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Effects of Electromagnetic Field Produced by Mobile Phones on the Oxidant and Antioxidant Status of Rats

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Abstract: This study was designed to investigate the effect of EMR produced by GSM Mobile Phones (MP) on the oxidant and antioxidant status in rats. Rats were divided into three groups: (1) controls, (2) rats exposed to a fractionated dose of EMR (15 min day^{-1} for four days) (EMR-F) and (3) rats exposed to an acute dose of EMR (EMR-A). A net drop in the plasma concentration of vitamin C (-47 and -59.8%) was observed in EMR-F and EMR-A groups, respectively, when compared to controls. While, a significant decrease in the levels of lypophilic antioxidant vitamins: vitamin E (-33 and -65.8%), vitamin A (-44.4 and -46.8%) was observed in EMR-F and EMR-A groups, respectively, when compared to controls. A net drop in plasma level of reduced glutathione (GSH) (-19.8 and -35.3%) was observed in EMR-F and EMR-A groups, respectively. EMR exposure of rats produced a significant decrease in catalase (CAT) and superoxide dismutase (SOD) activities, with the values of these activities for EMR-A group is significantly lower than those of EMR-F. These results indicate that the effects of acute doses of EMR produced by mobile phones on the rat´s antioxidant status is significantly higher than those of fractionated doses of the same type of radiation. On the basis of present results, it can be concluded that exposure to acute doses of EMR produced by mobile phones is more hazardous than that produced by fractionated doses of the same type of radiation.

Key words: Mobile, electromagnetic, radiation, antioxidant

INTRODUCTION

Mobile phones and their base stations produce electromagnetic radiation (EMR). Electromagnetic radiation is absorbed in the body and produces heat, but the body´s normal thermoregularity processes carry this heat away. All established health effects of EMR exposure are clearly related to heating (thermal) effect. Since EMR from mobile phones can interact with body tissues at levels too low to cause any significant heating, no study has shown adverse thermal effects at exposure levels below international guideline limits. A typical mobile phone operates at a power output of 0.25 W, which results in a specific energy absorption rate of about 1.5 w kg^{-1} and an associated very low rise in brain temperature (maximum, 0.1°C) (Haramaki *et al*., 1994). Thus, the possible biologic effects from cellular phone use would not be expected to be thermal in nature. A thermal effects have been the most difficult to explain because the mechanism by which they affect biologic tissue is usually unknown. In the literature, there has been much inquiry and apprehension about the possible development of brain tumors because of exposure to mobile phones. One of the possible mechanisms for tumor development is increase in the permeability of the blood brain barrier, which may result in the entry of carcinogenic substances into the brain. In particular, a variety of neurological effects have been postulated to occur as a result of exposure to EMR, including headaches (Paglia and Valentine, 1967; Balickci *et al*., 2005; Al-Khlaiwi and Meo, 2004), changes in sleep patterns (Stopczyk *et al*., 2002; Loughran *et al*., 2005; Al-Khlaiwi and Meo, 2004), modifications in the electroencephalogram (EEG) (Bridi *et al*., 2001; Lebedeva *et al*., 2001), decrease in the reflex (Balikci *et al*., 2005), clicking sound in the ears (Balikci *et al*., 2005) and an increase in blood pressure have also been reported (DeFeudis, 1998).

Electromagnetic 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 whole body exposure to electromagnetic fields of mobile phones on the antioxidant capacity in the blood of rats.

MATERIALS AND METHODS

Exposure system and animals: Ninty-nine rats (each weighing 250-300 g at the time of experiment) were used.

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The whole body of each rat was exposed to electromagnetic waves emitted from the antenna of the mobile phone. In the present study, a 900 MHz electromagnetic near-field signal for GSM (Global System for Mobile communication at 900 MHz) system was used. The average Specific Absorption Rate (SAR) of the whole body was 0.25 w kg^{-1}.

The rats were divided into three groups; (1) controls, (2) EMR-F and (3) EMR-A. There were a total of 33 rats in the control group, 42 rats in the EMR-F group and 24 rats in EMR-A group.

Exposure periods: The rats of the EMR-F group were exposed to a fractionated dose of EMR (15 min day^{-1} for four days) and those of the EMR-A group were exposed to an acute dose of EMR for one hour.

Blood collection and preparation of hemolysate: Blood samples by cardiac puncture were drawn into heparinized tubes. The blood samples were centrifuged at 1000 rpm for 10 min to remove plasma. The buffy coat on the erythrocyte sediment was separated carefully after the plasma was removed.

The erythrocyte sediment was washed three times with 5-fold isotonic NaCl solution to remove the plasma remnant. After each procedure, erythrocyte saline mixture was centrifuged at 1000 rpm for 10 min. Aliquots of the samples were transferred into polyethylene tubes to be used in the assay of biochemical parameters. Erythrocyte sediment samples were stored at 30°C until analysis. At the time of analysis, erythrocyte sediments were treated with 4-fold ice-cold deionized water to obtain hemolysate. Determination of vitamin C in plasma, the plasma level of vitamin C was determined according to the method of Harris and Ray (1935)

Determination of Vitamin E in erythrocytes, it was determined following the method of Quaife *et al.* (1949). Determination of Vitamin A in plasma, it was determined according to the method of Stocker and Frei (1991). Determination of reduced glutathione (GSH) in erythrocytes, it was determine in erythrocytes following the method of Beutler *et al.* (1963). Catalase (CAT) activity determination, it was determined according to the method of Aebi (1984). Superoxide dismutase (SOD) activity determination, it was determined according to the method of Nishikimi *et al.* (1972).

Statistical analysis: Data are presented as means±-SD Student′s t-test was used for determination of the level of significance of difference between different groups. The difference is considered significant at $p<0.05$.

RESULTS AND DISCUSSION

Whole body exposure of rats either to a fractionated dose or an acute dose of EMR produced a significant decrease in the plasma concentrations of vitamins C, E and A when compared to controls ($p<0.00005$). The magnitude of this effect in EMR-A group is significantly higher than that of EMR-F for all vitamins, except vitamin A (Table 1).

A significant decrease in these parameters is observed in both EMR-F and EMR-A groups when compared to controls ($p<0.00005$). The values of these parameters for EMR-A group is significantly lower than those for EMR-F group (Table 2).

Electromagnetic fields 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.

Oxidative stress refers to an imbalance between the intracellular production of Reactive oxygen species (ROS) and the cellular defense mechanisms. Proteins, lipids and DNA are sensitive targets of ROS. An excess availability of free radicals accompanied with a reduction of the capacity of the natural antioxidants systems lead to cellular dysfunction and death (Atilla *et al.*, 2004). Hydroxyl radical ($^{\bullet}OH$), $O_2^{\bullet-}$ are the predominant cellular

Table 1: Plasma concentrations of vitamins C, A and erythrocyte concentration of vitamin E in control, EMR-F and EMR-A groups of rats

Groups	Vit. C (mg L^{-1})	Vit. A (μM L^{-1})	Vit. E (μg mL^{-1} RBCs)
C	224.2±0.95	2.95±0.64	2.87±0.32
EMR-F	118.6±0.87	1.64±0.53	1.92±0.26
EMR-A	90.2±0.66	1.57±0.51	0.98±0.18
p-value			
EMR-F-C	0.00005	0.00005	0.00005
EMR-A-C	0.00005	0.00005	0.00005
EMR-F-EMR-A	0.00005	ns	0.00005

ns: not significant, C: Controls, EMR-F: A group of rats exposed to fractionated dose of EMR, EMR-A: A group of rats exposed to an acute dose of EMR

Table 2: Activities of catalase, superoxide dismutase and concentration of reduced glutathione in erythrocytes of control, EMR-F and EMR-A groups of rats

Groups	CAT (μ mL^{-1})	SOD (μ mL^{-1})	GSH (mmol L^{-1})
C	89.0±0.23	54.37±0.18	24.52±0.14
EMR-F	52.0±0.19	43.82±0.17	19.67±0.12
EMR-A	43.0±0.16	42.46±0.14	15.86±0.11
p-value			
EMR-F-C	0.00005	0.00005	0.00005
EMR-A-C	0.00005	0.00005	0.00005
EMR-F-EMR-A	0.00005	0.00005	0.00005

C: Control, EMR-F: A group of rats exposed to fractionated dose of EMR, EMR-A: A group of rats exposed to an acute dose of EMR

free radicals, while hydrogen peroxide (H_2O_2) and $ONOO^-$, although not themselves free radicals, aid substantially to the cellular redox state (Lowry *et al*., 1951). The cytotoxicity of free radicals is related to the ability of theses molecules to oxidize cell constituents, particularly lipids and nucleic acids. An array of cellular defense systems exists to counterbalance free radicals. These include enzymatic and nonenzymatic antioxidants that lower the steady-state concentrations of free radical species, oppose sources that generate cellular oxidants and limit the likelihood that oxidative damage will occur. Cellular antioxidant defense mechanisms include low-molecular weight molecules such as reduced glutathione and vitamins C, E and A and antioxidant enzymes such as SOD, glutathione peroxidase (GSH-Px) and CAT (Atilla *et al*., 2004; Lowry *et al*., 1951; Sundram *et al*., 1996; Piacentini *et al*., 2001; Nahed *et al*., 2004; Goh and Barlow, 2002).

There are several reports in the literature, which indicate that free radicals are involved in EMR-induced tissue damage. Stopczyk *et al*. (2002) investigated *in vitro* effect of EMR produced by mobile phones on the activity of SOD and the level of malondialdehyde (MDA) in human blood platelets. They demonstrated that EMR significantly depleted SOD activity after 1, 2 and 7 min exposure and increased after 3 min in comparison with the control test. The also found the increased MDA concentration after 1, 5 and 7 min and decrease after 3 min of exposure as compared with the control test. In another study reported by Moustafa *et al*. (2001), which include healthy adult male volunteers, it was showed that the plasma level of lipid peroxide was significantly increased after 1, 2 and 4 h of exposure to EMR of the mobile 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. In a recent *in vivo* study, EMR exposure produced a significant increase in MDA and Nitric Oxide (NO) levels and a significant decrease in SOD, CAT and GSH-Px activities in rat´s plasma and erythrocytes (Atilla *et al*., 2004).

In the present study, we found decreased plasma concentrations of vitamins C and A and decreased concentration of vitamin E in erythrocytes of EMR-exposured rats. This finding could be attributed to more oxidation, more free radicals induced by EMR and thus antioxidant vitamins C, A and E were consumed as scavengers of free radicals. We found also decreased GSH concentration and decreased SOD and CAT activities in erythrocytes of EMR-exposured rats compared to controls. This decreased antioxidant capacity may reflect a cellular oxidative stress due to EMR exposure.

As a marker of oxidative stress, lipid peroxidation was monitored by measuring of MDA which results from free radical damage to membrane components of the cells. (Esterbauer *et al*., 1991). We observed an increase in the MDA concentration in the plasma of rats which were exposed to EMR.

The magnitude of these effects in EMR-A group is significantly higher than that of EMR-F for all parameters, except vitamin A, indicating that exposure to acute doses of EMR is more effective in induction of oxidative stress and in reducing the antioxidative capacity than fractionated doses of the same type of radiation.

In conclusion, present results provide evidence of a probable role of ROS in the adverse effects of EMR from MP. Our results indicate also that the exposure to acute doses of EMR produced by MP is more hazardous than the exposure to fractionated doses of the same type of radiation.

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