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Effect of 900 MHz Radio Frequency Radiation on Beta Amyloid Protein, Protein Carbonyl, and Malondialdehyde in the Brain

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Recently, many studies have been carried out in relation to 900 MHz radiofrequency radiation (RF) emitted from a mobile phone on the brain. However, there is little data concerning possible mechanisms between long-term exposure of RF radiation and biomolecules in brain. Therefore, we aimed to investigate long-term effects of 900 MHz radiofrequency radiation on beta amyloid protein, protein carbonyl, and malondialdehyde in the rat brain. The study was carried out on 17 Wistar Albino adult male rats. The rat heads in a carousel were exposed to 900 MHz radiofrequency radiation emitted from a generator, simulating mobile phones. For the study group (n: 10), rats were exposed to the radiation 2 h per day (7 days a week) for 10 months. For the sham group (n: 7), rats were placed into the carousel and the same procedure was applied except that the generator was turned off. In this study, rats were euthanized after 10 months of exposure and their brains were removed. Beta amyloid protein, protein carbonyl, and malondialdehyde levels were found to be higher in the brain of rats exposed to 900 MHz radiofrequency radiation. However, only the increase of protein carbonyl in the brain of rats exposed to 900 MHz radiofrequency radiation was found to be statistically significant ($p < 0.001$). In conclusion, 900 MHz radiation emitted from mobile/cellular phones can be an agent to alter some biomolecules such as protein. However, further studies are necessary.

Keywords Mobile phones, Alzheimer's disease, Beta amyloid protein, Protein carbonyl, Malondialdehyde (MDA)

INTRODUCTION

Use of wireless communication, such as cellular phones and other types of handheld phones, has been increasing day by day. Therefore, public opinion and concern about the potential human health hazards of short and long-term effect of exposure to radiofrequency (RF) radiation (Dasdag et al., 2009; Karadede et al., 2009) has also increased.

There have been many studies to determine the health effects of radiation emitted from cellular phones. These include the possibility of initiation and/or promotion of

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carcinogenesis and the induction of genetic damage (WHO, 1993). Some studies indicated that exposure to radiofrequencies promoted cancer in conventional mice and lymphoma in transgenic mice (Em-pim 1) that are prone to lymphoma (Szmigielski et al., 1982; Szudzinski et al., 1982; Repacholi et al., 1997). However, Sommer et al. (2004) showed that mobile phone exposure does not promote lymphoma in AKR/J mice.

It has been firmly established that a significant level of DNA damage arises from endogenous products of cellular metabolism. For example, oxygen radicals generated in vivo during reduction of O_2 are responsible for DNA damage, which is not associated with the exposure to environmental carcinogens. Oxygen radicals can attack DNA bases or the deoxyribosyl backbone to produce damaged bases or strand breaks. They can also oxidize other cellular macromolecules such as lipids or proteins, to generate reactive electrophiles that bind covalently to DNA bases forming adduct. Endogenous DNA lesions are genotoxic and induce mutations. The level of DNA damage arising from endogenous cellular sources exceeds the level of lesions induced by exposure to exogenous chemical carcinogens or physical agents (Pluskota-Karwatka et al., 2006). Therefore, studies on the bio-effects of mobile phone exposure, which is one of physical agents, have been focused on oxidative process in the living systems recently. One of the biomarkers to measure the level of oxidative stress in an organism is malondialdehyde (MDA). MDA is one of the many important parameters that cause toxic stress in living cells. MDA is the breakdown product of the major chain reactions leading to oxidation of polyunsaturated fatty acids and thus serves as a reliable marker of oxidative stress mediated lipid peroxidation in tissues (Dasdag et al., 2008). However, MDA is a naturally occurring product of lipid peroxidation and prostaglandin biosynthesis, two processes implicated in the pathogenesis of a number of cancers. Moreover, malonaldehyde-DNA lesions have been detected in a number of human tissues (Pluskota-Karwatka et al., 2006). Because of the importance of the oxidative stress in living systems, the number of studies that investigate effects of mobile phone exposure on oxidative stress has been increasing rapidly (Dasdag et al., 2004, 2008, 2009).

Proteins also constitute one of the major targets of reactive oxygen species (ROS). Oxidation of proteins can lead to a loss of protein function as well as conversion of proteins to forms that are more susceptible to degradation by proteinases. However, Barreiro et al. (2005) stated that investigators have described carbonylation of several cytosolic proteins including β-tubulin, β-actin, and creatine kinase in brain samples of patients with Alzheimer's disease. Alzheimer's disease (AD) is one of the most common diseases in older people and it was recently reported that exposure to radiofrequency fields (RF) provides cognitive benefits for both normal and transgenic mice (Arendash et al., 2010). Arendash et al. (2010) stated that long-term exposure of radiofrequency field (RF, 918 MHz; 0.25 W/kg) provided cognitive benefits in an Alzheimer's diseases (AD) mice by means of reducing brain amyloid-beta (Abeta) deposition. They also stated that radiofrequency radiation exposure reduced brain amyloid-beta (A beta) deposition through decreased aggregation of A beta and increase in soluble A beta levels (Arendash et al., 2010). However, Soderqvist et al. (2010) stated that in a provocation study, they exposed temporal area of brain of 41 people for 30 min to an 890-MHz GSM signal with specific absorption rate of 1.0 Watt/kg and found a significant increase in serum transthyretin (TTR) 60 min after exposure. However, in their cross-sectional study, they found that use of oral snuff also yielded increased serum TTR concentrations and they also stated that nicotine has been associated with decreased risk for Alzheimer's disease and to up regulate the TTR gene in choroid plexus. As far as the authors of the study stated, TTR sequesters amyloid beta, thereby preventing the formation of amyloid

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beta plaques in the brain and patients with Alzheimer's disease have lowered TTR concentrations in the cerebrospinal fluid and have attributed the onset of Alzheimer's disease to insufficient sequestering of a beta by TTR. Based on Soderqvist et al.'s findings of increased serum concentrations of transthyretin (TTR) among long-term users of wireless phones, i.e., mobile phone and cordless phone, and in a provocation study they postulate that TTR might be involved in the preventive effect on Alzheimer's disease by exposure to radiofrequency radiation (Soderqvist et al., 2009a,b, 2010).

Because of the importance of the parameters such as beta amyloid protein, protein carbonyl, and malondialdehyde in living systems, the aim of this study is to investigate long-term effects of 900 MHz mobile phone exposure on the parameters mentioned here in rat brain.

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MATERIALS AND METHODS

Seventeen Wistar Albino adult male rats were obtained from the Medical Science Application and Research Center of Dicle University (Diyarbakır, Turkey), caged individually, and fed with standard pelletted food (TAVAS Inc., Adana, Turkey). They were separated into two groups, sham exposed (n: 7) and exposure (n: 10), and kept on a 14/10 h light/dark schedule. During the study, the ambient temperature (22°C) and relative humidity (45%) were maintained in the normal range for these animals. All animal procedures were in agreement with the Principles of Laboratory Animal Care and the rules of Scientific and Ethics Committee of Dicle University Health Research Center.

Exposure and Measurement of Radiation

A generator (GSM Simulator 900PM10 type Everest Comp., Adapazarı, Turkey), which produces 900 MHz radiofrequency radiation, was used in this study to represent exposure of global systems for mobile communication (GSM). Emitted power (circular space distribution) of the generator was fixed at 2W during exposure. Antenna of the generator was equivalent to antenna of mobile phones. The rats were confined in a Plexiglas carousel, and rat heads in the carousel were exposed to 900 MHz microwave exposure emitted from the generator. For the study group, rats were exposed to the radiation 2 h per day (7 days a week) for 10 months. For the sham group, rats were placed into the carousel and the same procedure was applied to the rats (2 h/day/7 day in a week for 10 months), except that the generator was turned off. Antenna of the generator was placed at the center of Plexiglas carousel to provide ideal exposure. Distance of antenna to head of the rats was 1 cm. Power Density inside Plexiglas carousel cages was measured by EMR 300 (NARDA, Pfullingen, Germany).

On the last day of the study, immediately after the last exposure, the rats were euthanized with a lethal dose of intraperitoneal pentobarbital, and brains were removed for measurement of MDA, protein carbonyl and beta amyloid protein.

Determination of Thiobarbituric Acid Reactive Substances (TBARS) in the Tissue Samples

Thiobarbituric acid reactive substances (TBARS) as an indicator of lipid peroxidation were determined fluorometrically (Levine et al., 1990). Briefly, 1 ml of each sample was mixed with 1 ml of trichloroacetic acid (TCA) 10% and 1 ml of TBA 0.67% and then heated in a boiling water bath for 15 min. Tubes were chilled on ice and the rose-colored trimethin-complex was extracted into 3 ml of n-butanol. The organic phase was separated by centrifugation for 10 min at 3000 rpm, malondialdehyde (MDA) an intermediate product of lipoperoxidation, was determined by absorbance

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at 535 nm. A standard curve for TBARS was prepared with 1, 1, 3, 3-tetramethoxypropanol in a concentration range of 0.1–10 nmol.

Determination of Protein Carbonyls in Tissue Samples

The oxidative damage to proteins was measured by the quantification of carbonyl groups based on the reaction with 2, 4-dinitro-phenylhydrazine (DNPH). 500 μl of tissue homogenates were mixed with 2 ml dinitrophenylhydrazine (0.2% in 2.5 mol/L HCl) and incubated for 1 h at room temperature. 5 ml TCA solution (20%) were added to the samples for protein precipitation. The tubes were cooled in an ice bath for 10 min and subsequently centrifuged at 10,000 × g for 5 min to collect the protein fraction. The protein pellets were washed once with 4 ml TCA (10 g/L) and three times with 4 ml of a mixture of ethanol/ethylacetate (1:1, v/v). The resulting protein precipitates were dissolved in guanidium HCl (6 mol/L in 20 mmol/L potassium phosphate, pH 2.3) and incubated for 10 min at 37°C. The absorbance was read at 370 nm (Levine et al., 1990).

Measurement of Beta Amyloid Protein

Beta amyloid was analyzed by using ELISA (sandwich enzyme- linked immunosorbent assay) (Uscn Life Science Inc. Wuhan- Mouse Amyloid Beta peptide 1-40(Ab1-40). In this study, standards or samples were added to the appropriate microtiter plate wells with a biotin-conjugated polyclonal antibody preparation specific for Aβ1-40. Next, Avidin conjugated to Horseradish Peroxidase (HRP) was added to each microplate well and incubated. Then a TMB substrate solution was added to each well. Only those wells that contain Aβ1-40 biotin-conjugated antibody and enzyme-conjugated Avidin will exhibit a change in color. The enzyme-substrate reaction was terminated by the addition of a sulphuric acid solution and the color change was measured spectrophotometrically at a wavelength of 450 nm. The concentration in the samples was then determined by comparing the O.D. of the samples to the standard curve. The detection limit (LOD) was 3.12–200 pg/ml. The minimum detectable dose of human InhB was typically less than 1.6 pg/ml.

Measurement of Power Density and Specific Absorption Rate (SAR)

Power density and electrical field measurements were performed by EMR 300 radiation meter (NARDA, Pfullingen, Germany). Average power density around the brain was measured between 0.05–0.33 mW/cm^2. However, electric field was measured between 16.2–29.4 V/m. The specific absorption rate was approximately calculated according to formula: SAR $= \sigma/\rho\ |E_{rms}|^2$ [mW/g], where E_{rms} is the root mean square value of the electrical field, σ is the mean electrical conductivity of tissue, and ρ is the mass intensity. The values used to measure SAR based on article of Peyman et al. (2001). SAR values were found approximately between 0.17–0.58 W/kg, adopting $\sigma = 0.7$ S/m and $\rho = 1040$ kg/m^3.

Statistical Analysis

Nonparametric Mann-Whitney U test was used to analyze the results of the study. A computer program (Med Calc) was used for statistical analysis.

RESULTS

The results of this study showed that long-term exposure of 900 MHz radiation used in this study increased MDA, Protein carbonyl and beta amyloid protein in the brain of the rat (Table 1). However, protein carbonyl was only found to be statistically significant ($p < 0.001$). Average power density around the brain was measured

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TABLE 1 Comparison of the results of sham and exposure group (n: sample size).

Groups	Parameters	Mean	SD	Median	P
Sham (n = 7)	MDA (nmol/ml)	7.499	0.9512	7.680	**> 0.05**
Exposure (n = 10)	MDA (nmol/ml)	8.546	0.9310	8.100	
Sham (n = 7)	Protein Carbonyl (nmol/mg protein)	0.749	0.01773	0.750	**< 0.001**
Exposure (n = 10)	Protein Carbonyl (nmol/mg protein)	0.830	0.05055	0.810	
Sham (n = 7)	Beta amyloid protein (Aβ 40) (pg/ml)	266.642	7.6212	267.640	**> 0.05**
Exposure (n = 10)	Beta amyloid protein (Aβ 40) (pg/ml)	276.420	10.8691	279.070	

between 0.052–0.338 mW/cm^2. However, electric field was measured between 16.26–29.43 V/m.

DISCUSSION

The frequent use of mobile and cordless phones in our daily life has led to concern regarding possible health effects, cancer risk in particular, from frequent exposure to radiofrequency radiation. Radiofrequency radiation can be a physical agent to affect biological systems by increasing free radical production. Some of the studies were performed to know whether radiofrequency field can modulate oxidative stress and antioxidant systems in tissues by measuring free radical levels and antioxidant enzyme activities (Dasdag et al., 2004, 2008, 2009; Yokus et al., 2008). The number of studies on relation between mobile phone exposure and oxidative stress increased for explaining the mechanism of some determined adverse effects (Dasdag et al., 2004, 2008, 2009). Lai and Singh (1997, 2005) also reported that radiofrequency radiation can cause an increase in DNA single- and double-strand breaks in brain cells of the rat. However, scientific evidence on a possible mobile phone and cancer relation has still not clear enough. Therefore, investigators have still been showing high performance to clarify this topic (Dasdag et al., 2009).

Under normal circumstances, cells are able to defend themselves against damage sourced from reactive oxygen species (ROS). However, sometimes it may not be possible because of exogenous physical agents such as radiation. ROS elicit a variety of modifications in amino acid residues, including cysteines, methionine, tryptophan, arginine, lysine, proline, and histidine. Among amino acid modifications by ROS is the formation of carbonyls as a result of oxidation of arginine, lysine, threonine, or proline amino acids. Protein carbonylation is the result of secondary reactions of amino groups of lysine residues with reducing sugars or their oxidation production (glycation/glycoxidation reactions) and also by reactions of lysine, cysteines, or histidine amino acids with α- and β-unsaturated aldehydes formed during the peroxidation of polyunsaturated fatty acids. Despite this wide use of protein carbonyl detection as an index of oxidative modification of proteins, little information is yet available about the exact identity of proteins targeted by ROS (Barreiro et al., 2005).

Ozgur et al. (2010) stated that lipid peroxidation is one of the major consequences of oxidative stress and malondialdehyde (MDA) is the end-product of lipid peroxidation by reactive oxygen species. It is therefore generally used as a biomarker for the oxidative stress of an organism. However, the authors observed a significant increase in malondialdehyde (MDA) level in the liver of guinea pigs after 1800 MHz radiofrequency exposure (Ozgur et al., 2010)

In our previous study, we measured levels of catalase (CAT), myeloperoxidase (MPO), malondialdehyde (MDA), total antioxidant capacity (TAC), total oxidant status (TOS), and oxidative stress index (OSI) in the livers of rats exposed to long-term 900 MHz radiofrequency radiation. We observed that long-term 900 MHz

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exposure increased MDA and TOS in liver. Therefore, we stated that RF radiation emitted from GSM cellular phone may play a role to induce oxidative damage by; increasing lipid peroxidation and oxidative stress (Dasdag et al., 2008). In this study, parallel to our previous study, we also observed an increase in MDA level in rat brain. However, the increase of MDA observed in rat brain was not found to be significant statistically ($p > 0.05$).

Yokus et al. (2008) showed that ELF-MF exposure generates oxidatively induced DNA base modifications which are mutagenic in mammalian cells, such as FapyGua, FapyAde, and 8-OH-Gua, in vivo. In this study, we observed that long-term exposure of 900 MHz radiofrequency radiation increased protein carbonyl level in rat brain ($p < 0.001$). These findings support the hypothesis that long-term mobile phone exposure may be potentially genotoxic in brain. However, the intensity and exposure duration of exposure is an important parameter in this process (Yokus et al., 2008). Dasdag et al. (2004) investigated the effects of cell phone exposure on the fatty acid composition in phospholipids, malondialdehyde concentration, p53 immune reactivity, and histological structure of the rat brain. They did not find any histological changes in brain phospholipid fatty acids composition in rat. However, they found that malondialdehyde concentration in exposed brains was significantly higher than sham (Dasdag et al., 2004). The results of this study are also parallel to the results of Dasdag et al. (2004). However, the result of MDA, which is measured in this study, was not found to be significant statistically ($p > 0.05$). On the other hand, results of protein carbonyl was found to be statistically significant in this study ($p < 0.001$).

Guler et al. (2009) stated that protein carbonyl content (PCO) may be classified as the most general indicator and by far the most commonly used marker of ROS mediated protein oxidation. As they stated, oxidative damage can modify proteins once it reaches significant levels in the body and following the exposure of a protein to ROS, some amino acid side chains are modified, leading the protein structure to significant alterations (Guler et al., 2009)

Memory capabilities decline with age, evident in human degenerative diseases such as Alzheimer's disease, which is accompanied by an accumulation of oxidative damage. Current studies demonstrate that the accumulation of ROS can decrease an organism's fitness because oxidative damage is a contributor to senescence. In particular, the accumulation of oxidative damage may lead to cognitive dysfunction, as demonstrated in a study in which old rats were given mitochondrial metabolites and then given cognitive tests. Results showed that the rats performed better after receiving the metabolites, suggesting that the metabolites reduced oxidative damage and improved mitochondrial function. Accumulating oxidative damage can then affect the efficiency of mitochondria and further increase the rate of ROS production. The accumulation of oxidative damage and its implications for aging depends on the particular tissue type where the damage is occurring. Additional experimental results suggest that oxidative damage is responsible for age-related decline in brain functioning. Older gerbils were found to have higher levels of oxidized protein in comparison to younger gerbils. When old and young mice were treated with a spin trapping compound, the level of oxidized proteins decreased in older gerbils but did not have an effect on younger gerbils. In addition, older gerbils performed cognitive tasks better during treatment but ceased functional capacity when treatment was discontinued, causing oxidized protein levels to increase. This lead researcher to conclude that oxidation of cellular proteins is potentially important for brain function (Wikipedia, 2011). As is mentioned above, we found that long-term exposure of 900 MHz radiofrequency radiation increased protein carbonyl level in brain ($p < 0.001$). Therefore, it can be said that long-term

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exposure of radiofrequency radiation may cause some cognitive effects. However, it should be noted that many of studies showed that mobile phone exposure has a capacity to change cognitive process during driving.

In a study, Arendash et al. (2010) claimed that long-term cell phone use (918 MHz; 0.25 W/kg) provides cognitive benefits. They showed in mice with Alzheimer's disease that long-term EMF exposure reduced brain amyloid-beta (A beta) deposition through decreased aggregation of A beta and increase in soluble A beta levels. (Arendash et al., 2010). However, Soderqvist et al. (2010) proposed that transthyretin (TTR) might be involved in the findings of RF exposure benefit in Alzheimer's disease mice. In our study, we measured beta amyloid protein in healthy rats which were exposed to long-term 900 MHz radiofrequency radiation. We did not find any alteration in amyloid beta level of sham and exposed rat brains ($p > 0.05$). In light of the results of the studies of Arendash et al. (2010), we observed that there is not a parallel between the results of them and our study. We believe that contradiction between our studies may be sourced from the type of animal and exposure characteristics, because they exposed Alzheimer's disease mice while we exposed healthy rats.

In conclusion, our data showed that 10 months of exposure (2 h per day) of 900 MHz radiation increased protein carbonyl level in healthy rat brain tissue. Therefore, we can state that long-term exposure of 900 MHz exposure may alter some parameters in living systems.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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