

1800 MHz radio-frequency electromagnetic radiation induces oxidative stress in rat liver, kidney and brain tissues

Mehmet Berköz^{1*}, Badel Arslan², Metin Yıldırım³, Nurcan Aras², Serap Yalın³, Ülkü Çömelekoğlu⁴

¹Department of Pharmaceutical Biotechnology, Faculty of Pharmacy, Van Yuzuncu Yil University, Van, Turkey

²Department of Medical Biology, Faculty of Medicine, Mersin University, Mersin, Turkey

³Department of Biochemistry, Faculty of Pharmacy, Mersin University, Mersin, Turkey

⁴Department of Biophysics, Faculty of Medicine, Mersin University, Mersin, Turkey

ABSTRACT

Radio-frequency electromagnetic radiation (RF-EMR) represents one of the environmental factors that influence animal organisms to stress. In this study we determined the oxidative stress parameters from rat liver, kidney and brain tissues that were exposed to chronic 1800 MHz RF-EMR. Our study was designed in 3 groups as 9 animals in each group. These are; control, sham and RF-EMR exposed group. The control group was not exposed to any procedure; sham group was housed in the same room under the same conditions with equal time period, except that the generator was turned off. RF-EMR exposed group was subjected to 1800 MHz RF-EMR emitted from the signal generator for 2 h per day for eight weeks. All animals that completed the experimental period were sacrificed and liver, kidney and brain tissues of all rats were isolated for analyzing malondialdehyde (MDA), nitric oxide (NO) and reduced glutathione (GSH) levels and superoxide dismutase (SOD) and catalase activities. Liver, kidney and brain MDA and NO levels were higher and GSH level and SOD and catalase activities were significantly lower in RF-exposed group than control and sham groups ($p < 0.001$). No significant difference was observed in terms of tissue MDA, GSH and NO levels and SOD and catalase activities between control and sham groups in each tissue. The results of our study show that RF-EMR may act as an environmental stressor and cause oxidative and nitrosative damage in liver, kidney and brain tissues.

Key Words: Radio-frequency electromagnetic radiation, 1800 MHz, oxidative stress, liver, kidney, brain

Introduction

Mobile phone use has boomed out in recent years with an estimated 4.6 billion subscriptions globally. It has become an integral part of modern telecommunications. In many countries, over 50 % of the population uses mobile phones, and in some parts of the world, mobile phones are the most reliable or the only phones available. These gadgets use radiofrequency waves for transmitting data. The frequency band used for this varies from country to country (1). Generally the GSM (Global System for Mobile Communications) mobile phones use 900/1800 MHz frequency bands (2). Unlike ionizing radiation such as X-rays or gamma rays, radiofrequency fields can neither break chemical bonds nor cause ionization in the human body. Although this fact is true and well known to everyone, the interaction between this radiation and biological system is a major concern (3). The possible health effects of mobile phone

radio-frequency electromagnetic radiation (RF-EMR) are bothering the whole world because of the uncontrollable growth of the industry over the globe (4).

In particular, the average age of beginning mobile phone use has decreased rapidly to elementary school age, and durations of exposure to electromagnetic fields (EMFs) are also increasing (1,2). EMFs influence metabolic processes in the human body and exert various biological effects on cells through a range of mechanisms. EMF disrupts the chemical structures of tissue since a high degree electromagnetic energy absorption can change the electric current in the body. As a result of this exposure, the functions of organs are affected (5). Electric fields exert an oscillatory force on every free ion on the both sides of the plasma membrane and cause them to cross it. This movement of ions causes deterioration in the ion channels on the membrane, biochemical changes

*Corresponding Author: Mehmet BERKÖZ, Department of Pharmaceutical Biotechnology, Faculty of Pharmacy, Van Yuzuncu Yil University, Van, Turkey, Tel: 0 536 7197124, Fax: 0 432 2251514 E-mail: mehmet_berkoz@yahoo.com

Received: 23.11.2017, Accepted: 08.01.2018

in the membrane and consequently impairment of all cellular functions (6,7).

EMFs are reported to cause a rise in levels of oxygen free radicals in an experimental environment in plants and humans. Cellular damage at different levels (lipids, proteins, nucleic acids) lead to tissue injury caused by excessive generation of free radicals/reactive oxygen species (ROS) and attenuation of the antioxidants, which is named as oxidative stress (8,9). ROS damage to lipids lead to toxic aldehyde formation such as malondialdehyde (MDA). Lipid damage also induces disorder in cellular membrane functions. Superoxide dismutase (SOD) and catalase are low-molecular-weight antioxidants that directly defend the body against ROS (10,11).

Although some authors pointed out the possibility that RF-EMR radiation could affect the concentration of free radicals through the radical pair recombination thus increase the oxidant stress on cells or tissues, studies on some specific tissues are very limited. So we aimed to investigate whether 1800 MHz RF-EMR induces oxidative damage on liver, kidney and brain tissues by assessing the malondialdehyde (MDA), reduced glutathione (GSH) and nitric oxide (NO) levels and SOD and catalase activities on these tissues.

Materials and Methods

Animal Experiments

In this study, 27 female Wistar-Albino rats weighing 200-250 g (8-12 weeks) obtained from Mersin University Faculty of Medicine Experimental Research Center were used. Rats were matched to laboratory conditions one week prior and food and water consumption was not restricted. Rats were housed in a room with a temperature of $23 \pm 1^\circ\text{C}$ and a humidity of $55 \pm 10\%$ throughout the experiment. The ventilation of the laboratory where the animals were housed was provided by means of a window type aspirator and the 12 hour light to 12 hour darkness was observed for illumination. Rats were given medication every morning between 09⁰⁰-10⁰⁰ a.m. Rats were not fed for 12 hours prior to sacrifice. Ethical approval of the study was obtained from Mersin University Ethical Commission for Animal Experiments.

Experimental Groups

Our study was designed in 3 groups as 9 animals in each group. These are; control, sham and RF-EMR exposed group. The control group was not exposed to any procedure; sham group was housed in the same room under the same conditions with equal time period, except that the

generator was turned off. RF-EMR exposed group was subjected to 1800 MHz RF-EMR emitted from the signal generator for 2 h per day for eight weeks.

Electromagnetic Exposure

The electromagnetic radiation exposure system was designed by Mersin University Faculty of Medicine Department of Biophysics according to previous studies. 1800 MHz GSM simulator (GSM-1800 CW2; Adapazari, Turkey) was used for the RF-EMR. The electric field measurements of RF-EMR exposure were measured two times using Portable Electrical Field Meter (PMM 8053); first at the beginning and second at the end of the study. The electric field measurement methods of RF-EMR exposed from 1800 MHz GSM simulator was based on those used by Akar et al (12). The study group was placed inside the restrainer, and the rats were exposed to 6.8 ± 0.1 V/m RF-EMR for 2 h per day for eight weeks with a permitted power level of 1 W. Based on these data, the whole body specific absorption rate (SAR) was calculated to be 0.06 W/Kg using the SAR calculator.

Tissue Preparation

All animals that completed the experimental period were anesthetized with ketamine (200 mg / kg/ i.p.). Sacrification of the anesthetized rats was performed by cardiac puncture. Liver, kidney and brain tissues of all rats were isolated and the tissues were cleaned by passing through 0.9 % NaCl solution. Until the study day, all tissues were stored in deep freezing (-70°C).

On the day of the study, 0.1 g of tissue sample was taken from the deep-freeze-dried tissue samples and 1900 μl of cold phosphate buffer (PBS) was added onto it. All tissues were homogenized in a cold jacket for 3 minutes at 16,000 rpm. The homogenate was transferred to the tubes without increasing the heat and the tubes were numbered. The homogenates obtained were centrifuged at 3000 rpm for 10 minutes at $+4^\circ\text{C}$. The supernatants obtained after this procedure were used for lipid peroxidation determination and antioxidant enzyme assays.

Biochemical assays

MDA Level: MDA reacts with thiobarbituric acid (TBA), giving a spectroscopically readable final product at 532 nm. MDA levels were expressed as nmol/mg protein using the extinction coefficient value of $1.56 \times 10^5 \text{ M}^{-1} \cdot \text{cm}^{-1}$ (13).

SOD Activity: The principle of measurement of SOD enzyme activity, which accelerates the aquatic and molecular oxygen dismutation of

endogenous and exogenous sources of toxic superoxide radicals generated during the production of oxidative pathway energy, is based on the spectrophotometric measurement of superoxide radicals which are released by xanthine oxidase in the presence of xanthine in the presence of nitroblue tetrazolium (NBT) at 560 nm according to Sun et al (14).

Catalase Activity: The activity of the enzyme catalase was analyzed according to Aebi method (15), measuring the initial rate of H₂O₂ decomposition at 240 nm. Catalase activity was expressed as U/mg protein.

GSH level: GSH analysis was performed according to the method reported by Beutler et al (16). In this method, all proteins that do not carry the sulfhydryl in the tissue homogenates are precipitated. In the obtained clear liquid, the yellow complex formed by 5,5'-Dithiobis (2-nitrobenzoic acid) (DTNB) and sulfhydryl groups is measured colorimetrically at a wavelength of 412 nm.

NO level: Serum nitric oxide is rapidly converted to nitrate and nitrite in aqueous solutions. Hence, for accurate assay of the total nitric oxide, both nitrate and nitrite levels must be determined. Serum nitrate is chemically reduced to nitrite by granulated cadmium. Griess reagent reacts with total nitrite, and forms a coloured complex. The intensity of the colour is proportional to the concentration of the nitrite in the sample, which can be measured spectrophotometrically (17).

Determination of protein content: In the alkaline solution, a copper-protein complex is formed. This complex reduces the phosphomolybdate phosphotungstate reagent (Folin-Ciocalteus-Phenol Reagent) and forms a dark blue color. The resulting darkness is directly proportional to the protein concentration in the medium (18).

Statistical analysis:

SPSS 15.0 statistical program was used for all statistical evaluations. One way analysis of variance test (ANOVA) with Tukey's post-hoc test was used to determine statistical difference between groups. Reported data were presented as mean±standard deviation and values having p value <0.05 were considered to be significant.

Results

MDA level of liver and kidney tissues were significantly higher in RF-EMR exposed group in

comparison with control and sham groups (p<0.001). The most dramatic increase in the MDA level of the tissues was observed in brain tissue. MDA level of brain was significantly higher in RF-EMR exposed group than control and sham groups (p<0.0001). No significant difference was observed in terms of tissue MDA level between control and sham groups in each tissue (p>0.05) (Fig. 1).

Liver NO level was significantly higher in RF-exposed group than control and sham groups (p<0.001). Kidney and brain NO levels were quite high in RF-EMR exposed group than control and sham groups (p<0.0001). No statistically significant difference was seen between control and sham groups in each tissue (p>0.05) (Fig. 2).

Liver, kidney and brain catalase activities were significantly lower in RF-exposed group than control and sham groups (p<0.001). No significant difference was observed in terms of tissue catalase activities between control and sham groups in each tissue (p>0.05) (Fig. 3).

Liver, kidney and brain SOD activities were significantly lower in RF-exposed group than control and sham groups (p<0.001). There was no statistically significant difference between control and sham groups in each tissue (p>0.05) (Fig. 4).

Liver, kidney and brain GSH levels were statistically significantly lower in RF-exposed group than control and sham groups (p<0.001). No significant difference was observed in terms of tissue GSH levels between control and sham groups in each tissue (p>0.05) (Fig. 5).

Discussion

Exposure to RF-EMR can damage biological tissues by inducing changes, which can be explained in terms of thermal or non-thermal mechanisms. Thermal effects can occur with the conversion and absorption of heat by the body's electromagnetic energy. Increased body temperature is stabilized and alleviated by blood circulation (8). Although non-thermal effects do not raise the body temperature sufficiently to impair the structure of tissues, their effect is mediated by generation of reactive oxygen species (ROS). ROS are involved in various cellular functions and can be essential or extremely toxic to cellular homeostasis. Their cytotoxic effects derive from peroxidation of membrane phospholipids. This creates a change in the conductivity of the membrane and loss of membrane integrity (8,9).

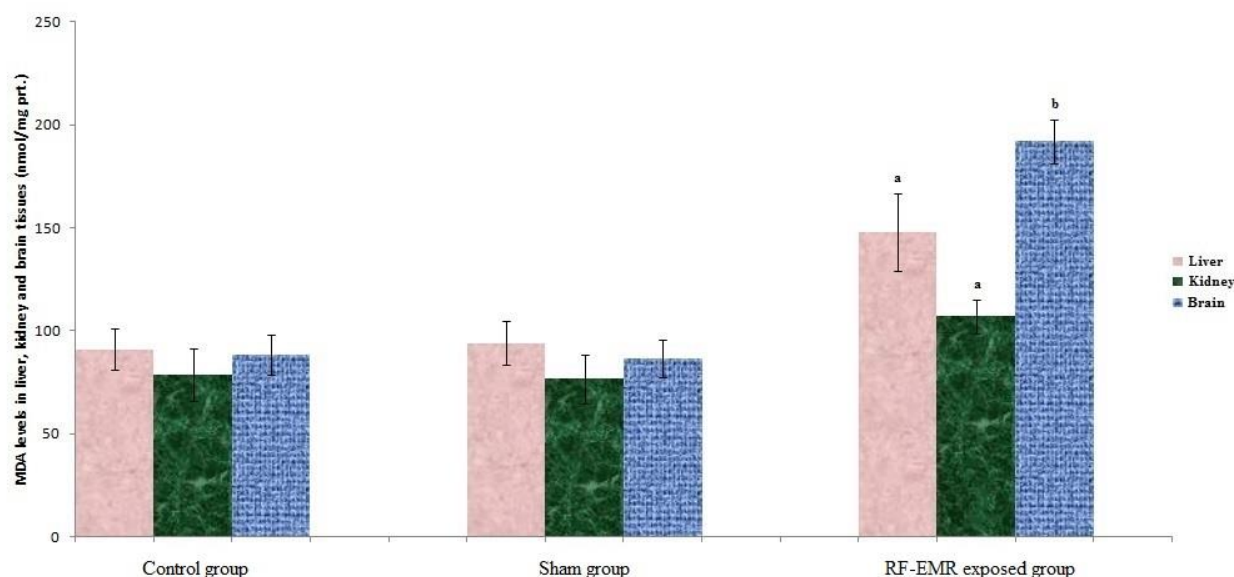


Fig. 1. MDA levels in liver, kidney and brain tissues

^a Significantly higher than the control and sham groups ($p < 0.001$)

^b Significantly higher than the control and sham groups ($p < 0.0001$)

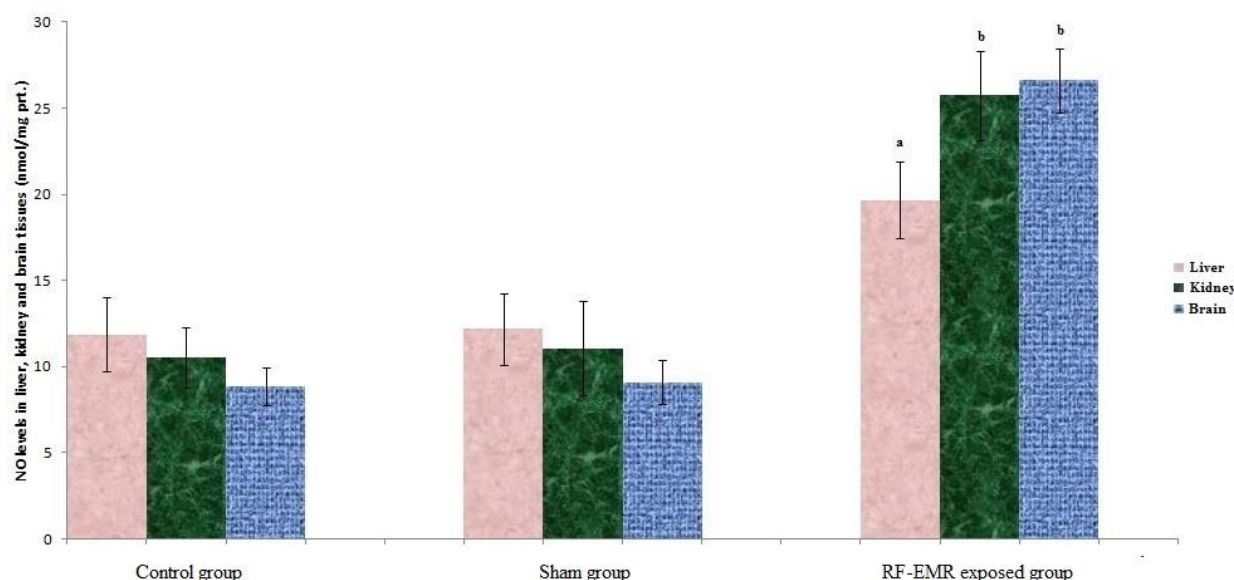


Fig. 2. NO levels in liver, kidney and brain tissues

^a Significantly higher than the control and sham groups ($p < 0.001$)

^b Significantly higher than the control and sham groups ($p < 0.0001$)

Increasing evidence has indicated that RF-EMR has the potential of inducing oxidative stress in biological systems via free radicals by enhancing lipid peroxidation and reducing antioxidant levels (11). In our study, chronic exposure to 1800 MHz RF-EMR can cause lipid peroxidation, nitrogenic stress and antioxidant suppression in liver, kidney and brain tissues.

MDA and NO levels of liver increased significantly in RF-EMR group compared to sham and control groups. However, SOD and catalase activities and GSH level showed significant reduction in liver

tissues. The results of our study verified the hypothesis that RF-EMR radiation may cause oxidative damage in liver tissue. The changes in MDA, NO and GSH levels and SOD and catalase activities in exposed rats reflected pathophysiological effects of electromagnetic field in liver tissue. Oxidative stress may accelerate the peroxidation reactions of lipids in liver (11). It has been reported that patients diagnosed with degenerative liver diseases have higher level of lipoperoxide in their liver tissues and several forms of liver diseases have been shown to be associated with

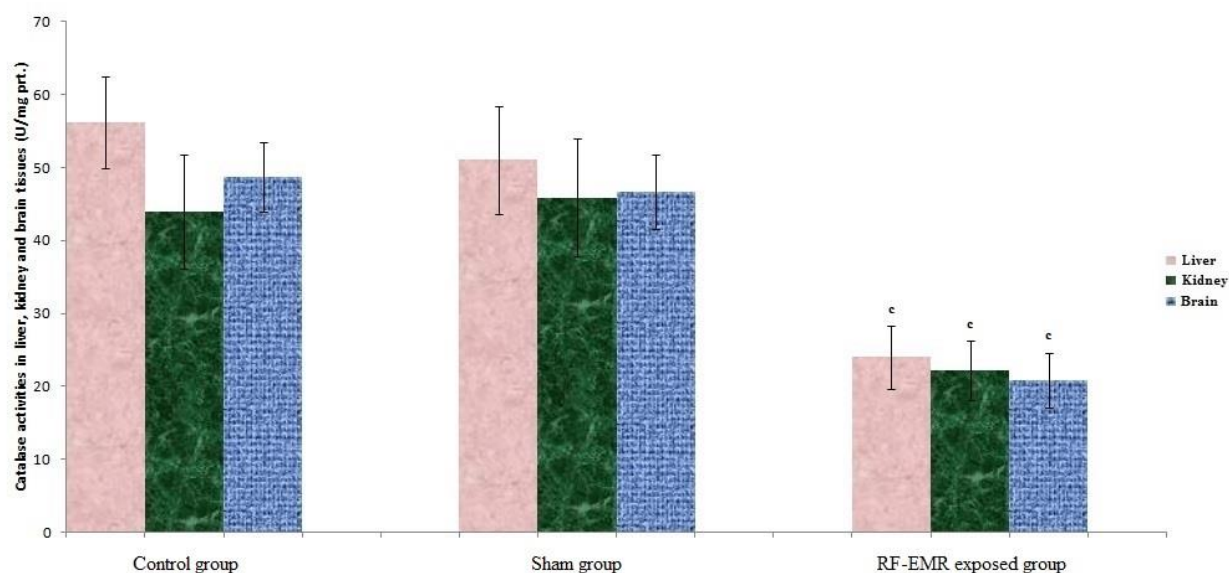


Fig. 3. Catalase activities in liver, kidney and brain tissues
^c Significantly lower than the control and sham groups ($p < 0.001$)

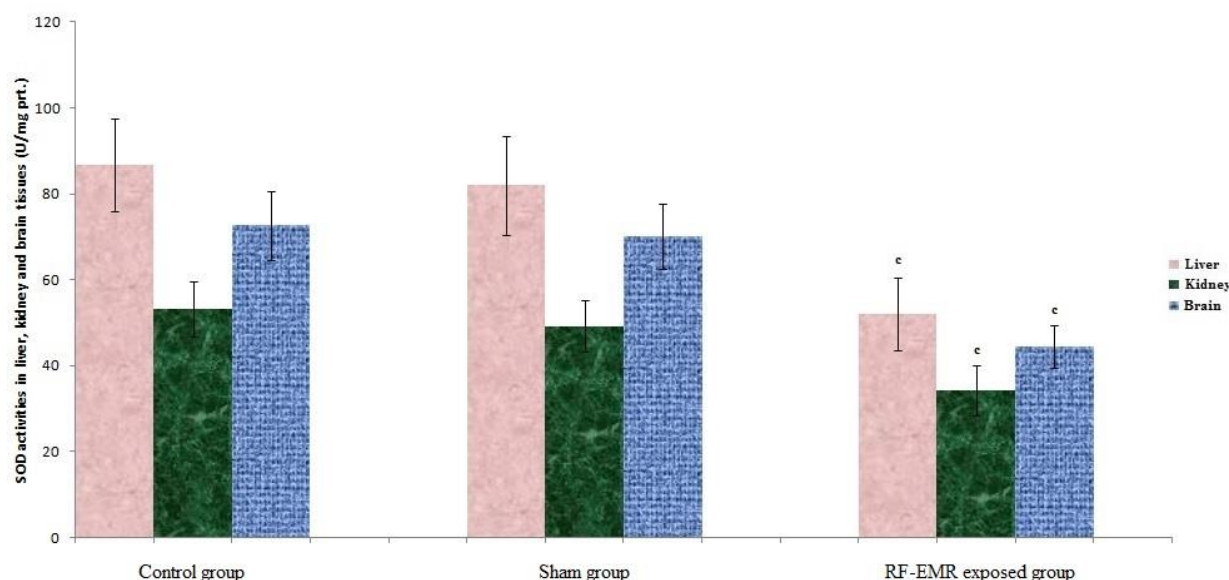


Fig. 4. SOD activities in liver, kidney and brain tissues
^c Significantly lower than the control and sham groups ($p < 0.001$)

oxidative tissue injury (19). Dasdag et al (19) reported increased levels of MDA and of the total oxidant status in liver tissue of the Wistar albino rats exposed to 900 MHz RF-EMR. However, they found no significant end points interms of catalase, myeloperoxidase, total antioxidant capacity levels. Koyu et al (20) investigated the effects of microwave radiation on the liver oxidant/antioxidant system, and the possible protective effects of caffeic acid phenethylester (CAPE) on liver tissue. They observed increased level of lipid peroxidation and decreased activity of GSH-Px in the liver tissue of the 1800 MHz RF-EMR exposed rats. Moreover the authors

noted that CAPE prevented the oxidative injury in liver tissue due to RF-EMR radiation exposure (20). The significant increase in lipid peroxidation level in exposed rat liver tissues may be due to an increase in NO level in the recent study. Overproduction of NO is one of the most important sources of oxidative damage in liver. This action of NO with O_2^- can produce the $ONOO^-$ which can cause lipid peroxidation (20).

In our study, chronic exposure to 1800 MHz RF-EMR increased MDA and NO levels and decreased SOD and catalase activities and GSH level in kidney tissues compared to sham and

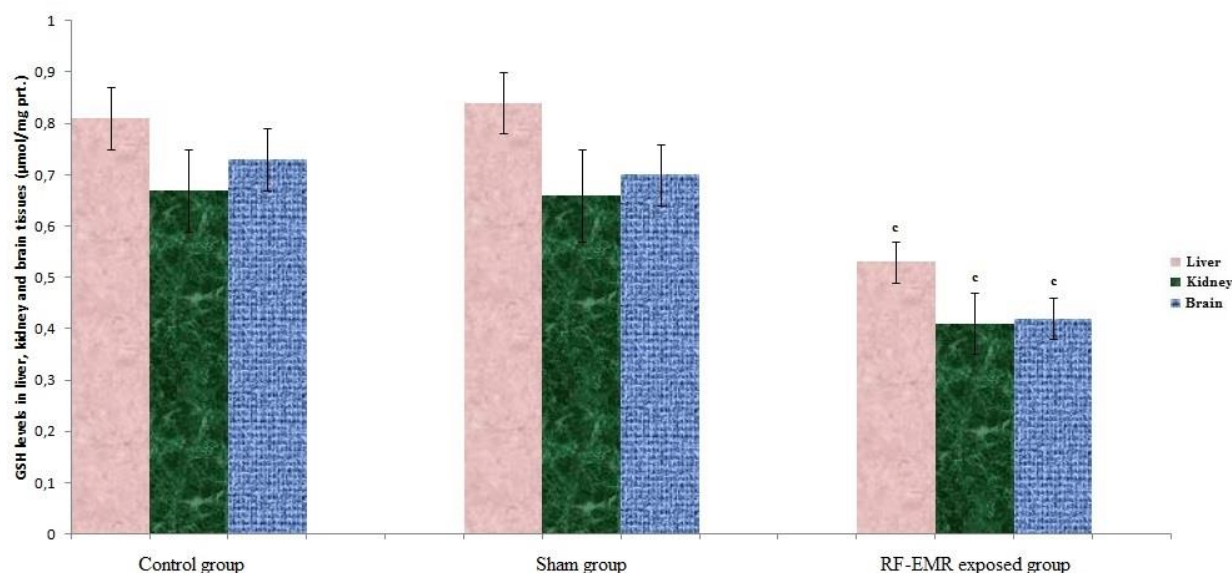


Fig. 5. GSH levels in liver, kidney and brain tissues

^c Significantly lower than the control and sham groups ($p < 0.001$)

control groups. These indicate that RF-EMR may act as a stressor on kidney tissue as well due to its sensitive ultrastructure to oxidative stress. Ozguner et al (21) investigated the effect on kidney tissue of exposure to 900 MHz RF-EMR in 8-week-old rats and reported an increase in MDA levels. MDA levels increased significantly in kidney and bladder tissue of rats exposed to the effect of a 900 MHz RF-EMR in adolescence compared to the control group (21). Ragy (22) determined an increased MDA level and a decrease in total antioxidant capacity in kidney tissue of adult rats. On the other hand, exposure to 900 MHz RF-EMR significantly decreased catalase and SOD activities and GSH level in rat kidney tissue compared to control groups (22). We thought that the kidneys have relatively weak enzymatic antioxidant defense systems capable of producing high levels of ROS through blood perfusion and high anaerobic metabolism so the alteration in lipid peroxidation is dramatic. Ozturk et al (23) reported a reduction in SOD and catalase activity but interestingly an increase in GSH activity in rat kidney tissue exposed to 900 MHz RF-EMR. Increased SOD activity may therefore be a response intended to balance or suppress high chain oxidation of GSH or decreased GSH level (23). Özorak et al (23) investigated that the effects of both Wi-Fi and 900 and 1800 MHz RF-EMR on oxidative stress in the new-born rats' kidney tissues. It has been observed that Wi-Fi and mobile phone-induced RF-EMR may cause oxidative kidney injury in new-born rats (23). Ozgur et al (25) reported that exposure to RF-EMR induces lipid peroxidation,

accompanied by decreased activity of SOD, myeloperoxidase and glutathione peroxidase (GSH-Px) in the kidney of guinea pig. At this point; the major effect of antioxidants on human health occurs through their radical scavenging mechanism. Increasing numbers of studies are focusing on the harmful effects of RF-EMR and on the use of antioxidants in order to minimize these (25).

The most dramatic increase in lipid peroxidation and decrease in antioxidant levels of the RF-EMR exposed rats were observed in brain tissue. We found that chronic exposure to 1800 MHz RF-EMR quite significantly increased MDA and NO levels and decreased SOD and catalase activities and GSH level in brain tissues compared to sham and control groups. The obtained results indicated that 1800 MHz RF-EMR radiation exposure is quite detrimental on brain tissue and electromagnetic fields induce an unbalance between production and the neutralization of prooxidant and antioxidant processes and cause oxidative damage in brain. Studies in the literature generally focus on the brain, since cell phones are held close to the head during use. There is considerable evidence that RF-EMR can affect neural functions in the human brain. Low frequency (0-300 Hz) and RF (10 MHz-300 GHz) EMR has also been reported to alter the permeability of the blood-brain barrier. At the same time, these changes in the blood-brain barrier may lead to excess accumulation of heavy metals and specifically of iron in the brain. This effect may trigger several neuronal disorders. Neurons are largely depending on oxidative

phosphorylation for energy and this makes them more vulnerable to oxidative stress compared to other cells. The metabolic activity of the brain and the demand for oxygen consumption are extremely high (26). Meral et al (27) indicated that 890-915 MHz RF-EMR emitted by cellular phones may generate oxidative stress in brain tissue. They showed that MDA levels increased and GSH level and catalase enzyme activity decreased, while vitamin A, E and D₃ levels remained unchanged in the brain tissue of guinea pigs (27). Ghanbari et al reported that 50-day exposure to EMR causes oxidative stress by increasing MDA levels and reducing SOD activity, and observed that treatment with vitamin E prevented oxidative stress and lipid peroxidation in the substantia nigra part of the brain tissue. Several studies have shown neuronal damage and cellular losses caused by exposure to RF-EMR in many regions of the brain, including the cortex, basal ganglia, hippocampus and cerebellum (29). Rubin et al (29) noted that the pain level of headache may increase during exposure but decreased immediately when exposure ceased.

In the recent study, we showed that chronic exposure to 1800 MHz RF-EMR is capable of inducing oxidative stress. This induction was mediated by increase of lipid peroxidation and the reduction of enzymatic and non-enzymatic antioxidants. The study also gave evidence that RF-EMR might enhance NO production. The results of our study are evident that RF-EMR may act as an environmental stressor and cause oxidative and nitrosative damage in liver, kidney and brain tissues. Oxidative stress is known to underlie many human diseases including chronic liver diseases, renal failure, hypertension, neurodegenerative diseases and also several types of cancer. Our results suggested that using the electromagnetic sources should be limited for protecting the human health.

References

1. Swerdlow AJ, Feychting M, Green AC, et al. Mobile phones, brain tumors, and the interphone study: where are we now? *Environ Health Perspect* 2011; 119: 1534-1538.
2. Pourlis AF. Reproductive and developmental effects of EMF in vertebrate animal models. *Pathophysiology* 2009; 16: 179-189.
3. Feychting M, Ahlbom A. Magnetic fields and cancer in children residing near Swedish high-voltage power lines. *Am J Epidemiol* 1993; 138: 467-481.
4. Valberg PA, van Deventer TE, Repacholi MH. Workgroup report: base stations and wireless networks-radiofrequency (RF) exposures and health consequences. *Environ Health Perspect* 2007; 115: 416-424.
5. Megha K, Deshmukh PS, Banerjee BD, Tripathi AK, Abegaonkar MP. Microwave radiation induced oxidative stress, cognitive impairment and inflammation in brain of Fischer rats. *Indian J Exp Biol* 2012; 50: 889-896.
6. Challis LJ. Mechanisms for interaction between RF fields and biological tissue. *Bioelectromagnetics* 2005; 7: 98-106.
7. Foster KR, Repacholi MH. Biological effects of radiofrequency fields: does modulation matter? *Radiat Res* 2004; 162: 219-225.
8. Kesari KK, Meena R, Nirala J, Kumar J, Verma HN. Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the hsp27/p38MAPK stress pathway in rat brain. *Cell Biochem Biophys* 2014; 68: 347-358.
9. Sepehrimanesh M, Kazemipour N, Saeb M, Nazifi S. Analysis of rat testicular proteome following 30-day exposure to 900 MHz electromagnetic field radiation. *Electrophoresis* 2014; 35: 3331-3338.
10. Kovacic P, Somanathan R. Electromagnetic fields: mechanism, cell signaling, other bioprocesses, toxicity, radicals, antioxidants and beneficial effects. *J Recept Signal Transduct Res* 2010; 30: 214-226.
11. Luo X, Chen M, Duan Y, et al. Chemoprotective action of lotus seedpod procyanidins on oxidative stress in mice induced by extremely low-frequency electromagnetic field exposure. *Biomed Pharmacother* 2016; 82: 640-648.
12. Akar A, Karayıgıt MÖ, Bolat D, et al. Effects of low level electromagnetic field exposure at 2.45 GHz on rat cornea. *Int J Radiat Biol* 2013; 89: 243-249.
13. Yagi K. Simple procedure for specific enzyme of lipid hydroperoxides in serum or plasma. *Methods Mol Biol* 1998; 108: 107-110.
14. Sun Y, Oberley LW, Ying L. A simple method for clinical assay of superoxide dismutase. *Clin Chem* 1988; 34: 497-500.
15. Aebi H. Catalase in vitro. *Methods Enzymol* 1984; 105: 121-126.
16. Beutler E, Duron O, Kelly BM. Improved method for the determination of blood glutathione. *J Lab Clin Med* 1963; 61: 882-888.
17. Miranda KM, Espey MG, Wink DA. A rapid, simple spectrophotometric method for simultaneous detection of nitrate and nitrite. *Nitric Oxide* 2001; 5: 62-71.
18. Lowry OH, Rosebrough NJ, Farr AL, Randall RJ. Protein measurement with the Folin phenol reagent. *J Biol Chem* 1961; 193: 265-275.
19. Dasdag S, Bilgin HM, Akdag MZ, Celik H, Aksen F. Effect of long term mobile phone exposure on

- oxidative-antioxidative processes and nitric oxide in rats. *Biotechnol & Biotechnol* 2008; 22: 992-997
20. Koyu A, Naziroglu M, Özgüner F. Caffeic acid phenethyl ester modulates 1800 MHz microwave-induced oxidative stress in rat liver. *Electromagn Biol Med* 2005; 24: 135-142.
 21. Ozguner F, Oktem F, Ayata A, Koyu A, Yilmaz HR. 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. *Mol Cell Biochem* 2005; 277: 73-80.
 22. Ragy MM. Effect of exposure and withdrawal of 900-MHz-electromagnetic waves on brain, kidney and liver oxidative stress and some biochemical parameters in male rats. *Electromagn Biol Med* 2015; 34: 279-284.
 23. Ozturk A, Baltaci AK, Mogulkoc R, Oztekin E. Zinc prevention of electromagnetically induced damage to rat testicle and kidney tissues. *Biol Trace Elem Res* 2003; 96: 247-254.
 24. Özorak A, Naziroğlu M, Çelik Ö, et al. Wi-Fi (2.45 GHz)- and mobile phone (900 and 1800 MHz)-induced risks on oxidative stress and elements in kidney and testis of rats during pregnancy and the development of offspring. *Biol Trace Elem Res* 2013; 156: 221-229.
 25. Ozgur E, Güler G, Seyhan N. Mobile phone radiation-induced free radical damage in the liver is inhibited by the antioxidants N-acetyl cysteine and epigallocatechin-gallate. *Int J Radiat Biol* 2010; 86: 935-945.
 26. Nittby H, Grafström G, Eberhardt JL, et al. Radiofrequency and extremely low-frequency electromagnetic field effects on the blood-brain barrier. *Electromagn Biol Med* 2008; 27: 103-126.
 27. Meral I, Mert H, Mert N, et al. Effects of 900-MHz electromagnetic field emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs. *Brain Res* 2007; 1169: 120-124.
 28. Ghanbari M, Mortazavi SB, Khavanin A, Khazaei M. The effects of cell phone waves (900 MHz-GSM band) on sperm parameters and total antioxidant capacity in rats. *Int J Fertil Steril* 2013; 7: 21-28.
 29. Rubin GJ, Hahn G, Everitt BS, Cleare AJ, Wessely S. Are some people sensitive to mobile phone signals? Within participants double blind randomised provocation study. *BMJ* 2006; 332: 886-891.