kg b.w. The second group was treated both with MPTP and nerve growth factor (NGF) in dose of 7 ng/ml. NGF was applied immediately after the neurotoxin. Control group was treated with 0.9% saline solution in the same manner. Animals were decapitated 7 days after the treatment. In the group treated with MPTP, the activity of superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) was decreased in ipsilateral thalamus, compared to control values as well as to the contralateral thalamus. In the same structures superoxide anion production was increased, compared to controls. Following the application of both MPTP and NGF, the activity of SOD and GSH-Px remained on control values, while the superoxide anion content was decreased, compared to controls. These results indicate a temporal and spatial propagation of oxidative stress and spread protective effects of NGF on the thalamus, the structure that is distant, but very tightly connected with striatum, the place of direct neurotoxic damage.

PMID: 11039304 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=17191663&query_hl=7&itool=pubmed_docsum

Acta Physiol Hung. 2006 Dec;93(4):315-23.

Oxidative stress in the rats brain capillaries in sepsis--the influence of 7-nitroindazole.

Ninkovic M, **Malicevic I**, **Jelenkovic A**, **Jovanovic DM**, **Dukic M**, **Vasiljevic I**.

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As a part of blood-brain barrier, brain capillaries participate in pathophysiological events during systemic inflammation. We investigated the effects of 7-nitroindazole (7-NI), selective neuronal nitric oxide synthase (NOS) inhibitor, to oxidative status (OS) of brain capillaries. Adult Wistar rats were randomized at groups: control group (CG) (sham operated), sepsis group (GS) (cecal ligation and perforation with inoculation of Escherichia coli (ATCC 25922), 7-NI group (G7-NI), (30 mg/kg b/w i.p.) and 7-NI + sepsis group (G7-NIS), (7-NI was applied 30 minutes before operation). Lipid peroxidation index (LPI), nitrite concentration, superoxide dismutase (SOD) activity and superoxide anion (O2*-) content were determined 3, 6, 24 and 48 hour in each group. Cerebral capillaries were separated from non-vascular brain tissue using sucrose gradient. Compared to controls, LPI, nitrite and O2*- increased at SG. In the G7-NIS, LPI reached control values at the 24th and 48th hour, while nitrite were decreased at the 3rd and 24th hour, compared to controls. In the same group, O2*- decreased at the 3rd, 6th and 24th hour, although SOD showed variable activity. The systematic nNOS inhibition with 7-NI forces OS on early terms of sepsis, but lately it contributes to the normalization of OS in cerebral capillaries.

PMID: 17191663 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15950073&dopt=Abstract

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

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

Oktem F, **Ozguner F**, **Mollaoglu H**, **Koyu A**, **Uz E**.

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

PMID: 15950073 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=16132682&dopt=Abstract

Mol Cell Biochem. 2005 Aug;276(1-2):31-7.

Comparative analysis of the protective effects of melatonin and caffeic acid phenethyl ester (CAPE) on mobile phone-induced renal impairment in rat.

Ozguner F, **Oktem F**, **Armagan A**, **Yilmaz R**, **Koyu A**, **Demirel R**, **Vural H**, **Uz E**.

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Melatonin and caffeic acid phenethyl ester (CAPE), a component of honeybee propolis, were recently found to be potent free radical scavengers and antioxidants. There are a number of reports on the effects induced by electromagnetic radiation (EMR) in various cellular systems. Mechanisms of adverse effects of EMR indicate that reactive oxygen species may play a role in the biological effects of this radiation. The present study was carried out to compare the protective effects of melatonin and CAPE against 900 MHz EMR emitted mobile phone-induced renal tubular injury. Melatonin was administered whereas CAPE was given for 10 days before the exposure. Urinary N-acetyl-beta-D-glucosaminidase (NAG, a marker of renal tubular injury) and malondialdehyde (MDA, an index of lipid peroxidation), were used as markers of oxidative stress-induced renal impairment in rats exposed to EMR. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status in renal tissue. Urinary NAG and renal MDA were increased in EMR exposed rats while both melatonin and CAPE caused a significant reduction in the levels of these parameters. Likewise, renal SOD and GSH-Px activities were decreased in EMR exposed animals while melatonin caused a significant increase in the activities of these antioxidant enzymes but CAPE did not. Melatonin caused a significant decrease in urinary NAG activity and MDA levels which were increased because of EMR exposure. CAPE also reduced elevated MDA levels in EMR exposed renal tissue, but the effect of melatonin was more potent than that of CAPE. Furthermore, treatment of EMR exposed rats with melatonin increased activities of SOD and GSH-Px to higher levels than those of control rats.

In conclusion, melatonin and CAPE prevent renal tubular injury by reducing oxidative stress and protect the kidney from oxidative damage induced by 900 MHz mobile phone. Nevertheless, melatonin seems to be a more potent antioxidant compared with CAPE in kidney. (Mol Cell Biochem 276: 31-37, 2005).

PMID: 16132682 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=16132717&dopt=Abstract

Mol Cell Biochem. 2005 Sep;277(1-2):73-80.

A novel antioxidant agent caffeic acid phenethyl ester prevents long-term mobile phone exposure-induced renal impairment in rat. Prognostic value of malondialdehyde, N-acetyl-beta-D-glucosaminidase and nitric oxide determination.

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Caffeic acid phenethyl ester (CAPE), a flavonoid like compound, is one of the major components of honeybee propolis. It has been used in folk medicine for many years in Middle East countries. It was found to be a potent free radical scavenger and antioxidant recently. The aim of this study was to examine long-term applied 900 MHz emitting mobile phone-induced oxidative stress that promotes production of reactive oxygen species (ROS) and, was to investigate the role of CAPE on kidney tissue against the possible electromagnetic radiation (EMR)-induced renal impairment in rats. In particular, the ROS such as superoxide and nitric oxide (NO) may contribute to the pathophysiology of EMR-induced renal impairment. Malondialdehyde (MDA, an index of lipid peroxidation) levels, urinary N-acetyl-beta-D-glucosaminidase (NAG, a marker of renal tubular injury) and nitric oxide (NO, an oxidant product) levels were used as markers of oxidative stress-induced renal impairment and the success of CAPE treatment. The activities of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) in renal tissue were determined to evaluate the changes of antioxidant status. The rats used in the study were randomly grouped (10 each) as follows: i) Control group (without stress and EMR), ii) Sham-operated rats stayed without exposure to EMR (exposure device off), iii) Rats exposed to 900 MHz EMR (EMR group), and iv) A 900 MHz EMR exposed + CAPE treated group (EMR + CAPE group). In the EMR exposed group, while tissue MDA, NO levels and urinary NAG levels increased ($p < 0.0001$), the activities of SOD, CAT, and GSH-Px in renal tissue were reduced ($p < 0.001$). CAPE treatment reversed these effects as well ($p < 0.0001$, $p < 0.001$ respectively).

In conclusion, the increase in NO and MDA levels of renal tissue, and in urinary NAG with the decrease in renal SOD, CAT, GSH-Px activities demonstrate the role of oxidative mechanisms in 900 MHz mobile phone-induced renal tissue damage, and CAPE, via its free radical scavenging and antioxidant properties, ameliorates oxidative renal damage. These results strongly suggest that CAPE exhibits a protective effect on mobile phone-induced and free radical mediated oxidative renal impairment in rats.

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cmd=Retrieve&db=PubMed&list_uids=16342473&dopt=Abstract

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

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

Ozguner F, **Altinbas A**, **Ozaydin M**, **Dogan A**, **Vural H**, **Kisioglu AN**, **Cesur G**, **Yildirim NG**.

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

In this study, the increased levels of MDA and NO and the decreased levels of myocardial SOD, CAT and GSH-Px activities demonstrate the role of oxidative mechanisms in 900 MHz mobile phone-induced heart tissue damage, and CAPE, via its free radical scavenging and antioxidant properties, ameliorates oxidative heart injury. These results show that CAPE exhibits a protective effect on mobile phone-induced and free radical mediated oxidative heart impairment in rats.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16317515&query_hl=1

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Protective effects of melatonin and caffeic acid phenethyl ester against retinal oxidative stress in long-term use of mobile phone: a comparative study.

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There are numerous reports on the effects of electromagnetic radiation (EMR) in various cellular systems. Melatonin and caffeic acid phenethyl ester (CAPE), a component of honeybee propolis, were recently found to be potent free radical scavengers and antioxidants. Mechanisms of adverse effects of EMR indicate that reactive oxygen species may play a role in the biological effects of this radiation. The present study was carried out to compare the efficacy of the protective effects of melatonin and CAPE against retinal oxidative stress due to long-term exposure to 900 MHz EMR emitting mobile phones. Melatonin and CAPE were administered daily for 60 days to the rats prior to their EMR exposure during our study. Nitric oxide (NO, an oxidant product) levels and malondialdehyde (MDA, an index of lipid peroxidation), were used as markers of retinal oxidative stress in rats following to use of EMR. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) activities were studied to evaluate the changes of antioxidant status in retinal tissue. Retinal levels of NO and MDA increased in EMR exposed rats while both melatonin and CAPE caused a significant reduction in the levels of NO and MDA. Likewise, retinal SOD, GSH-Px and CAT activities decreased in EMR exposed animals while melatonin and CAPE caused a significant increase in the activities of these antioxidant enzymes. Treatment of EMR exposed rats with melatonin or CAPE increased the activities of SOD, GSH-Px and CAT to higher levels than those of control rats. In conclusion, melatonin and CAPE reduce retinal oxidative stress after long-term exposure to 900 MHz emitting mobile phone. Nevertheless, there was no statistically significant difference between the efficacies of these two antioxidants against to EMR induced oxidative stress in rat retina. The difference was in only GSH-Px activity in rat retina. Melatonin stimulated the retinal GSH-Px activity more efficiently than CAPE did.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=12006087&dopt=Abstract

Biochem J. 2002 Aug 15;366(Pt 1):203-9.

Selenium deficiency increases the expression of inducible nitric oxide synthase in RAW 264.7 macrophages: role of nuclear factor-kappaB in up-regulation.

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The inducible isoform of nitric oxide synthase (iNOS) is implicated in atherosclerosis, malignancy, rheumatoid arthritis, tissue and reperfusion injuries. A key determinant of the pro-oxidant versus protective effects of NO is the underlying redox status of the tissue. Selenoproteins, such as glutathione peroxidases (GPxs) and thioredoxin reductases, are key components of cellular defence and promote optimal antioxidant/oxidant balance. In this study, we have investigated the relationship between Se status, iNOS expression and NO production in Se-deficient and Se-supplemented RAW 264.7 macrophage cell lines. The cellular GPx activity, a measure of Se status, was 17-fold lower in Se-deficient RAW 264.7 cells and the total cellular oxidative tone, as assessed by flow cytometry with 2',7'-dichlorodihydrofluorescein diacetate, was higher in the Se-deficient cells than the Se-supplemented cells. Upon lipopolysaccharide (LPS) stimulation of these cells in culture, we found significantly higher iNOS transcript and protein expression levels with an increase in NO production in Se-deficient RAW 264.7 cells than the Se-supplemented cells. Electrophoretic mobility-shift assays, nuclear factor-kappaB (NF-kappaB)-luciferase reporter assays and Western blot analyses indicate that the increased expression of iNOS in Se deficiency could be due to an increased activation and consequent nuclear localization of the redox-sensitive transcription factor NF-kappaB.

These results suggest an inverse relationship between cellular Se status and iNOS expression in LPS-stimulated RAW 264.7 cells and provide evidence for the beneficial effects of dietary Se supplementation in the prevention and/or treatment of oxidative-stress-mediated inflammatory diseases.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=12474410&query_hl=35&itool=pubmed_docsum

Med Pr. 2002;53(4):311-4.

[Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA) --in vitro study]

[Article in Polish]

Stopczyk D, **Gnitecki W**, **Buczynski A**, **Markuszewski L**, **Buczynski J**.

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The aim of the study was to assess in vitro the effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA) in human blood platelets. The suspension of blood platelets was exposed to the electromagnetic field with the frequency of 900 MHz for 1, 3, 5, and 7 min. Our studies demonstrated that microwaves produced by mobile phones significantly depleted SOD-1 activity after 1, 5, and 7 min of exposure and increased after 3 min in comparison with the control test. There was a significant increase in the concentration of MDA after 1, 5, and 7 min and decrease after 3 min of exposure as compared with the control test. On the grounds of our results we conclude that oxidative stress after exposure to microwaves may be the reason for many adverse changes in cells and may cause a number of systemic disturbances in the human body.

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Ann Acad Med Stetin. 2005;51 Suppl 1:125-8.

[Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1)--in vitro researches]

[Article in Polish]

Stopczyk D, **Gnitecki W**, **Buczynski A**, **Kowalski W**, **Buczynska M**, **Kroc A**.

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The aim of the paper was to estimate in vitro the effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) in human blood platelets. Suspension of blood platelets exposed to the electromagnetic field of 900 MHz frequency for 1, 3, 5, 7 minutes. Our studies demonstrated that microwaves produced by mobiles significally depleted the activity of SOD-1 after 1, 5, 7 min. of exposition and increased after 3 min. nn comparison wit control test. On the ground of our results we conclude that oxidative stress after exposition to microwaves can be the reason of many disadvantageous changes in cells and may cause many systemic consequences in human organism.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=12552762&query_hl=28&itool=pubmed_docsum

Sheng Wu Yi Xue Gong Cheng Xue Za Zhi. 1999 Sep;16(3):359-61.

[The influence of pulsed magnetic fields on SOD's activity and MDA value in metabolism of mice]

[Article in Chinese]

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This experiment aimed to investigate the influence of pulsed extremely-low-frequency-magnetic fields on free radical metabolism of mice. Thirty-two mice were randomly divided into four groups and were exposed to 0 T, 0.25 T, 0.34 T and 0.64 T intensity pulsed magnetic fields of 20 Hz for 40 min. The 0 T exposed group was the control group. The free radical metabolism, SOD's activity and MDA, of mice were measured respectively. The result showed the SOD's activity and MDA of the 0.34 T exposed group were both significantly lower ($P < 0.01$, $P < 0.05$) than that of the control group, while the 0.25 T and 0.64 T exposed groups were not significantly different from the control group. This demonstrates that the biological effect of pulsed extremely-low-frequency magnetic fields on free radical metabolism of mice varies and depends on the intensity of the fields. The "window" effect may exist.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16954120&query_hl=3&itool=pubmed_docsum

Electromagn Biol Med. 2006;25(3):177-88.

GSM base station electromagnetic radiation and oxidative stress in rats.

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The ever increasing use of cellular phones and the increasing number of associated base stations are becoming a widespread source of nonionizing electromagnetic radiation. Some biological effects are likely to occur even at low-level EM fields. In this study, a gigahertz transverse electromagnetic (GTEM) cell was used as an exposure environment for plane wave conditions of far-field free space EM field propagation at the GSM base transceiver station (BTS) frequency of 945 MHz, and effects on oxidative stress in rats were investigated. When EM fields at a power density of 3.67 W/m2 (specific absorption rate = 11.3 mW/kg), which is well below current exposure limits, were applied, MDA (malondialdehyde) level was found to increase and GSH (reduced glutathione) concentration was found to decrease significantly ($p < 0.0001$). Additionally, there was a less significant ($p = 0.0190$) increase in SOD (superoxide dismutase) activity under EM exposure.

PMID: 16954120 [PubMed - in process]

http://www.buergerwelle.de/pdf/microwave_and_oxidative_stress_studies.htm
L'étude complète par lien n'est pas complète

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Effect of extremely low frequency of electromagnetic fields on cell proliferation, antioxidative enzyme activities and lipid peroxidation in 3T3-L1 preadipocytes - an in vitro study.

Zwirska-Korczala K, **Jochem J** , **Adamczyk-Sowa M** , **Sowa P** , **Polaniak R** , **Birkner E** , **Latocha M** , **Pilc K** , **Suchanek R** .

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The exposure to extremely low frequency electromagnetic field (ELF-MF, frequencies less than 200-300 Hz) can alter the transcription and translation of genes, influence the cell proliferation rate and affect enzyme activities. Moreover, the hypothesis that ELF-MF increases free oxygen metabolites generation has been proposed. Since recent in vivo studies suggest that electric and magnetic fields are able to affect adipose cells metabolism. The aim of the study was to examine the effects of ELF-MF (frequency of basic impulse 180-195 Hz, induction 120 muT) on cell proliferation, antioxidative enzyme activities and malondialdehyde (MDA) concentration in 3T3-L1 preadipocyte cell culture. We found that ELF-MF application lasting 36 minutes daily failed to influence cell count after 24h and 48 h of incubation. After 24 h, in the ELF-MF treated group, manganese- and copper-zinc-containing superoxide dismutase (MnSOD and Cu/ZnSOD) isoenzymes media activities were decreased, catalase activity was increased, whereas there were no significant differences in glutathione peroxidase (GSH-Px) and glutathione reductase (GSSG-Rd) activities in comparison to the control. After 48 h of incubation, all enzyme activities were reduced, except for GSSG-Rd, in which no changes were noticed. MDA concentration at 24 h after incubation with the exposure to ELF-MF was significantly higher in comparison to the control, without ELF-MF. After 48 h of incubation, MDA levels were significantly lower in both groups with no differences between the groups without and with ELF-MF. We conclude that ELF-MF influences antioxidative enzyme activities and increases lipid peroxidation in 3T3-L1 preadipocyte cultures.