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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.

Keywords Antioxidants; Base transceiver station; Electromagnetic field; GTEM; Oxidative stress.

Introduction

The fast-growing, second generation mobile phone system, global system for mobile communications (GSM), has become the world's largest mobile telecommunications

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system, with its one billion subscribers in over 200 countries (NRPB, 2004). Electromagnetic (EM) radiation produced by mobile phone and their base station antennas are in the 890–960 MHz frequency range for the GSM900 system. While mobile phones emit low power (2 watts or less) EM fields in the 890–915 MHz range, the base station emissions are much higher in power and are in the 935–960 MHz range. Since mobile phones are kept and used near the body, in particular near the head, emissions should be analyzed in the near field. On the other hand, base station radiation must be modeled with far-field considerations. Another concern with base stations is the continuous emission of EM radiation. Therefore, residents who live close to base stations may have a bigger health concern than casual users of the mobile phones.

There are a number of governmental and independent organizations working on the regulation of the EM radiation from mobile phones and their base stations. As a result useful guidelines and directives have been placed in action (CENELEC, 2001; ICNIRP, 1998). These guidelines, however, focus on the SAR values, which address the energy deposition in the body in terms of heat. In *IEGMP Mobile Phones and Health Report* (IEGMP, 2000), concerns were reaffirmed in the following statement as: “There is now scientific evidence, however, which suggests that there may be biological effects occurring at exposures below these guidelines.”

A relatively recent comprehensive survey on mobile phone health effects, published by NRPB (UK) (Sienkiewicz and Kowalczyk, 2005), summarizes 26 reports from several countries. The survey concludes that although the possibility of low-level EM field exposure causing adverse health effects remain “unproven,” additional well targeted, high-quality research will be valuable to explore the remaining uncertainties further.

One of the areas of interest by scientists in recent years is the EM radiation-induced oxidative stress in biological systems. The issue was first studied for the extremely low-frequency (ELF) range, particularly for the power-line frequency of magnetic fields (Brocklehurst and McLauchlan, 1996; Lacy-Hubert et al., 1998). Some recent studies address also the effects in radio frequency (RF) range, especially for mobile telecommunications emissions (Dasdag et al., 2004; Ilhan et al., 2004; Ozguner et al., 2005; Zmyslony et al., 2004).

A free radical is a molecule or ion that has an unpaired electron in its outer orbit. Radical particles are unstable and are very reactive. Most common free radicals are produced from oxygen metabolism and are, thus, called reactive oxygen species (ROS). Free radicals start chain reactions as the unpaired electron is transferred from one molecule to another. One known consequence of this kind of a chain reaction is the lipid peroxidation, where reactive radicals, such as NO_2^- , OH^- , or CCl_3O_2^- , abstract an atom of hydrogen from poly-unsaturated fatty acid (PUFA) side chains in membranes or lipoproteins. This leaves an unpaired electron on carbon. The carbon radical reacts with oxygen and the resulting peroxy radical attacks adjacent fatty acid side chains to generate new carbon radicals and so the chain reaction continues. The attack of one reactive free radical can oxidize multiple fatty acid side chains to lipid peroxides, damaging membrane proteins. This makes the membrane leaky and eventually causes complete membrane breakdown (Anderson, 1995). MDA is one of the end products of lipid peroxidation, which is mostly used as an oxidative stress marker.

There is experimental evidence that oxidative stress and consequent free radical production are important causative factors in the pathology of several degenerative

disorders, such as neurodegenerative diseases, carcinogenesis, atherosclerosis, hypertension, and diabetes mellitus (Mahboob et al., 2001; Maritim et al., 2003; Simonian and Coyle, 1996; Turner, 1996). Oxidative stress refers to the cytotoxic consequences of oxygen radicals, such as superoxide anion ($O_2^{\cdot-}$), hydroxyl radical ($OH\cdot$), and hydrogen peroxide (H_2O_2), which are generated as byproducts of cellular aerobic metabolism. Oxygen radicals can attack DNA, proteins, and lipid membranes, thus disturbing their physical functions.

Fortunately, there are enzymatic systems to counterbalance the production of ROS. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) are enzymes responsible for degradation of $O_2^{\cdot-}$. While SOD converts $O_2^{\cdot-}$ to H_2O_2 , GSH-Px converts H_2O_2 to water and oxygen. GSH-Px uses H_2O_2 to oxidize reduced glutathione (GSH). Decreased activity of GSH-Px leads to accumulation of H_2O_2 , which in the presence of iron and copper promotes the Fenton reaction to yield $OH\cdot$. Hydroxyl radical is the most reactive form of oxygen radicals that can initiate a chain reaction to generate numerous toxic reactants. GSH-Px also participates in detoxification of lipid peroxyl radicals (Coyle and Puttfarcken, 1993; Stadtman, 1998).

In this article, we submit an animal study in a confined, well-controlled environment, with an intention to investigate the effects of GSM base transceiver station (BTS) EM radiation on oxidative stress and generation of free radical. During this study a group of young adult male Wistar albino rats was exposed to EM fields inside a test chamber, called gigahertz transverse electromagnetic (GTEM) cell, while another group was sham-exposed under the same conditions. Malondialdehyde (MDA) levels were assessed as a marker of lipid peroxidation and the activities of GSH and SOD were determined in order to evaluate antioxidant status in the blood samples of exposed rats.

Methods

Exposure Environment

The test setup is a general purpose EM shielded chamber that is suitable to house laboratory animals. In this exposure environment electromagnetic radiation of specific energy, frequency, and waveform can be applied. The main element of the setup (Figure 1) is a GTEM cell (GTEM 1750, MEB, Germany) installed in the Electromagnetic Compatibility (EMC) Laboratory of Tubitak-Uekae.

GTEM cell is a radiated field generation and measurement device that has a single input/output port for EM immunity and emission test purposes. GTEM cell, in essence, is a transmission line terminated with $50\ \Omega$ load. The asymmetrical inner conductor (called "septum") is designed to match the impedance of a tapered, rectangular wave guide cross section. The outer surface of the GTEM cell is made of metal sheets with an attenuation of approximately 60 dB at high frequency EM fields. Note that honeycomb waveguides are installed at the bottom of the GTEM cell for ventilation purposes. Electric field level is directly proportional to the amplitude of the voltage applied to the input port and inversely proportional to the septum height. In order to obtain increased field level with constant input power, cages that contain the test subjects are designed and placed closer to the input port of the GTEM cell for the purpose of biological effects study, as illustrated in Figure 2.

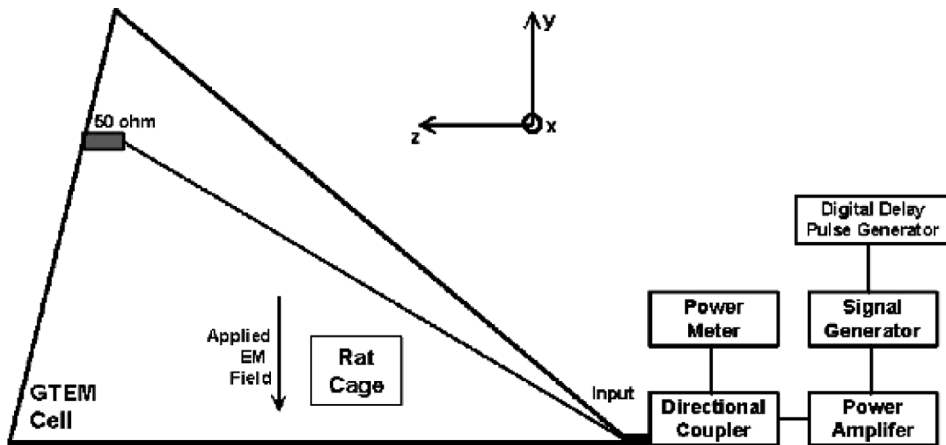


Figure 1. Electromagnetic field exposure setup.

Signal generators are used to feed the input port of the GTEM cell in order to imitate the electromagnetic fields emitted from base transceiver stations. A properly selected signal generator (SMY01, Rohde & Schwarz, Germany) generates the 945 MHz carrier wave, which is then pulse modulated at 217 Hz (using Stanford Research Systems DG535 Digital Delay/Pulse Generator, USA) in accordance with GSM specifications. Output of the signal generator is then connected to a 100 W power amplifier (757LCB-CE, Kalmus, USA). Power feed to the GTEM cell input is provided through a directional coupler. One port of the directional coupler is connected to a power meter (Rohde & Schwarz NRVS with thermal probe NRV-Z51) through which applied power level is monitored (Figure 1).

The rats are enclosed inside two *metabolic cages*, which are placed adjacent to each other. These cages are designed to feed and water the rats and collect urine samples through its structure. The cages are composed of transparent Plexiglas material. It is thus assumed that the cages have minimal effect on the applied electric field. The rats are free to move inside a volume of 24 cm (W) $\times$ 34 cm (L) $\times$ 12.5 cm (H). The applied electric field is perpendicular to the plane on which the rats move; therefore, each animal is exposed to the field without any obstruction caused by its “neighbors.”

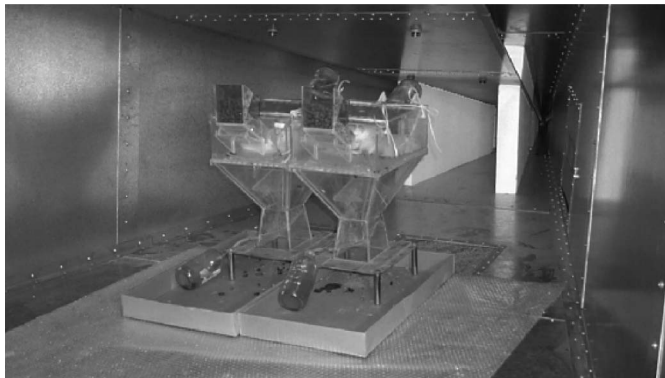


Figure 2. A group of rats contained in two metabolic cages inside GTEM cell.

SAR Calculation

It is necessary to make the dosimetric evaluation of an electromagnetic field exposure experiment in order to deduce meaningful results and compare various biological effects produced with different test setups. Dosimetric evaluation is performed, in general, in terms of specific absorption rate, or “SAR”. Since exposure from base transceiver stations (BTS) is generally in the far field, whole-body average SAR is taken into consideration in this experiment. Whole-body average SAR is defined as the total energy, transferred to the body per unit time, divided by the total mass (Chou et al., 1996).

The finite-difference time-domain (FDTD) method implemented in a MATLAB program was used to compute the expected SAR values on the subjects in a simulation environment.

In order to validate the numerical model and MATLAB implementation of the FDTD technique, electric field levels were measured at 10 selected positions on an XY plane located at the middle of the Z axis and compared with those that were obtained by the FDTD technique. These points were selected such that they represent the position of the rats in metabolic cages during actual EM exposure. The simulation software was executed for three times for different input power levels. Electric field levels were also measured by an electric field meter (Acterna EMR-300 with Type 8 E-field probe, Germany) again, at those specified input power levels. We found that the FDTD model simulates closely the actual exposure environment (less than 7% difference in the average electric field levels). Thus, the model is a good approximation of real-life measurements. Electric field measurement results also indicated that a fairly uniform electric field was applied to the test subjects in the exposure environment.

The test subject was, on the other hand, modeled roughly with an effort to maintain some details of its geometry. The rat, as a whole, was modeled using electrical properties at 945 MHz, calculated for average muscle tissue, corresponding to $\varepsilon = 55.853462$ and $\sigma = 0.985515$. Rat muscle tissue density was taken as $\rho = 1040 \text{ mg/cm}^3$. For the sake of simplicity, rat cages were not included in the model since they are transparent to electromagnetic radiation. Normalized SAR value for this exposure environment was found to be 0.90 mW/kg per input W. Thus, when this exposure setup is used for assessment of biological effects, we can compute the SAR values through the FDTD model easily once the GTEM input power level is given.

Electromagnetic Exposure

The calculations show that, in this test environment, the SAR values we can induce are well below the safety standards (CENELEC, 2001). A set of exposure experiments were performed in two phases to investigate the biological effects of the electromagnetic fields at GSM BTS frequency. The first phase was the EM field exposure experiment and the second was the sham exposure (see Table 1). In each phase, the following procedure was applied.

The animals were obtained from the Experiment Animals Center of Istanbul University's Cerrahpasa Medical School. For each of the two phases, a set of nine healthy, young adult male Wistar albino rats were used. The rats were cared for in accordance with the *Guide for the Care and Use of Laboratory Animals* (National Research Council, 1996). The rats were fed with standard pellet food and watered

Table 1
Exposure parameters

Phase	# Rat	GTEM input power (W)	Applied E field level (V/m)	Power density (W/m ²)	SAR (mW/kg)	Exposure duration (days)	Effective exposure time (hours/day)	Time spent in GTEM cell (hours/day)
Experiment	9	12.5	37.2	3.67	11.3	8	7	7
Sham exposure	9	–	–	–	–	8	–	7

by tap water *ad libitum*. The rats were weighed before, after, and at each day of the experiment. Average weights of both groups before and after the experiment and their average food and water consumption are given in Table 2.

The subjects were placed inside two metabolic cages, five rats in one cage and four rats in the other. The cages were placed in the test volume inside the GTEM cell, as shown in Figure 2. Radiofrequency EM field (3.67 W/m² power density) was applied onto the exposure group for the first phase, while there was no EM field exposure onto the sham group, in the second phase. We could only apply a low-level power, which corresponds to a SAR value of 11.3 mW/kg, because of the time-averaging of the applied peak power level in accordance with the GSM modulation of the carrier wave. The cages containing the rats were kept inside the GTEM cell for seven hours for a period of eight days for each group.

Metabolite Gathering and Analysis

At the end of each phase, the animals were anesthetized. After exploration of the thorax, intracardiac blood was quickly obtained. The tubes containing the processed blood samples were stored at –24°C until they were analyzed at the Cerrahpasa Medical School Biochemistry Laboratory. These samples were processed for measuring malondialdehyde (MDA), reduced glutathione (GSH) and superoxide dismutase (SOD) levels. The materials collected were analyzed using a spectrophotometer (JASCO V-530 UV/VIS), according to standard laboratory procedures (Burtis et al., 1999; Yi et al., 1988).

Table 2
Average weights of the rates before and after the experiment and their average food and water consumption

Groups	Initial weights (g)	Weights after exposure (g)	Average consumed food per day by single rat (g)	Average consumed water per day by single rat (ml)
Experiment	190 ± 40	220 ± 38	22	21
Sham Exposure	162 ± 43	191 ± 44	30	18

MDA level was determined in plasma using the principle of spectrophotometric determination of absorbance at 535nm of colored complex, which is formed by condensation of two molecules of thiobarbituric acid and one molecule of the material that it reacts with. For this purpose, 1ml of a stock solution, which was prepared from trichloroacetic acid, thiobarbituric acid, and hydrochloric acid, was added to 0.5ml of plasma. After pippeting, tubes were closed and put inside a boiling water bath for 20 minutes. After cooled, they were centrifuged at 2000cycles/min. for 10 minutes. Absorbance of the supernatant solution was read against the blind solution. MDA level is given in millimoles per liter (mM/L).

GSH concentration was determined in total blood by color reading at 412 nm of the solution that is formed by the reaction of free sulfhydryl compounds in the whole blood with 5,5'-Dithiobis (2-nitrobenzoic acid) (DTNB). Note that DTNB is a disulfide chromogen that is readily reduced by sulfhydryl compounds to an intensely yellow compound, and the absorbance of the reduced compound is directly proportional to the GSH concentration. Cuverts were prepared for GSH calibrator, erythrocyte, and precipitating agent. After incubating at room temperature for five minutes, the solutions were centrifuged at 3000cycles/min. for 10 minutes. Upper phase was extracted and phophate buffer, DTNB, and distilled water were added. The color reading was performed at 412nm against the blind solution in four minutes. The calculation was done by using glutathione standard curve. GSH concentration was expressed in milligrams per gram hemoglobin (mg/g Hb).

The method of determination of SOD activity was based on the reduction of nitroblue tetrazolium by superoxide anions that were formed by xanthine/xanthine-

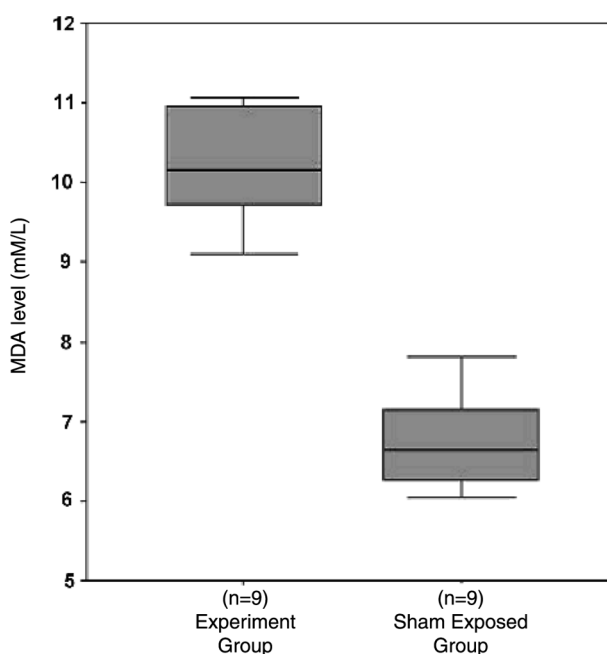


Figure 3. Effects of 945MHz electromagnetic radiation on lipid peroxidation levels in rat blood samples (mean $\pm$ SD). $p < 0.0001$ vs. sham according to Mann-Whitney U test.

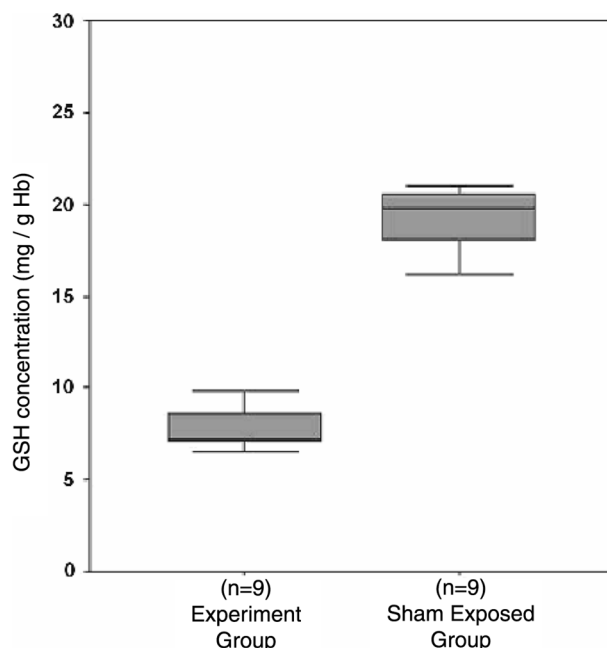


Figure 4. Effects of 945 MHz electromagnetic radiation on GSH concentration in rat blood samples (mean $\pm$ SD). $p < 0.0001$ vs. sham according to Mann-Whitney U test.

oxidase system and then inhibition of superoxide anions by SOD. The reaction mixture used to determine the SOD activity in erythrocytes was prepared using xanthine, EDTA, nitroblue tetrazolium, Na_2CO_3 , and bovin serum albumin at specified concentrations inside a 200ml flask. The reaction mixture was set to a pH level of 10.2. Supernatant solution was prepared using chloroform letanal, erythrocyte, and distilled water. This solution was centrifuged at 15000 cycles/min. for 10 minutes. Test and blind solutions of 1.07ml were incubated at 25°C for 20 minutes. After adding 0.05 ml of CuCl_2 , (8 mM/l) reaction was stopped. Note that the 50% inhibition with respect to the blind inside the reaction tube corresponds to one unit of SOD activity. The units of SOD activity were determined by simple linear ratio. But it had to be further determined with respect to the hemoglobin concentration inside the solution, so the resultant SOD activity was formulated in terms of units per gram hemoglobin (or U/g Hb).

Hemoglobin concentration for GSH and SOD analysis was determined using Drabkin's reagent. Three mL reagent was mixed with 1:20 hemolysate and kept in the dark for 10 minutes. Absorbance at 540nm was read against a blank of Drabkin's solution and hemolysate hemoglobin concentration was determined from a calibration curve.

Data was presented as means standard deviation. The SPSS software package (SPSS for Windows release 11.50) was used for statistical analysis. The analysis results were evaluated based on Mann-Whitney U test statistics and a $p < 0.05$ was considered significant.

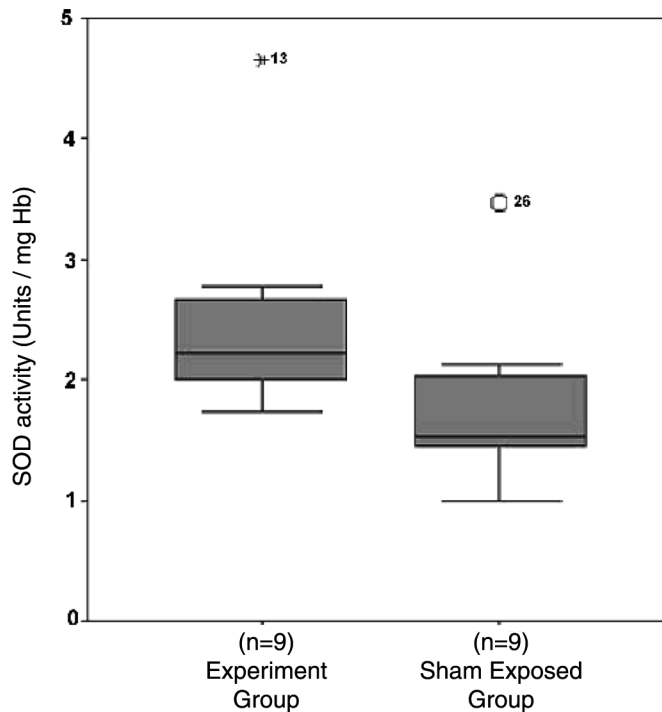


Figure 5. Effects of 945 MHz electromagnetic radiation on SOD activity in rat blood samples (mean $\pm$ SD). $p = 0.019 < 0.05$ vs. sham according to Mann-Whitney U test.

Results

Lipid peroxidation (MDA – malondialdehyde), GSH (reduced glutathione), and SOD (superoxide dismutase) analysis results are given in Figures 3–5, respectively. Levels of all the three parameters of interest have been changed significantly under EM radiation effect. The MDA level was increased significantly ($p < 0.0001$) for the experiment group (10.20 ± 0.78 mM/L) when compared to the sham-exposed group (6.77 ± 0.60 mM/L). SOD activity was also increased for the experiment group (2.53 ± 0.87 units/mg Hb), although less significantly ($p = 0.019$), when compared to the sham-exposed group (1.79 ± 0.72 units/mg Hb). GSH concentration, on the other hand, decreased significantly ($p < 0.0001$) for the experiment group (7.84 ± 1.15 mg/g Hb), when compared to the sham exposed group (19.19 ± 1.64 mg/g Hb).

Discussion and Conclusion

A two-phase experiment was designed in order to investigate the effects of EM fields at GSM BTS transmitter frequencies. In the first phase, EM field at 945 MHz of 3.67 W/m² power density, which is calculated to produce a SAR value of 11.3 mW/kg, was applied on a group of nine rats inside a GTEM cell. In the second phase, the same number of rats was sham exposed under the same conditions. The duration was seven hours/day for a period of eight days in each phase.

A biochemical analysis of blood samples indicates that the GSM BTS EM radiation-exposed rats have higher concentration levels of MDA (malondialdehyde)

and lower GSH (glutathione) concentrations, when compared to the sham exposed group. This outcome is in agreement with the results of some previous studies. Dasdag et al. (2004) studied the effects of mobile phone EM radiation on rat brain tissues, where rats are exposed to EM fields for 20 min./day for 30 days (SAR = 0.52 W/kg). The MDA levels in rat brain tissues increased significantly after exposure. In another study by Ozguner et al. (2005), renal impairment in rats was investigated under mobile phone radiation (SAR = 0.016 W/kg). Rats were exposed 30 min./days for 90 days and the result was an increase of renal tissue MDA, NO (nitric oxide), and urinary NAG (N-acetyl- β -D-glucosaminidase) levels, and a significant decrease of tissue SOD, CAT (catalase), and GSH-Px (glutathione peroxidase) levels. Ilhan et al. (2004) studied mobile phone-induced oxidative stress in rat brain. Rats were exposed to mobile phone emissions (SAR = 0.25 W/kg) for one hour/day for seven days. MDA and NO levels were increased, whereas GSH-Px and SOD activities were decreased significantly in rat brain tissue. Additionally, XO and ADA levels were also increased. Moreover, Moustapha et al. (2001) have studied acute mobile phone exposure effects on human volunteers and found out that MDA level in plasma was increased and GSH-Px and SOD activities were decreased in human erythrocytes after one hour, two hours, and four hours of exposure. No significant change was observed for CAT activity in this study.

However, some other studies in literature have concluded there is no relation between EM field exposure and free radical activity. Irmak et al. (2002) have shown that EM radiation from mobile phones applied 30 min./days for seven days on rabbits had no significant effect on rabbit brain, suggesting that oxygen free radicals were not generated. But, they have observed a significant increase in serum SOD activity in the exposed group. In addition, Zmyslony et al. (2004) have exposed rat lymphocytes *in vitro* to 930 MHz continuous wave (CW) radiation inside a GTEM cell (SAR = 1.5 W/kg). Five minutes and 15 minutes of acute exposure resulted in no change in reactive oxygen species production in living cells. However, change was observed when lymphocytes were treated with FeCl₂ as a stimulating agent.

Some research studies relate oxidative stress to certain diseases. In a Parkinson's disease model, SOD, CAT, and GSH-Px activities were increased in the striatum and ventral midbrain of mice (Cassarino et al., 1997). In another Parkinson's disease model, Thiffault et al. (1995) showed an increase in SOD activity with no increase in GSH-Px activity in mice brain. Neuronal GSH deficiency and age-dependent neurodegeneration were found to be related with the down-regulation of excitatory amino acid transporters in mice (Aoyama et al., 2005).

According to our results, the MDA level and SOD activity were increased, whereas GSH concentration was decreased in EM radiation-exposed rats, when compared to the sham-exposed group. We think that decreased GSH concentration may be due to the higher consumption of GSH for scavenging the higher production of free radicals. The lower GSH concentration may have some relation to the increased SOD activity. Note that the endogenous GSH has a protective function in scavenging radicals and in molecular repair. However, such scavenging can set up a superoxide-dependent chain production of H₂O₂ and oxidized glutathione, which would oxidatively stress the cell. Therefore, the increased SOD activity may be a response to suppress the higher chain oxidation of GSH, or a response to balance the decreased GSH concentration.

Thus, the upregulated SOD activity converts O₂^{·-} to H₂O₂, and downregulated GSH concentration leads to the insufficient detoxification and accumulation of

H₂O₂ in the system. H₂O₂ is known to be converted to hydroxyl radical in the presence of iron rapidly, which is the most damaging form of ROS. Note that the balance between GSH-Px and SOD appears to be important for the cellular resistance to oxidative stress (Amstad et al., 1994).

In conclusion, our results indicate that exposure to EM fields at BTS frequencies may modulate the oxidative stress of free radicals by enhancing lipid peroxidation and reducing the concentration of GSH. It is highly probable that detoxification of lipid peroxy radicals can not be accomplished in our system. We note that reactive oxygen species may have a probable role on adverse effects of EM fields at mobile telecommunication frequencies. The outcome of this study needs confirmation by repetitive laboratory studies, including higher dose and longer duration exposure, and theoretical mechanisms underlying this effect should also be addressed in the future.

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