Electromagnetic Biology and Medicine

ISSN: 1536-8378 (Print) 1536-8386 (Online) Journal homepage: https://www.tandfonline.com/loi/iebm20

The effects of long-term exposure of magnetic field via 900-MHz GSM radiation on some biochemical parameters and brain histology in rats

Saadet D. Celikozlu, M. Sabri Ozyurt, Ali Cimbiz, Melda Y. Yardimoglu, M. Kasim Cayci & Yusuf Ozay

To cite this article: Saadet D. Celikozlu, M. Sabri Ozyurt, Ali Cimbiz, Melda Y. Yardimoglu, M. Kasim Cayci & Yusuf Ozay (2012) The effects of long-term exposure of magnetic field via 900-MHz GSM radiation on some biochemical parameters and brain histology in rats, Electromagnetic Biology and Medicine, 31:4, 344-355, DOI: 10.3109/15368378.2012.662192

To link to this article: https://doi.org/10.3109/15368378.2012.662192

Published online: 07 Jun 2012.

Electromagnetic Biology and Medicine, 31(4): 344–355, 2012

ISSN: 1536-8378 print / 1536-8386 online
DOI: 10.3109/15368378.2012.662192

informa
healthcare

The effects of long-term exposure of magnetic field via 900-MHz GSM radiation on some biochemical parameters and brain histology in rats

Saadet D. Celikozlu[1], M. Sabri Ozyurt[2], Ali Cimbiz[3], Melda Y. Yardimoglu[4], M. Kasim Cayci[2] & Yusuf Ozay[5]

[1]Altintas Vocational School of Dumlupinar University, Altintas, Kütahya, Turkey, [2]Department of Biology, Dumlupinar University, Kutahya, Turkey, [3]Health Institution of Dumlupinar University, Kutahya, Turkey, [4]Department of Histology and Embriology, Kocaeli University, Kocaeli, Turkey, and [5]Health Institution of Ahi Evran University, Kirsehir, Turkey

The aim of this study is to determine the effects of magnetic field via cell phones on some blood parameters and neurons in the brain of rats. Animals have been classified into three groups: control, Magnetic Field (MF), and F2 groups. Throughout this study, cell phones were placed on the wall of the cages. Rats were exposed to the effects of cell phones during prenatal and postnatal periods until they were 80 days old. During the study, the exposure procedure of rats was that the phone was in standby mode for a whole day and in talking mode for 30 min per day. The waves of cell phones caused an increased blood glucose level from 96.52 $\pm$ 5.64 mg/dl to 132.14 $\pm$ 5.93 mg/dl and an increased serum protein level from 131.14 $\pm$ 6.19 mg/dl to 319.29 $\pm$ 6.73 mg/dl compared to control. Statistically, significant differences wasn't observed in the blood cholesterol concentration between the groups compared to the control. Weekly weight gain decreased in all groups compared to the control. MF exposure decreased pyramidal neuron numbers 51.15% and increased ischemic neuron numbers 73% at cortex region of brain. In addition, vascular dilatations have increased clearly in group F2.Whereas the procedure of MF did not have any effects on hippocampal pyramidal cell numbers, magnetic fields increased the amount of ischemic neurons three-fold compared to the control. In conclusion, MF affected some biochemical parameters, especially the cortex region of the brain.

Keywords Biochemistry, Brain, Histopathology, Mobile phone, Rat

INTRODUCTION

Numerous biochemical and histological studies have been carried out to evaluate the effects of electric and magnetic fields on the metabolism of cell cultures, animals, and humans (Ahlbom, 2001; Boguslaw et al., 1999; Hakansson et al., 2003; Skinner and Mee, 2002). Magnetic Field (MF) penetrate the human body and act on all organs, altering the cell membrane potential and the distribution of ions and dipoles (Loew, 1992). These alterations may influence biochemical processes in the cell, thus changing both biochemical and enzyme activities of serum. Previous reports showed significant distribution in the carbohydrate, lipid, and protein metabolism reflected by altered

Address correspondence to Saadet D. Celikozlu, Altintas Meslek Yuksek Okulu, 43800, Altintas, Kutahya, Turkey. E-mail: scelikozlu@hotmail.com

blood glucose levels and accelerated glycosis and glyconeogenolysis (Boguslaw et al., 1999; Kumosani and Qari, 2001). MF is known to be strongly lypolytic and glycogenolytic in rats, inducing a prominent increase in blood glucagon, cortisol, and thyroxin levels (Chernysheva, 1990; Gorczynska, and Wegrzynowicz, 1991). Exposure of mice to MF suppressed the eating and drinking behavior and increased the blood urea nitrogen, glucose, and kreatinin concentrations (Tsuji et al., 1996).

Epidemiological studies suggest a possible link between MF exposure and clinically recognized medical disorders in people, such as leukemia, brain cancer, breast cancer, kidney cancer, and other kinds of cancers, as well as cardiovascular diseases (Bethwaite et al., 2001; Hakansson et al., 2003; Skinner and Mee, 2002). The characteristic biological effects of MF appear to be functional changes in the central nervous, endocrine, immune, and reproduction systems (Ahlbom, 2001; Aitken, 2005; Elbetieha et al., 2002).

One of the most important MF sources is a cell phone. The most widely used GSM cell phones operate with microwave carrier frequencies in the near GHz range (850–1900 MHz). In addition, the GSM radiation contains ELF components too, because the carrier signal is pulsed at 217 Hz (Hyland, 2000).

Mobile phones and their base stations produce radiofrequency (RF) radiation. RF is absorbed in the body and produces heat, but the body's normal thermoregulatory processes carry this heat away. All established health effects of RF exposure are related to thermal effect. Since RF 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 and an associated very low rise in brain temperature (maximum, 0.1°C) (Van Leeuwen et al., 1999). Thus, the possible biological effects from cellular phone use would not be expected to be thermal in nature. Furthermore, because RF does not possess enough energy to remove electrons from atoms or molecules, it is impossible for ionization to occur. The RF is thus termed ''non ionizing'' and is very different from ionizing radiations of much higher frequencies, such as X-rays and g-rays, which are genotoxic and known to damage DNA molecules either directly or indirectly through free-radical formation. The greatest mystery about the mobile phones is their non thermal effects on living tissues. 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 (Stam, 2010). In particular, a variety of neurological effects have been postulated to occur as a result of exposure to EMR, including headache (Frey, 1998), changes in sleep patterns (Wagner et al., 1998), modifications in the electroencephalogram (EEG) (Freude et al., 2000), and an increase in blood pressure (Braune et al., 1998). Exposure to 900 MHz EMF decreases neuron number and causes neuronal damage in the cortex, cerebellum, hippocampus, and basal ganglia in animals (Ammari et al., 2008; Brillaud et al., 2007; Mausset et al., 2001; Salford et al., 2003).

Ilhan et al. (2004) demonstrated that mobile phones cause oxidative damage biochemically by increasing the levels of nitric oxide, malondialdehyde, as well as xanthine oxidase and adenosine deaminase activities in brain tissue in a rat model of exposure to RF. Recent studies have shown an increasingly important role for oxidative stress in the pathogenesis a number of pathological entities including inflammation (Neumann, 2001), AIDS (Westwndorp et al., 1995), atherosclerosis (Keaney and Vita, 1995), multiple sclerosis (Smith et al., 1999), and carcinogenesis (Adelman et al., 1988), but also in physiological ageing (Oliver et al., 1987). Because the nervous system is particularly vulnerable to reactive oxygen species (ROS) due to its high metabolic rate,

its deficient oxidant defense mechanisms and its diminished cellular turn over, oxidative stress is discussed as a contributor to the initiation or progression of neurodegenerative diseases. Various neurodegenerative disorders such as Parkinson's disease (Fahn and Cohen, 1992), Alzheimer's disease (Benzi and Moretti, 1995), and amyotrophic lateral sclerosis (Bergeron, 1995) have been causally linked to the generation of reactive oxygen species and oxidative stress.

Furthermore, RF radiation might alter intracellular signaling pathways through changes in ionic distribution and membrane fluidity (Hossmann and Hermann, 2003) or change Ca^{2+} permeability across cell membranes (Adey, 1981). RF could also alter the conformational energy of glycoproteins in the cell membrane to open Ca^{2+} channels (Thomas et al., 2000). These changes could cause pathophysiological changes in the brain such as tumorigenesis, neural degeneration, and cognitive deficits.

Several studies showed acute effects of MF at prenatal or postnatal period. In contrast, less is known about chronic effects of MF. Hence, the present study was carried out to determine chronic effects of MF on both prenatal and postnatal rats.

MATERIAL AND METHODS

Animal studies were performed after taking approval from Animal Care and Ethic Committee of Medical Faculty of Dumlupinar University and "Principles of laboratory animal care" (NIH publication no. 605–873, received) guidelines were followed.

Animals

Albino Wistar male and female rats obtained from the central animal house of Dumlupinar University were used for the study. Rats were housed individually in cages, maintained under standard condition (12 h light / 12 h dark cycle; 25 $\pm$ 3^{o}C), and were fed with standard pellet and water ad libitum. At the time of this study, three groups were designed: **1**- Control, **2**- Magnetic field (MF) (F1 generation), and **3**- F2 generation group with MF.

Exposure Procedure

A conversional commercially available cellular telephone with Global System for Mobile Communication (GSM-900) digital technology was used for MF exposure. In assays, cell phones were placed inside wall of cages. Rats were exposed to the effect of cell phones during prenatal and postnatal periods until they were 80 days old. During the study, the exposure procedure of the phone was in standby mode for the whole day and in talking mode for 30 min per day. The control group was kept in the same conditions without exposure to GSM. The control group cage positioned 8 m away from other cages. Therefore, the effect of MF on other animals was prevented. Body weight was recorded twice per week during the study.

Blood Glucose and Total Cholesterol Determination

Capillary blood glucose and cholesterol was determined by glucose and total cholesterol test strips (Roche Accutrend, Roche Diagnostics GmbH Firm, Mannheim, Germany).

Total Serum Protein Determination

Rats were sacrificed with cervical dislocation. Blood for serum protein determination was collected from jugular vein. Serum samples were separated by centrifugation and total protein was detected with spectrophotometric method. Total protein determination kit was obtained from Biocode Hycel Firm, Liege, Belgium.

TABLE 1 The comparison of effects of the magnetic field exposure from mobile phone on blood glucose, cholesterol, protein levels, and body weight in rats.

Parameters*	Control (F1)	MF (F1)	F2 (with MF)
Blood Glucose (mg/dl)	96.57 ± 5.64[a]	132.14 ± 5.93[b]	99.43 ± 3.42[a]
T. Blood Cholesterol (mg/dl)	146.86 ± 0.99[a]	155.86 ± 2.89[a]	153.71 ± 4.16[a]
T. Blood Protein (mg/dl)	131.14 ± 6.19[a]	319.29 ± 6.73[b]	265.43 ± 18.22[c]
Body Weight (gr)	15.71 ± 0.88[b]	10.21 ± 0.88[a]	11.39 ± 0.94[a]

Data are presented as Mean ± SD, MF: Magnetic Field, F1: F1 generation, F2: F2 generation. *Values with same letter are statistically not significant ($p < 0.05$). Values with different letters are statistically significant ($p > 0.05$).

Microscopic Measures and Counting

Investigations were done with coronal brain sections. In this study, Histology Atlas of Di Fiore (Eroschenko, 2001) and stereotaxic rat brain atlas of Paxinos (Paxinos and Watson, 1994) were used. Neuron counting was done at semi-quantitative image area that was calculated with micrometric ocular (Manesse et al., 1998). The counting as 0.0255 mm^2 in an image area on cortex and hippocampus were made totally by 0.255 mm^2 in 10 image areas.

Statistical Analyses

The results were expressed as mean± SD; statistical differences between experimental and control groups were determined by one-way analysis of variance (ANOVA) followed by Post-Hock multiple comparisons Tukey tests using SPSS 14.0 software. The level of significance was set to $p < 0.05$.

RESULTS

The detailed biochemical results of the present study are given in Table 1 for all groups.

At the MF group, the mean glucose concentration increased significantly when compared to control ($p < 0.05$). However, it did not cause significant alteration in blood glucose concentration of F2 group compared to control ($p > 0.05$). Statistically significant difference was not observed between groups at mean cholesterol level compared to control group ($p > 0.05$). MF exposure caused a statistically significant increase in blood total protein level of MF and F2 groups compared to control ($p < 0.05$). After MF exposure, a significant decrease was observed in average body weight increases of all groups compared to control group ($p < 0.05$).

Histological results of the study are presented in Table 2 and Fig 1–6 for all groups.

At the MF group, the average number of cortical pyramidal cell was decreased of 51.15% and average number of cortical ischemic cell increase of 73%. There was no

TABLE 2 The comparison of the effects of the magnetic field exposure on cortical and hippocampal neuron numbers in rats.

Groups	Parameters*			
	Pyramidal Neuron Number		Ischemic Neuron Number	
	Cortex	Hippocampus	Cortex	Hippocampus
Control (F1)	165.80 ± 5.65[b]	259.00 ± 17.48[a]	13.70 ± 2.34[a]	21.90 ± 1.27[a]
MF (F1)	81.00 ± 7.97[a]	181.00 ± 26.10[a]	23.70 ± 3.18[b]	66.20 ± 7.57[b]
F2 (with MF)	152.70 ± 5.51[b]	246.40 ± 22.85[a]	18.60 ± 1.43[a]	29.00 ± 2.09[a]

Data are presented as Mean ± SD, MF: Magnetic Field, F1: F1 generation, F2: F2 generation. *Values with same letter are statistically not significant ($p < 0.05$). Values with different letters are statistically significant ($p > 0.05$).

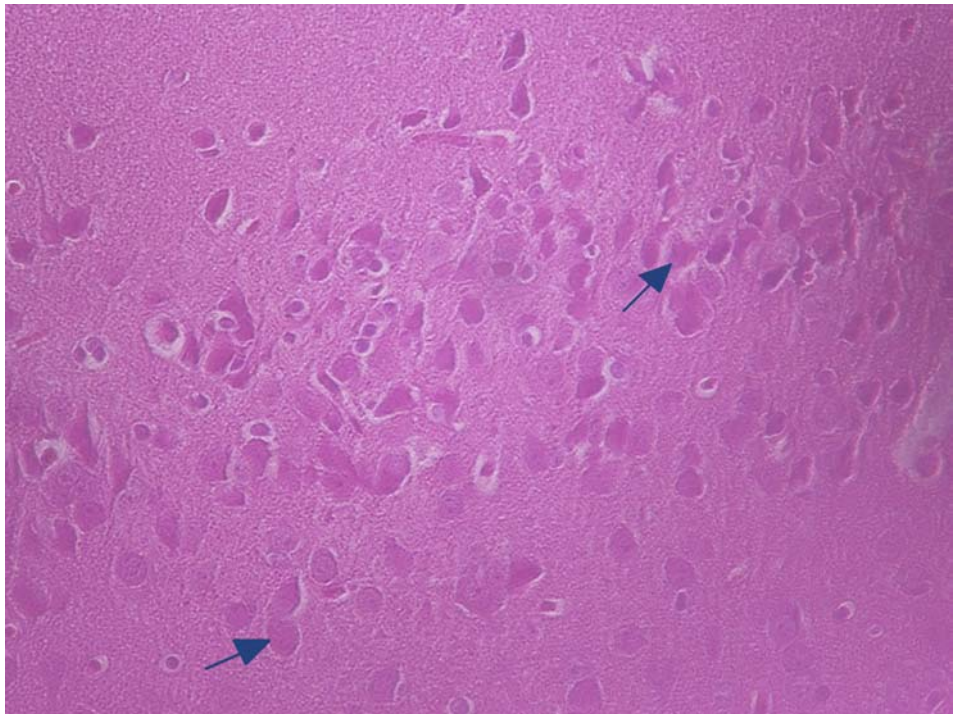

FIGURE 1 Pyramidal neurons of control group at cortex region of brain. H&E, 40X.

significant difference in average number of cortical pyramidal and ischemic neuron of F2 group compared to control ($p < 0.05$). Significant increase was observed in number of vascular dilatation of F2 group compared to control. MF exposure did not change hippocampal pyramidal cell number of MF group ($p > 0.05$) but MF induced a statistically significant increase in hippocampal ischemic neuron number of MF group compared to control ($p < 0.05$). Hippocampal pyramidal and ischemic neuron numbers of F2 group were not changed compared to control ($p > 0.05$), whereas a number of vascular dilatation of F2 group was increased significantly when compared to the control group.

DISCUSSION

The results showed that the exposure to MF originates different biochemical and histological distributions on living organisms.

In the present study, results show that the exposure to MF causes an increase in blood glucose level of MF group compared to control. The observation of hyperglycaemia, following the exposure of MF, could be explained by structural and functional changes in the pancreas, in response to MF stress (Amara et al., 2006). Gorczynska and Wegrzynowicz (1991) reported that an increase of serum glucose level is associated with a decrease of insulin and an increase of glucagon level. This might implicate a diabetic-like response in rats exposed to the MF (Chater et al., 2006). Another reason for the increase of blood glucose might be a condition of stress. The reason for that result is the monoamine metabolism is influenced by MF (Kabuto et al., 2000). Also, Koyu et al. (2005) showed a significant increase of cortisol level after MF exposure. In addition to this, MF causes the modification of Ca^{+} influx through Ca^{+} channels and the attenuations of insulin secretion. This also causes an

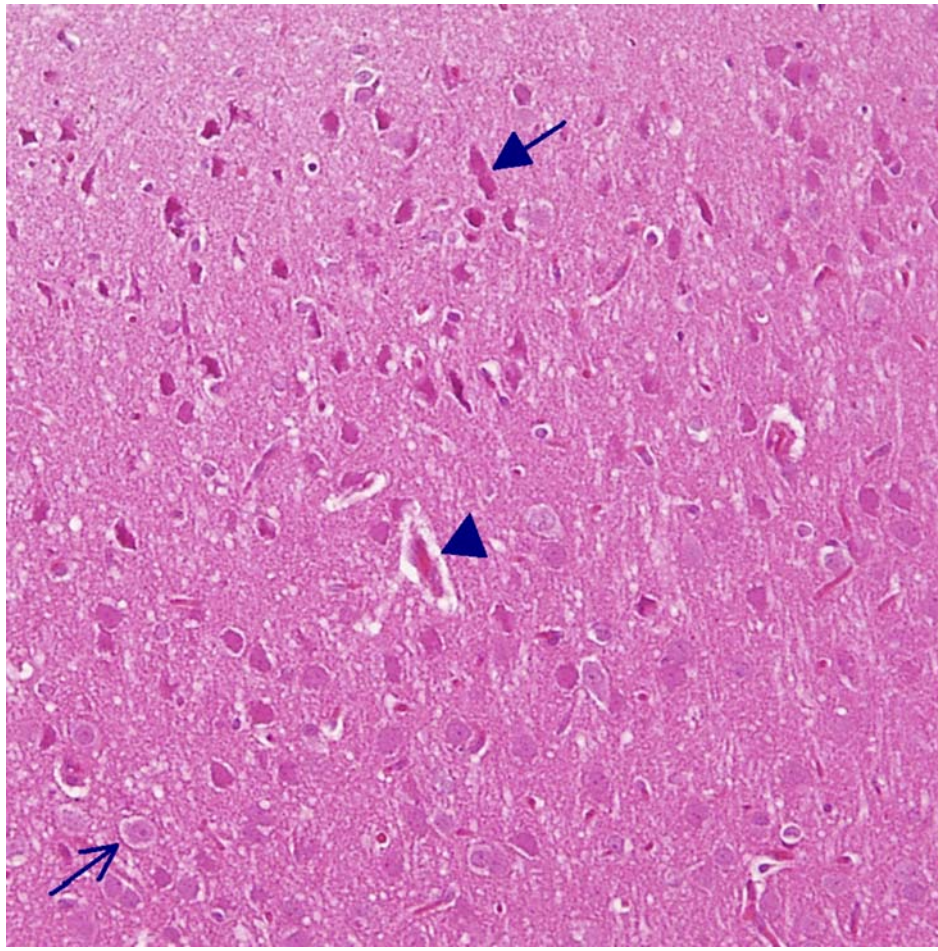

FIGURE 2 Pyramidal (→), ischemic (⇒) neurons and vascular dilatations (▽) of MF group at cortex region of brain. H&E, 20X.

increase in blood glucose level (Schwartz et al., 1990). Results show that exposure to MF did not cause any significant alteration in blood glucose concentration of F2 generation with MF compared to control group. The lack of effect on glycaemia observed in F2 rats suggests a probability of an adaptive response of carbohydrate metabolism subsequent of a long exposure.

In this study, a statistically significant difference was not observed in blood cholesterol level between groups and controls. Previous studies support our findings. Kim et al. (2006) reported that chronically exposure to MF showed no significant effect in blood cholesterol level. But, at some studies, acute exposure to MF caused different results. Babych (1995) recorded that after 5 days' exposure to MF reduces the blood cholesterol level. Also, Chernysheva (1990) showed that exposure to 50 Hz MF for 15 days increased plasma cholesterol esters. The present study shows that no significant effect on cholesterol level after chronically exposure to MF suggests probably an adaptive response of lipid metabolism.

Present results show that MF exposure causes an increase in total protein level of MF group compared to control group. Boguslaw et al. (1999) showed a slight decrease of total serum protein levels in steelworkers exposed to electromagnetic field. This discrepancy could be attributed to the difference of the intensity of the MF and exposure scenario and duration. MF increase transcript levels of specific genes.

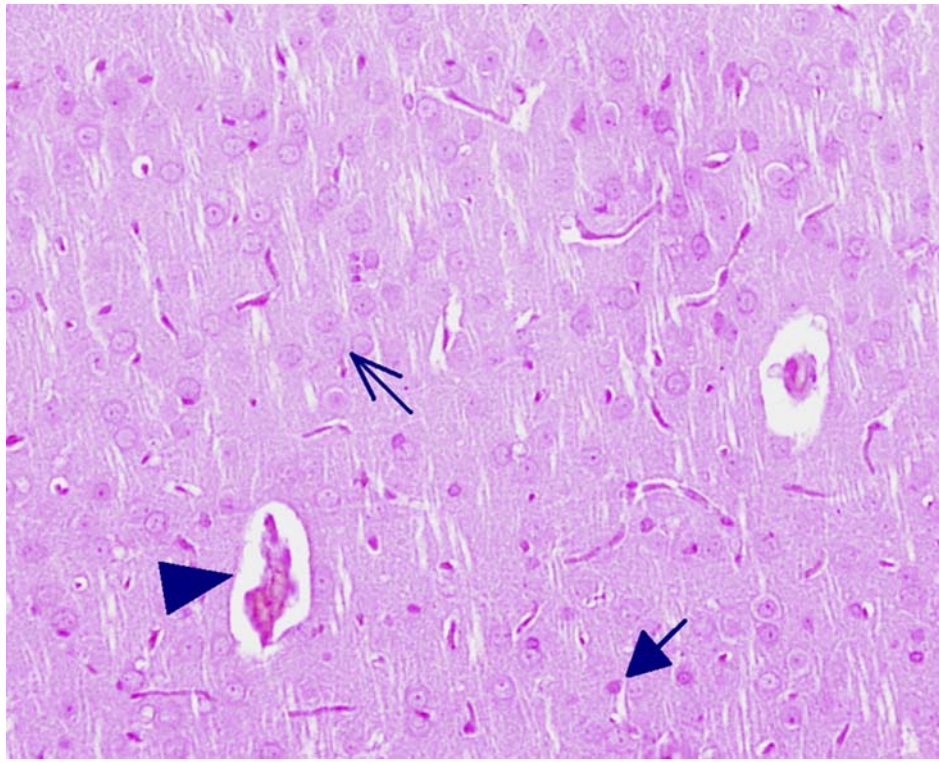

FIGURE 3 Pyramidal (→), ischemic (⇒) neurons and vascular dilatations (▽) of F2 group at cortex region of brain. H&E, 20X.

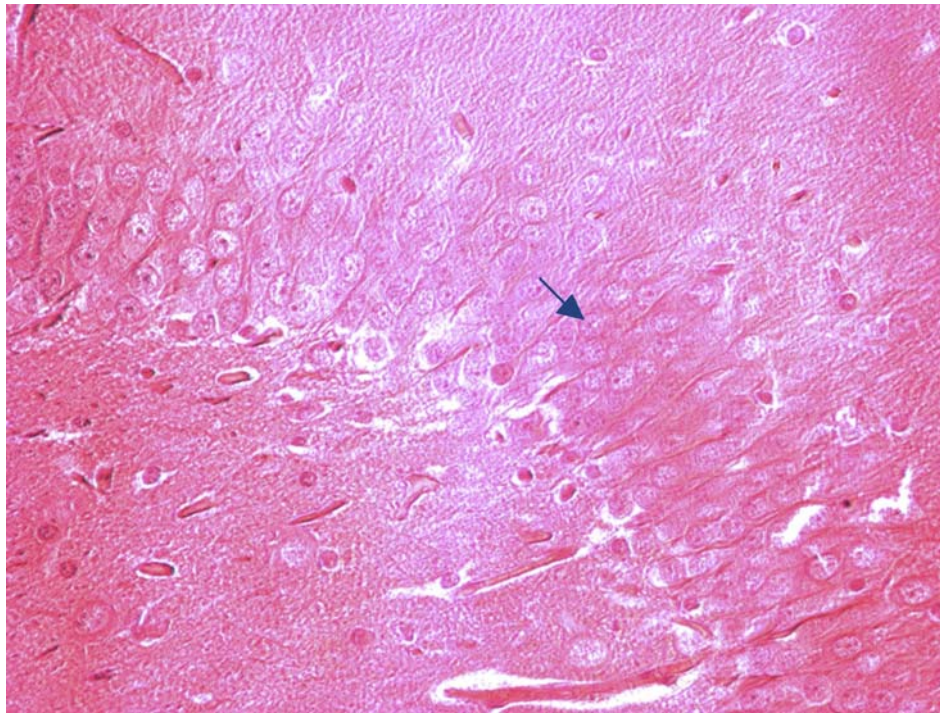

FIGURE 4 Pyramidal neurons of control group at hippocampus region of brain. H&E, 40X.

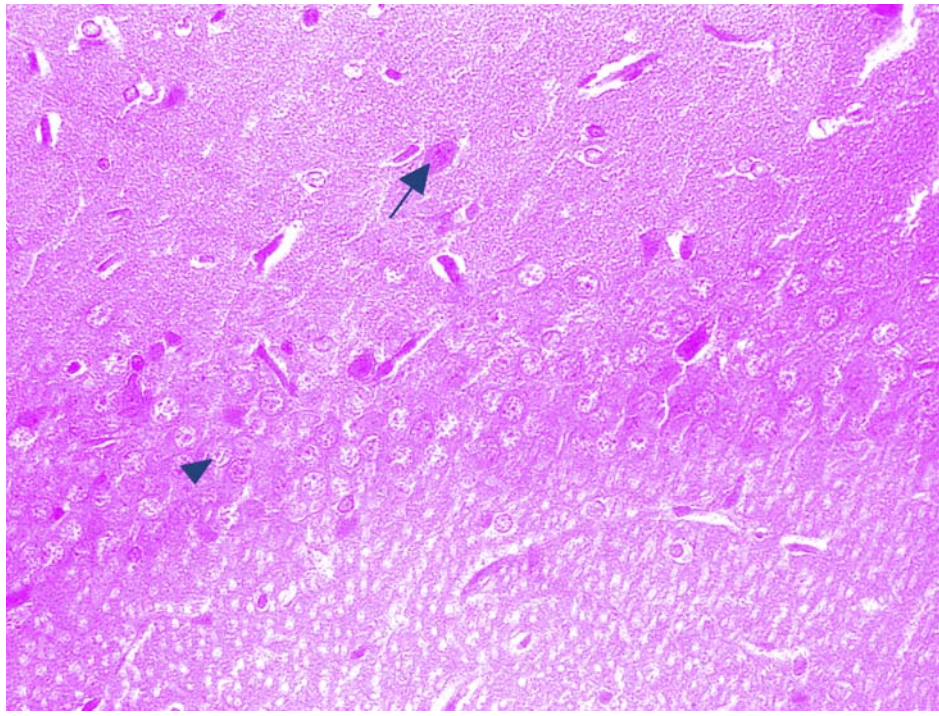

FIGURE 5 Pyramidal (▽) and ischemic (⇒) neurons of MF group at hippocampus region of brain. H&E, 40X.

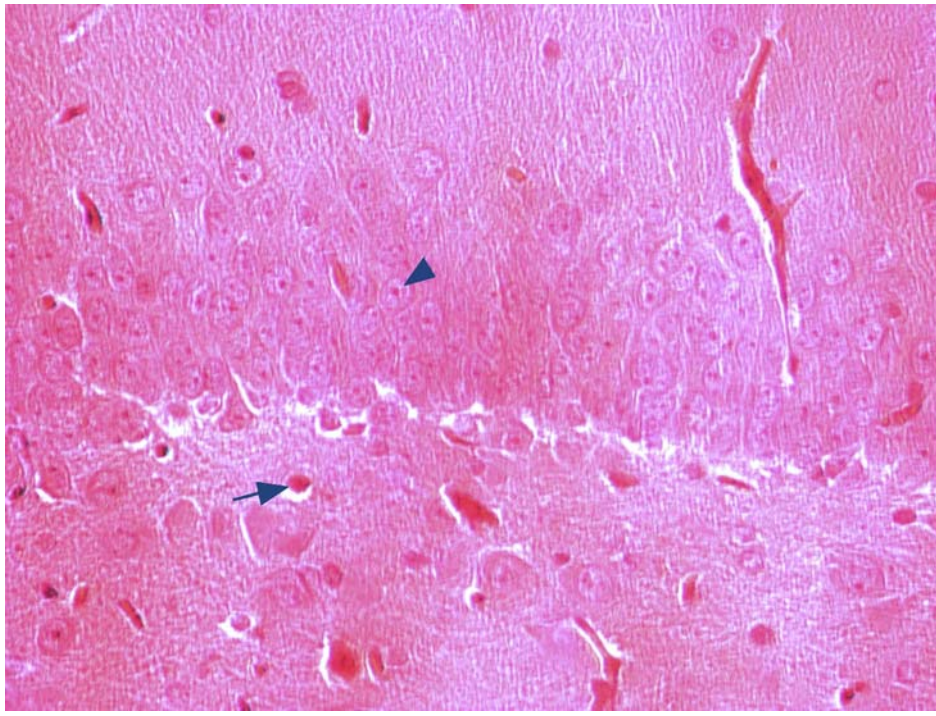

FIGURE 6 Pyramidal neurons (▽) vascular dilatations (⇒) of F2 group at hippocampus region of brain. H&E, 40X.

However, MF also interacts directly with electrons in DNA to affect protein biosynthesis (Goodman and Blank, 2002).

As a result of our data, a significant decrease was observed in average body weight increase in all experimental groups compared to control groups. In a previous study, MF exposure significantly suppressed food and water consumption and weight gain in mice (Tsuji et al., 1996). The metabolic changes due to the MF exposure were demonstrated by the marked decrease in the level of glucose and total protein in serum of the exposed mice. Therefore, it is possible to consider that the decreased body weight might be due to the decrease of body fluid and protein content or other factors including hormonal changes (Hashish et al., 2007). Body weight decrease could be related to the hypoxia status known to alter the body weight (Neckar et al., 2003). MF induced an increase in hematocrit level, haemoglobin concentration, LDH levels, and sempatic hyperactivity, suggesting an hypoxia-like state (Chater et al., 2006, Abdelmelek et al., 2005). Previous studies showed that chronic hypoxia was associated with increased oxidative stress as evidenced by marked lipid peroxidation and the induction of antioxidant enzyme response in various tissues and organs (Nakanishi et al., 1995).

The present study shows that the average numbers of pyramidal cells at cortex were reduced significantly compared to control groups. Also, average numbers of acidophilic ischemic neurons of MF group were reduced 73% compared to control groups. MF caused damage of pyramidal cell of brain cortex. Acidophilia is one of the hallmarks of acute neuronal damage and death due to brain ischemia, excitotoxic and traumatic lesion,s and epileptic seizures (Victorov et al., 2000). At a previous study, authors found significant evidence for neuronal damage in hippocampus cortex and basal ganglia to MF (Salford et al., 2003). It can be explained that the occurrence of abnormal neurons could be due to the opening of the blood-brain barrier (Salford et al., 1994). Oscar and Hawkins (1977) demonstrated that MF exposure caused a significant leakage of mannitol, and also dextran from capillaries into the surrounding cerebellar tissue. Another reason of ischemic damage may be caused by the effect of MF on melatonin hormone. One of the important hormones is melatonin, preventing ischemic damage in the brain (Lee et al., 2007). But previous studies show that MF reduces melatonin secretion (Jarupat et al., 2003). Jelenković et al. (2006) demonstrated that a 7 days' exposure to MF can be harmful to the brain, especially to the basal forebrain and frontal cortex due to developmental of lipid peroxidation. At the MF group, the average number of pyramidal cells were reduced by 55.43% compared to control group. At F2 group, a significant increase in the number of vascular dilatation was observed compared to control groups. The vascular dilatation is a significant evidence of tissue damage.

The present study shows that MF does not cause significant effect in pyramidal cells at brain hippocampus region. Also, Ragbetli et al. (2009) found no change in hippocampal pyramidal cell number of the mouse exposed to a mobile phone. However, Salford et al. (2003) demonstrated that, by susceptibility to RF exposure, neuronal damages in the hippocampus, cortex, and basal ganglia were reported. Hippocampus, especially granule cells of the dentate gyrus (Odaci et al., 2008) and the CA areas (Bas et al., 2009a, 2009b), is selectively vulnerable to RF exposure. Loss of pyramidal cells in the CA areas has been reported after prenatal period and 4 weeks of exposure at 900 MHz in adult rats (Bas et al., 2009a,b). RF exposure has also resulted in the decrease of granule cell number in the rat dentate gyrus (Odaci et al., 2008). Similar studies of radiation effect have reported vulnerability of the granule cells in dentate gyrus (Nagai et al., 2000; Jenrow et al., 2004). These previous reports may indicate the deleterious effect of RF exposure to the hippocampal formation. Furthermore, at the previous study, the chronic exposure to GSM significantly

decreased excitatory synaptic activity and the number of excitatory synapses in cultured hippocampal neurons (Xu et al., 2006). Barcal and Vozeh (2007) showed that a change of cortical and hippocampal activity was observed during MF exposure. A previous study reported that extremely low-frequency MF penetrates deep tissues but high-frequency MF affects only superficial tissues, because if the frequency increases, the penetration decreases (Ozguner and Mollaoglu, 2006). As a result of this study, MF affected pyramidal neurons of cortex, but it did not affect pyramidal cells of hippocampus region.

In conclusion, MF affected blood glucose, total protein levels, and especially structure of the cortex region of the brain.

Acknowledgments

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

REFERENCES

Abdelmelek, H., Molnar, S., Servais, S., et al. (2005). Skeletal muscle HSP72 and norepinefrine response to static magnetic field in rat. *J. Neur. Trasmiss.* 1–7.

Adelman, R., Saul, R. L., Ames, B. N. (1998). Oxidative damage to DNA: relation to species metabolic rate and life span. *Proc. Natl. Acad. Sci. USA* 85:2706–2708.

Adey, W. R. (1981). Tissue interactions with non-ionising electromagnetic fields. *Physiol. Rev.* 61:435–514.

Ahlbom, A. (2001). Neurodegenerative diseases, suicide and depressive symptoms in relation to EMF. *Bioelectromagn. Suppl.* 5:132–143.

Aitken, R. J., Bennetts, L. E., Sawyer, D. (2005). Impact of radiofrequency electromagnetic radiation on DNA integrity in the male germline. *Int. J. Androl.* 28:171.

Amara, S., Abdelmelek, H., Ben Salem, M., et al. (2006). Effect of static magnetic field exposure on hematological and biochemical parameters in rats. *Braz. Arch. Biol. Techn.* 49:889–895.

Ammari, M., Brillaud, E., Gamez, C. (2008). Effect of a chronic GSM 900 MHz exposure on glia in the rat brain. *Biomed. Pharmacother.* 62:273–281.

Babych, V. I. (1995). The characteristics of tissue lipid peroxidation in the internal organs and the lipid metabolic indices of the blood plasma in a low geomagnetic field. *Fiziol. Zh.* 41(5–6):44–49.

Barcal, J., Vozeh, F. (2007). Effect of whole-body exposure to high-frequency electromagnetic field on the brain cortical neuron and hippocampal activity in mouse experimental model. *NeuroQuantology* 3: 292–303.

Bas, O., Odaci, E., Mollaoglu, H. (2009a). Chronic prenatal exposure to the 900 megahertz electromagnetic field induces pyramidal cell loss in the hippocampus of newborn rats. *Toxicol. Ind. Health* 25: 377–384.

Bas, O., Odaci, E., Kaplan, S., Acer, N. (2009b). 900 MHz electromagnetic field exposure affects qualitative and quantitative features of hippocampal pyramidal cells in adult rat. *Brain Res.* 1265:178–185.

Benzi, G., Moretti, A. (1995). Are reactive oxygen species involved in Alzheimer's disease? *Neurobiol. Aging* 16:661–674.

Bethwaite, P., Cook, A., Kenedy, J., Pearce, N. (2001). Acute leukemia in electrical workers: A New Zeland case-control study. *Cancer Cause Contr.* 12:683–689.

Boguslaw, K., Andrezej, S., Rozalia, G., Danuta, P. (1999). Effect of electromagnetic field on serum biochemical parameters in steelworkers. *J. Occup. Health* 41:177–180.

Braune, S., Wrocklage, C., Raczek, J. (1998). Resting blood pressure increase during exposure to a radiofrequency electromagnetic field. *Lancet* 351:1857–1858.

Brillaud, E., Piotrowski, A., de Seze, R. (2007). Effect of an acute 900MHz GSM exposure on glia in the rat brain: a time-dependent study. *Toxicology* 16:23–33.

Chater, S., Abdelmelek, H., Pequignot, J. M. (2006). Effects of sub-acute exposure to static magnetic field on haematological and biochemical parameters in pregnant rats. *Electromagn. Biol. Med.* 25(3):135–144.

Chernoff, N., Rogersm, J. M., Kavet, R. (1992). A review of the literature on potential reproductive and developmental toxicity of electric and magnetic field. *Toxicology* 74:91–126.

Chernysheva, O. N. (1990). Status of the lipid phase of plasma membranes of the rat hearth after repeated exposure to an alternate magnetic field of 50 Hz frequency. *Kosmicheskaia Biologiia Aviakosmicheskaia Meditsina* 24(1):30–31.

Elbetieha, A., Al-Akhras, M., Darmani, H. (2002). Long-term exposure of male and female mice to 50 Hz magnetic field: Effects on fertility. *Bioelectromagnetics* 23(2):168–172.

Eroschenko, P. V. (2001). *di Fiore's Atlas of Histology-with Functional Correlations*, 10th ed Copyright © Lippincott Williams & Wilkins 351 West Camden Street, Baltimore, MD.

Fahn, S., Cohen, G. (1992). The oxidant stress hypothesis in Parkinson's disease: evidence supporting it. *Ann. Neurol.* 32:804–812.

Frey, A. H. (1998). Headaches from cellular telephones: are they real and what are the implications? *Environ. Health Perspect.* 106:101–103.

Freude, G., Ullsperger, P., Eggert, S., Ruppe, I. (2000). Microwaves emitted by cellular telephones affect human slowbrain potentials. *Eur. J. Appl. Physiol.* 81:18–27.

Goodman, R., Blank, M. (2002). Insights into electromagnetic interaction mechanisms. *J. Cell. Physiol.* 192(1):16–22.

Gorczynska, E., Wegrzynowicz, R. (1991). Glucose homeostasis in rats exposed to magnetic fields. *Invest. Radiol.* 26(12):1095–1100.

Hakansson, N., Gustavsson, P., Sastre, A., Floderus, B. (2003). Occupationally exposure to extremely low frequency magnetic fields and mortality from cardiovascular disease. *Amer. J. Epidemiol.* 158:534–542.

Hashish, A. H., El-Missiry, M. A., Abdelkader, H. I., Abou-Saleh, R. H. (2007). Assessment of biological changes of continuous whole body exposure to static magnetic field and extremely low frequency electromagnetic fields in mice. *Ecotox and Environ Safe* 71(3):895–902.

Hossmann, K. A., Hermann, D. M. (2003). Effects of electromagnetic radiation of mobile phones on the central nervous system. *Bioelectromagnetics* 24:49–62.

Hyland, G. J. (2000). Physics and biology of mobile telephony. *Lancet* 356:1833–1836.

Ilhan, A., Gurel, A., Armutcu, F. (2004). Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain. *Clin. Chim. Acta* 340:153–162.

Jarupat, S., Kawabata, A., Tokura, H., Borkiewicz, A. (2003). Effects of the 1900 MHz electromagnetic field emitted from cellular phone on noctural melatonin secretion. *J. Physiol. Anthropol.* 22:61–63.

Jelenković, A., Janać, B., Pešić, V., et al. (2006). Effects of extremely low –frequency magnetic field in the brain of rats. *Brain Res. Bull.* 68:355–360.

Jenrow, K. A., Ratkewicz, A. E., Lemke, N. W. (2004). Effects of kindling and irradiation on neuronal density in the rat dentate gyrus. *Neurosci. Lett.* 371:45–50.

Kabuto, H., Yokoi, I., Mori, A., Ogawa, N. (2000). Effects of an in vivo 60Hz magnetic fields on monoamine levels in mouse brain. *Pathophysiology* 7:115–119.

Keaney, Jr., J. F., Vita, J. A. (1995). Atherosclerosis, oxidative stress, and antioxidant protection in endothelium-derived relaxing factor action. *Prog. Cardiovasc. Dis.* 38:129–154.

Kim, S. H., Lee, H. J., Choi, S. Y. (2006). Toxicity bioassay in spraque-dawley rats exposed to 20 KHz triangular magnetic field for 90 days. *Bioelectromagnetics* 27(2):105–111.

Koyu, A., Cesur, G., Özgüner, F., Elmas, O. (2005). Cep telefonlarından yayılan 900 MHz elektromanyetik alanın serum kortizol ve testesteron hormonu üzerine etkisi. *SDÜ Tıp Fakültesi Dergisi* 12(1): 52–56.

Kumosani, T. A., Qari, M. H. (2003). The effect of magnetic field on the biochemical parameters of mice blood. *Pak. J. Med. Sci.* 19(1):36–40.

Lee, M. Y., Kuan, Y. H., Chen, H. Y. (2007). Intravenous administration of melatonin reduces the intracerebral cellular inflammatory response following transient focal cerebral ischemia in rats. *J. Pineal. Res.* 42(3):297–309.

Loew, L. M. (1992). Voltage-sensitive dyes. Measurement of membrane potentials induced by DC and AC electric fields. *Bioelectromagnetics* 1:179–189.

Manesse, M., Delverdier, M., Abella-Bourges, N. (1998). An immunohistochemical study of bovine palatine and pharyngeal tonsils at 21, 60 and 300 days of age. *Anatomia, Histologia Embriologia* 27(3):179–185.

Mausset, A. L., de Seze, R., Montpeyroux, F., Privat, A. (2001). Effects of radiofrequency exposure on the GABAergic system in the rat cerebellum: clues from semiquantitative immunohistochemistry. *Brain Res.* 912:33–46.

Nagai, R., Tsunoda, S., Hori, Y., Asada, H. (2000). Selective vulnerability to radiation in the hippocampal dentate granule cells. *Surg. Neurol.* 53:503–506.

Nakanishi, K., Tajima, F., Nakamura, A., Yagura, S. (1995). Effect of hypobaric hypoxia on antioxidant enzymes in rats. *J. Physiol-London* 489:863–876.

Neckar, J., Szarszoi, O., Herget, J. (2003). Cardioprotective effect of chronic hypoxia is blunted by concomitant hypercapnia. *Acad. Sci. Czech Repub.* 52:171–175.

Neumann, H. (2001). Control of glial immune function by neurons. *Glia* 36:191–199.

Odaci, E., Bas, O., Kaplan, S. (2008). Effects of prenatal exposure to a 900 megahertz electromagnetic field on the dentate gyrus of rats: a stereological and histopathological study. *Brain Res.* 1238:224–229.

Oliver, C. N., Ahn, B. W., Moerman, E. J., et al. (1987). Age related changes in oxidized proteins. *J. Biol. Chem.* 262:5488–5491.

Oscar, K., Hawkins, T. (1977). Microwave alteration of the blood brain barrier system of rats. *Brain Res.* 126: 281–293.

Ozguner, F., Mollaoglu, H. (2006). Manyetik alanın organizma üzerindeki biyolojik etkileri. *SDÜ Tıp Fakültesi Dergisi* 13(1):38–41.

Paxinos, G., Watson, C. (1994). *The Rat Brain in Stereotaxic Coordinates*, 3th ed, Academic Press Inc., 525 B Street, Suite 1900, San Diego, California, USA.

Ragbetli, M. C., Aydinlioglu, A., Koyun, N., Ragbetli, C. (2009). Effect of prenatal exposure to mobile phone on pyramidal cell numbers in the mouse hippocampus: a stereological study. *Int. J. Neurosci.* 119: 1031–1041.

Salford, L. G., Brun, A. E., Eberhardt, J. L. (2003). Nerve cell damage in mammalian brain after exposure to microwave from GSM mobile phones. *Environ. Health Perspect.* 111:881–883.

Salford, L. G., Brun, A., Sturesson, K., et al. (1994). Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50 and 200 Hz. *Microsc. Res. Techniq.* 27:535–542.

Schwartz, J. L., House, D. E., Mealing, G. A. (1990). Exposure of frog hearts to CW or amplitude-modulated VHF fields: selective efflux of calcium ions at 16 Hz. *Bioelectromagnetics* 11:349–358.

Skinner, J., Mee, T. J. (2002). Exposure to power frequency electric fields and the risk of childhood cancer in the UK. *Brit. J. Cancer* 87:1257–1266.

Smith, K. J., Kapoor, R., Felts, P. A. (1999). Demyelination: the role of reactive oxygen and nitrogen species. *Brain Pathol.* 9:69–92.

Stam, R. (2010). Electromagnetic fields and the blood-brain barrier. *Brain Res. Rev.* 65:80–97.

Thomas, D., Tovey, S. C., Collins, T. J. (2000). A comparison of fluorescent Ca2 + indicator properties and their use in measuring elementary and global signals. *Cell. Calcium* 28:213–223.

Tsuji, Y., Nakagawa, M., Suzuki, Y. (1996). Five-tesla static magnetic fields suppress food and water consumption and weight gain in mice. *Ind. Health* 34(4):347–357.

Van Leeuwen, G. M., Lagendijk, J. J., Van Leersum, B. J., et al. (1999). Calculation of change in brain temperatures due to exposure to a mobile phone. *Phys. Med. Biol.* 44:2367–2379.

Victorov, I. V., Prass, K., Dirnagl, U. (2000). Improved selective, simple and contrast staining of acidophilic neurons with vanadium acid fuchsin. *Brain Res. Prot.* 5(2):135–139.

Wagner, P., Roschke, J., Mann, K. (1998). Human sleep under the influence of pulsed radiofrequency electromagnetic fields: a polysomnographic study using standardized conditions. *Bioelectromagnetics* 19:199–202.

Westendorp, M. O., Shatrov, V. A., Schulze-Osthoff, K. (1995). HIV-1 Tat potentiates TNF-induced NF-kappa B activation and cytotoxicity by altering the cellular redox state. *EMBO J.* 14:546–554.

Wolf, F. I., Torsello, A. (2005). 50-Hz extremely low frequency electromagnetic fields enhance cell proliferation and DNA damage: possible involvement of a redox mechanism. *BBA* 1743:120–129.

Xu, S., Ning, W., Xu, Z. (2006). Chronic exposure to GSM 1800-MHz microwaves reduces excitatory synaptic activity in cultured hippocampal neurons. *Neurosci. Lett.* 398:253–257.