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ORIGINAL ARTICLE

The effects of prenatal exposure to a 900-MHz electromagnetic field on the 21-day-old male rat heart

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Abstract

The growing spread of mobile phone use is raising concerns about the effect on human health of the electromagnetic field (EMF) these devices emit. The purpose of this study was to investigate the effects on rat pup heart tissue of prenatal exposure to a 900 megahertz (MHz) EMF. For this purpose, pregnant rats were divided into experimental and control groups. Experimental group rats were exposed to a 900 MHz EMF (1 h/d) on days 13–21 of pregnancy. Measurements were performed with rats inside the exposure box in order to determine the distribution of EMF intensity. Our measurements showed that pregnant experimental group rats were exposed to a mean electrical field intensity of 13.77 V/m inside the box (0.50 W/m^2). This study continued with male rat pups obtained from both groups. Pups were sacrificed on postnatal day 21, and the heart tissues were extracted. Malondialdehyde, superoxide dismutase and catalase values were significantly higher in the experimental group rats, while glutathione values were lower. Light microscopy revealed irregularities in heart muscle fibers and apoptotic changes in the experimental group. Electron microscopy revealed crista loss and swelling in the mitochondria, degeneration in myofibrils and structural impairments in Z bands. Our study results suggest that exposure to EMF in the prenatal period causes oxidative stress and histopathological changes in male rat pup heart tissue.

Keywords

Apoptosis, electromagnetic field, electron microscopy, heart, male rat, oxidative stress

History

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Introduction

The increasingly widespread use of the mobile phone, the most important communication device in our time, is giving rise to serious concerns regarding the effect on human health of the electromagnetic field (EMF) emitted by these (Fychting et al., 2005; Genuis, 2008; Hardell and Sage, 2008; Lu et al., 2012; Repacholi, 2001). Many studies on this subject have concentrated on the various causes and mechanisms of these effects. Some researchers maintain that EMF causes cell damage by leading to an increase in free radicals (Lu et al., 2012). There are strong grounds for this claim, because EMF can affect chemical bonds between compound atoms as a result of the production of reactive oxygen radicals (ROS). ROS and oxygen-free radicals, known as reactive nitrogen species, cause oxidative damage to cellular structures and molecules such as lipids, proteins and nucleic acid. Cellular damage induced by oxidative stress can also trigger apoptosis. ROS arising from EMF can therefore cause the onset of apoptotic or necrotic cell death (Kiray et al., 2013).

The heart is an organ that generates its own rhythm and constantly contracts. Due to its characteristic stimulation properties, heart contraction or rhythm can be affected by external stimuli (Elmas et al., 2012). The duration of the antioxidant cycle in the heart, which consumes oxygen at a low level, is very short. The heart has less protection against ROS damage compared to other tissues (Goraca et al., 2009). ROS may cause myocyte atrophy, apoptosis and interstitial fibrosis in heart tissue. This may lead to the suppression of heart functions and the progression of heart disorders (Goraca et al., 2009).

The stage of development most affected by environmental factors is the prenatal period (Odacı et al., 2014; Poulletier de Gannes et al., 2012; Topal et al., 2014). EMF is one of the most important of these environmental factors. The effects of EMF on the embryo and fetus in the prenatal period are the subject of major debate. Some recent studies have emphasized that EMF can affect the development of the fetus or embryo and cause severe malformations in vital organs (Saito et al., 2006; Tenorio et al., 2011). One study reported a decrease in the number of pyramidal cells in the cornu ammonis of rats

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exposed to the effect of 900 megahertz (MHz) in the prenatal period (Sonmez et al., 2010). Another study reported malformations such as polydactylism, brain hernia and vestigial rib, brain hernia and curled tail in mice fetuses exposed to the effect of magnetic field (Saito et al., 2006). Our scan of the literature revealed no previous studies investigating the effect on heart tissue in the postnatal period in rats exposed to 900 MHz EMF in the prenatal period. The purpose of this study was therefore to investigate the effects on the male rat pup heart of exposure to 900 MHz EMF on days 13–21 of the prenatal period as the most critical stage of the developmental process of the rat heart.

Materials and methods

Animals and experimental procedures

The experiment was run under blind conditions. Eighteen rats were used in this study, six healthy, pregnant Sprague Dawley rats (6–8 weeks old, weighing 180–250 g) and 12 newborn male rat pups obtained from these. Rats were obtained from the Karadeniz Technical University Surgical Research Center. The study was approved by the Institutional Animal Ethical Committee of Karadeniz Technical University, Trabzon, Turkey. All animals were kept at room temperature ($22 \pm 2\,^{\circ}C$) and humidity ($50\% \pm 10$) in a controlled (12/12 h) light/dark cycle. Standard laboratory chow and tap water were provided. At the start of the study, six female rats with two normal cycles were left to mate with six male rats in the same cage. Pregnancy was monitored the following day using vaginal smear. Six pregnant female rats were subsequently divided at random into two groups of three rats each. Mother rats in the first group represented the control group (CG), and no procedure was performed on rats in this group throughout pregnancy. Mother rats in the second group represented the experimental group (EMF group = EMFG). Rats in this group were placed in a Plexiglas box at the same time every day (10.00–11.00) on days 13–21 of pregnancy and were exposed to the effect of 900 MHz EMF for 1 h. Before giving birth, each mother rat was placed in a separate cage. After birth, pups were left to feed naturally with their mothers in the same cages. No procedure was performed on newborn rats after birth. Newborn male rats born to CG mothers were classified as the newborn control group (NCG), and male rats born to experimental group rats were classified as the newborn EMF group (NEMFG). At the end of the experimental period (postnatal 21st d), six rats chosen at random from each group were sacrificed under deep anesthesia (Ketalar 50 mg/kg) by decapitation. The rib cage was then opened. The heart was accessed and freed from surrounding structures and extracted with an apical incision. Heart tissue specimens from the left ventricle were set aside for biochemical examination and light and electron microscopy.

EMF exposure system

We used an EMF exposure system in order to subject experimental group rats to the effect of 900 MHz EMF (Baş et al., 2013; Hancı et al., 2013; İkinci et al., 2013; Odacı et al., 2013). Our EMF exposure system consisted of an ultra-high-frequency oscillator (1218-BV, Lockable Oscillator, 900–2000 MHz, General Radio Company, Concord, MA, Serial no. 1483) fed with an uninterrupted power source (1267-B Regulated Power Supply, General Radio Company, serial no. 903) with output power of approximately 300 mW and a frequency adjusted to 900-MHz. This was used to establish a 900-MHz EMF. The oscillator was attached to a half-wave dipole antenna made from a copper rod (1 mm × 15 cm) with the help of a coaxial cable. The antenna was installed into the central region, approximately 11 cm inside the open surface of a box made of Plexiglas produced specially for the study (42 cm × 30 cm × 50 cm in size; Figure 1).

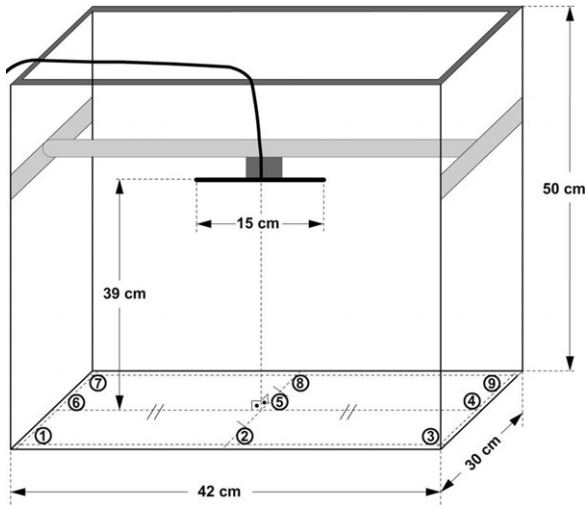

Figure 1. The dimensions of the Plexiglas box used to expose pregnant rats to a 900 MHz EMF. The positions of the nine points on the floor of the box at which EMF intensity were measured are shown, together with the location in the box of the 15-cm copper semi-wave dipole antenna.

Exposure to 900-MHz EMF

Pregnant rats were placed inside the box and exposed to the effect of 900 MHz EMF for 1 h. The intensity of EMF with rats inside the box was measured at nine points on the floor of the box (Figure 1) using a wide-band EMF measuring device (100 kHz–2.5 GHz range, Chauvin-Arnoux C.A 43 Fieldmeter, Chauvin-Arnoux Group, Paris, France). Our measurements showed that pregnant experimental group rats were exposed to a mean electrical field intensity of 13.77 V/m inside the box (0.50 W/m^2). The whole-body specific absorption rate (SAR) was calculated at 0.025 W/kg (RadHaz SAR Equivalency Calculator, Version 1.0, ©2000 Richard Tell Associates, Inc., Colville, WA). Electrical field intensity was measured using a wide-range measuring device with a measurement range of 100 kHz–2.5 GHz (Chauvin Arnoux CA43 Isotropic Electrical Field Intensity Meter).

Histological procedures

The dissected hearts were fixed in 10% formalin, processed using routine histological methods and embedded in paraffin blocks. For histopathological evaluation, the paraffin blocks were sliced into 5-μm sections using a fully automatic microtome (Leica RM 2255, Leica Instruments, Nussloch,

Germany) and stained with hematoxylin and eosin (H&E) for histopathological observations. Sections were also stained with Prussian blue to show iron pigments in the sections. Iron pigments were scored between 0 and 3 (0 = no iron accumulation, 1 = mild iron accumulation, 2 = medium iron accumulation and 3 = heavy iron accumulation; Guo et al., 2006). Heart sections were examined by a histologist under light microscopy (Olympus BX-51, Tokyo, Japan).

Terminal deoxynucleotidyl transferase deoxyuridine triphosphate nick end labeling assay assay

Apoptosis of heart tissue was analyzed using the terminal deoxynucleotidyl transferase deoxyuridine triphosphate nick end labeling assay (TUNEL) method. Sections 5 μm in thickness were taken from the paraffin blocks and subjected to standard deparaffinization. TUNEL staining of sections was performed using an *in situ* cell Death Detection Kit (Roche, Mannheim, Germany), in accordance with the manufacturer's instructions. Apoptosis evaluation from the TUNEL-stained slides was performed using a light microscope (Olympus, BX51) at a magnification of × 400 (Hanc1 et al., 2013). Five fields were randomly chosen for each slide, and a total of 100 cells per field were counted (Dogan et al., 2010). The percentage of apoptotic nuclei [apoptotic index (AI)] was calculated as apoptotic nuclei/total nuclei counted × 100%. All counting procedures were performed blind.

Biochemical estimations

Superoxide dismutase (SOD) and catalase (CAT) enzyme activities and the amounts of glutathione (GSH) and lipid peroxidation (LPO) in the tissues were determined. The level of LPO in tissues was determined by estimating malondialdehyde (MDA) using the thiobarbituric acid test (Ohkawa et al., 1979). The standard curve was obtained using 1,1,3,3-tetramethoxypropane. The results were expressed as nmol/g tissue. SOD activity was measured according to the method described by Sun et al. (1988). SOD activity was then measured at 560 nm by the degree of inhibition of this reaction and was expressed as mmol/min/mg of tissue (Sun et al., 1988). Decomposition of H_2O_2 in the presence of CAT was observed at 240 nm. CAT activity was defined as the amount of enzyme required to decompose 1 mmol of H_2O_2 per minute at 25 °C and pH 7.8. Results were expressed as mmol/min/mg of tissue (Aebi, 1984). The amount of GSH in the tissues was calculated by modifying the method described by Sedlak and Lindsay (1968). GSH level was expressed as nmol/g tissue.

Transmission electron microscopic procedures

For ultrastructural investigations, 1-mm myocardial heart tissue specimens were fixed in 100 mM phosphate buffer containing 2.5% glutaraldehyde for 2 h at 4 °C. These were washed in phosphate buffer and stored at 4 °C for later processing. Heart tissues were post-fixed with 1% osmium tetroxide dehydrated in an ethanol series, and then embedded in epoxy resin (Araldite CY212, Agar Scientific, Essex, UK). For light microscopy analysis, 750-nanometer thick sagittal sections were cut and stained with toluidine blue. Sections were then visualized and imaged using a Leica DM 6400 (Leica Microsystems, Wetzlar, Germany). For electron microscopy analysis, ultrathin sections 60–70-nm thick were cut using an ultramicrotome (LKB Nova, Bromma, Sweden) and set on 200-mesh copper or nickel grids. Ultra-thin sections at the top of the nickel grid were stained with uranyl acetate and Reynold's lead citrate. Images on TEM (JEOL 100SX, JEOL Ltd., Akishima, Tokyo, Japan) were obtained by means of photographic image capture (Kodak 4489, Eastman Kodak Company, Rochester, NY).

Statistical analysis

Kruskal–Wallis analysis of variance was used to compare differences between group parameters. A difference at $p < 0.01$ was considered statistically significant. Statistical analyses were performed using SPSS 13.1 (Statistical Package for the Social Sciences, version 13.1, SSPS Inc., Chicago, IL). Data were presented as mean ± standard deviation.

Results

Light microscopic observations

Histopathological evaluation of the H&E heart sections was performed under light microscopy at a magnification of × 400. No pathological finding was observed in the NCG rats' myocardial tissue (Figure 2A). Irregularity, degeneration, vacuolization and nucleus loss were observed in heart muscle fibers from NEMFG rats. Pyknotic nuclei and apoptotic alterations were also observed (Figure 2B). At light microscopic examination of sections stained with Prussian blue, no significant difference was determined between NCG (0.6 ± 0.54) and NEMFG (1 ± 0.7) in terms of hemosiderin granules ($p > 0.01$).

Nuclear changes suggestive of apoptosis were observed in TUNEL staining sections under light microscopy at a magnification of × 400. Apoptotic cells were heterogeneously distributed within the different regions of the heart tissue cell for each subject group. In NCG, there were a few apoptotic cells (Figure 2C), while there were a large number of apoptotic cells in NEMFG (Figure 2D). The numbers of TUNEL-positive cells in NEMFG were markedly increased, and statistically, the percentage of apoptotic cells, AI, in heart tissue cell in NEMFG was significantly higher than that in NCG ($p < 0.01$; Table 1).

Electron microscopic observations

Under TEM, normal myofilament structure was observed in samples from the NCG hearts. The structure was clear, and the sarcolemma was intact. Nucleus was normal (Figure 3A). The mitochondria from NCG exhibited well-defined double membranes with normal cristae arrangement. Z bands were regular in NCG samples, and the sarcomere boundary was clearly visible (Figure 4A). In addition, disorganized myofilament and dissolved focal myofilament were observed from in NEMFG specimens. Degeneration and fragmentation of myofibrils were present in NEMFG heart muscle (Figure 3B–D), together with perivascular and cytoplasmic vacancies, and disrupted structural integrity of the Z bands.

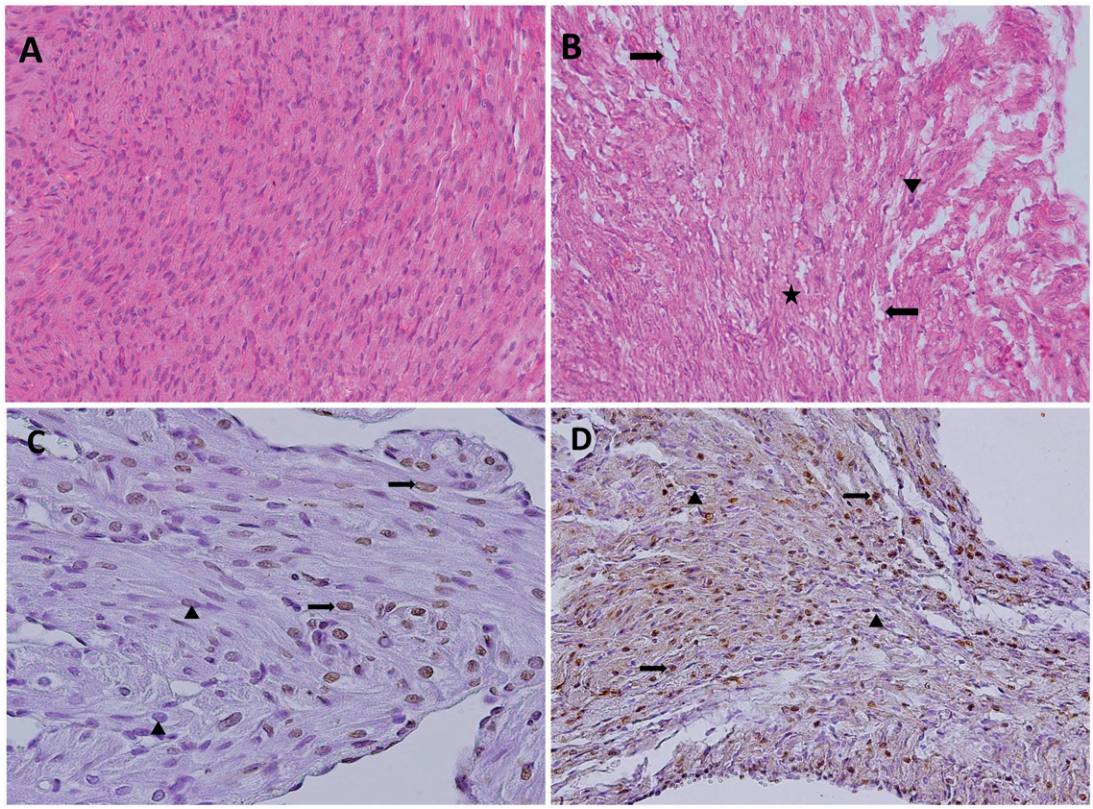

Figure 2. Light microscopic images from myocardial tissue sections from NCG (A and C) and NEMFG (B and D) rats. (A) No pathological finding was observed in the H&E sections of NCG rats. (B) In the H&E section of NEMG rats, irregularity and degeneration (right arrow), vacuolization (left arrow), nucleus loss (asterisk) and pyknotic nuclei (arrow head) were present in heart muscle fibers (400 ×). (C) In TUNEL staining sections, there were a few apoptotic cells in NCG. (D) A large number of apoptotic cells were observed in NEMFG TUNEL staining sections (arrows represent apoptotic cells and arrow heads represent normal cells in C and D pictures) (400×) (NCG = newborn control group, NEMFG = newborn electromagnetic field group).

Table 1. Biochemical analysis and AI results for heart tissues from NCG and NEMFG rats.

Biochemical parameters	NCG ($n=6$)	NEMFG ($n=6$)
MDA (nmol/mg tissue)	13.57 ± 0.28	20.30 ± 0.12^{a}
SOD (mmol/min/mg tissue)	2.42 ± 0.02	2.60 ± 0.04^{a}
CAT (mmol/min/mg tissue)	0.01 ± 0.002	0.02 ± 0.002^{a}
GSH (nmol/mg tissue)	0.42 ± 0.02	0.27 ± 0.01^{b}
AI (%)	18.33 ± 3.38	47.33 ± 6.34^{a}

The data represent mean ± SD.
[a]NEMF group values increased significantly compared with NCG ($p<0.01$).
[b]NEMF group values decreased significantly compared with NCG ($p<0.01$).
NCG, newborn control group; NEMFG, newborn experimental group rats; MDA, malondialdehyde; SOD, superoxide dismutase; CAT, catalase; GSH, glutathione; and AI, apoptotic index.

Electron-dense droplet location was also observed. In addition, mitochondrial swellings with vacuolation and decreased density of inner membrane cristae were seen in NEMFG (Figure 4B–D).

Biochemical analysis results

Based on the biochemical analysis results, tissue MDA, SOD and CAT values in NEMFG increased significantly compared to those of NCG ($p<0.01$), while GSH values decreased ($p<0.01$; Table 1).

Discussion

The effect of EMF emitted by mobile phones on the developing embryo/fetus during pregnancy is attracting considerable interest from researchers (Odacı et al., 2008, 2013, 2014; Topal et al., 2014). Studies on the subject have reported that exposure to the effect of EMF in the prenatal or postnatal period causes childhood leukemia (Wertheimer and Leeper, 1979), brain tumors in adults (Feychting et al., 1997), DNA damage (Lai and Singh, 2004), neurodegenerative diseases (Köktürk et al., 2013), immune system disorders (Kimata, 2004) and various cardiovascular diseases (Jeong et al., 2005; Hardell and Sage, 2008).

Heart development in rats begins with the formation of the cardiogenic area on the ninth day of the prenatal term and continues, albeit at a low level, after birth (Marcela et al., 2012). The most critical stage of the developmental process is the embryonic period. Numerous environmental factors can affect the embryo in terms of both vigor and the shaping of the anatomy in this period (Poulletier de Gannes et al., 2012). This study was intended to investigate changes that may occur in the heart tissue of rats exposed to the effect of 900 MHz EMF on days 13–21 of the prenatal period. GSM-900 is a widely used cellular communication system in Asia and Europe and the most popular standard for mobile phones worldwide (Bas et al., 2009a,b; Bhat, 2013; Odacı et al., 2008). That is why we selected to expose pregnant rats to 900 MHz EMF in this study. In our study, pregnant

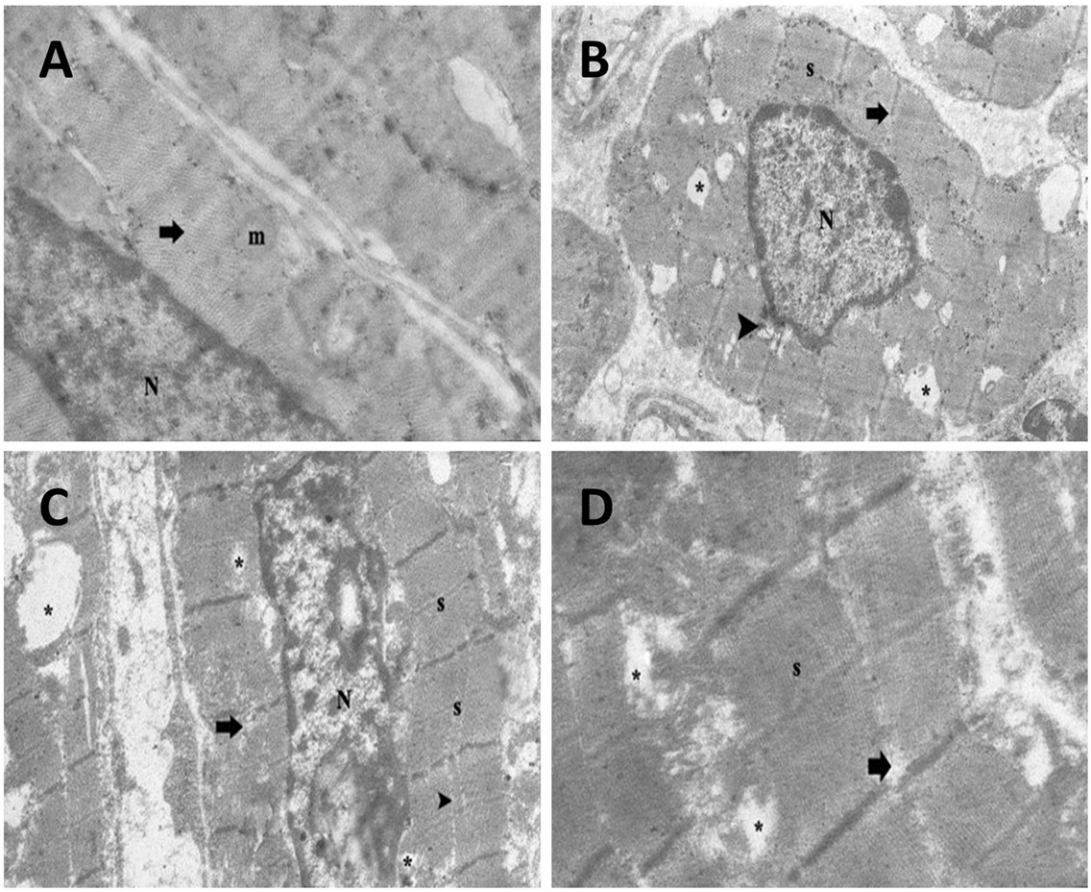

Figure 3. Transmission electron microscopic (TEM) photomicrographs of the ventricular myocardium in NCG (A) and NEMFG (B–D) rats. (A) Normal mitochondria (m) and normal myofilament structure in heart muscle (arrow) (TEM 15 000×). (B) Degeneration of Z band in heart muscle (arrow), electron-dense droplets (arrow head) and cytoplasmic vacancies (asterisk). Nucleus (N), sarcomeres (S) (TEM 6000×). (C) Disordered myofilament in heart muscle (arrow), cytoplasmic vacancies (asterisk) and nuclear damage (arrow head) (TEM 8000×). (D) Degeneration of myofilament in heart muscle (arrow), perivascular and cytoplasmic vacancies. Sarcomeres (S), cytoplasmic vacancies (asterisk) (TEM 15 000×).

experimental group rats were exposed to a mean electrical field intensity of 13.77 V/m inside the box (0.50 W/m^2). This value is regarded as equivalent to the intensity of the electrical field created in their immediate surroundings by mobile phones in speak mode (mean 1–10 V/m for variables such as model of mobile phone, its location, distance from the base station, etc.)

EMF originating from mobile phones leads to oxidative stress in biological systems due to an increase in free radicals and changes in antioxidant defense systems (Meral et al., 2007). The biological target of oxidative stress is generally lipids from the biomolecule class (Del Rio et al., 2005). Many studies have reported that EMF causes LPO (Koyu et al., 2009). MDA is an important product of compromise of chain reactions leading to the oxidation of unsaturated fatty acids and a biomarker for LPO. MDA is a marker of oxidative stress arising through LPO in heart muscle (Ozguner et al., 2005). A significant rise was observed in our study in MDA concentrations in heart tissue of rat pups exposed to the effect of a 900 MHz EMF in the prenatal period. This shows that prenatal exposure to the effect of 900 MHz EMF compromises the chain reaction that leads to the oxidation of fatty acids. One study reported that 900 MHz EMF caused an increase in MDA levels in heart, liver, testis and lung tissue (Esmekaya et al., 2011). Another study reported that exposure to 50 Hz EMF led to LPO in myocardial tissue in adult rats (Kiray et al., 2013). Studies show that the oxidative damage arising in association with EMF is LPO resulting from free oxygen radicals (Meral et al., 2007; Ozguner et al., 2005).

Under normal conditions, cells are protected against oxidative damage by several mechanisms and antioxidative molecules. SOD, CAT and GSH are some of the enzymes in oxidative damage mechanisms (Meral et al., 2007; Tkalec et al., 2013). SOD converts superoxide anion radicals into hydrogen peroxide (H_2O_2). H_2O_2 is broken down into oxygen and water by CAT in the peroxisome and by GSH peroxidase in the cytosol and mitochondria. The antioxidant defense mechanism that functions in this way under normal conditions may be compromised by EMF, leading to oxidative stress. The heart is rich in unsaturated fatty acids and consumes high levels of oxygen. In addition, the heart has a relatively lower level of antioxidant enzyme activity than other tissues (Goraca et al., 2009). The heart is therefore less protected against free radical damage than other tissues (Rozenberg et al., 2006).

In this study, SOD and CAT activities increased in the heart tissue of rat pups exposed to 900 MHz EMF on days 13–21 of the prenatal period compared to NCG rats. In contrast to our studies, Ozguner et al. (2005) reported a decrease in SOD, CAT and GSH-PX enzyme activities in myocardial tissue of rats they exposed to 900 MHz EMF for

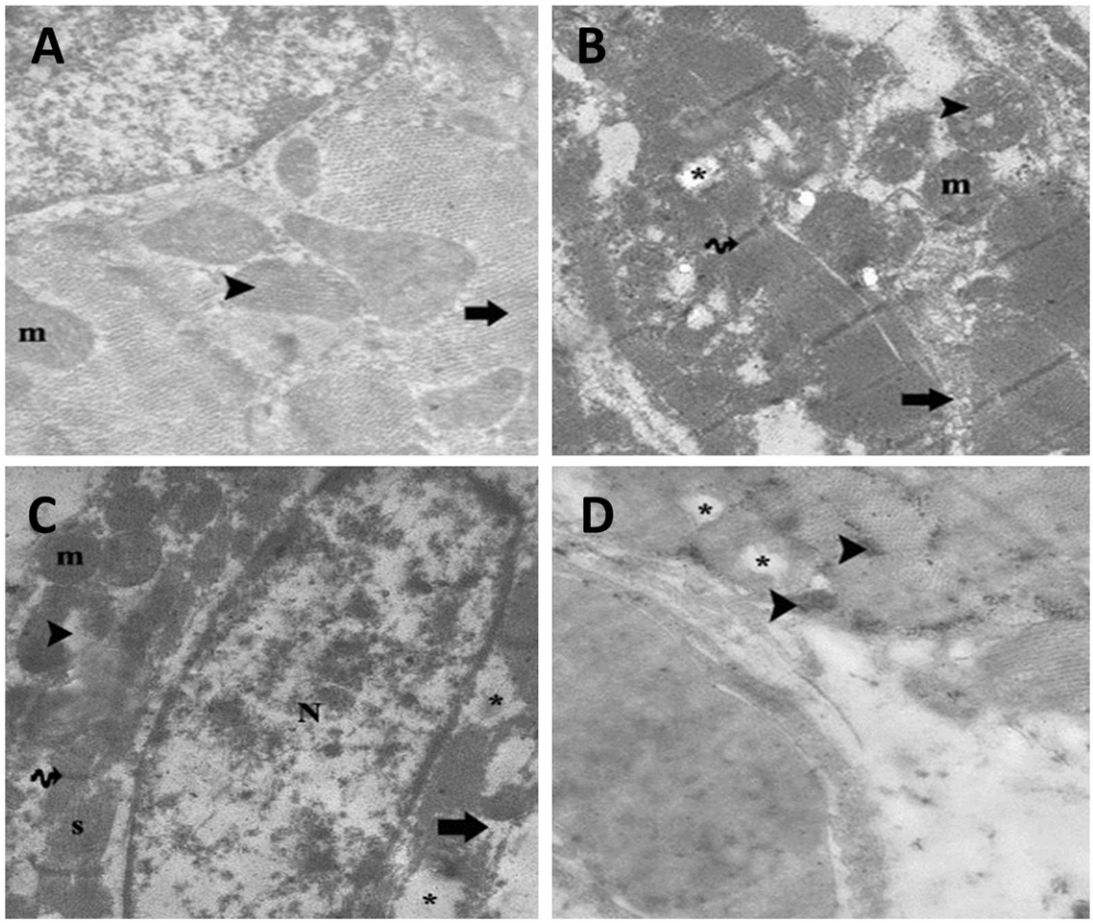

Figure 4. Transmission electron microscopic (TEM) photomicrographs of the ventricular myocardium of the control group (A) and experimental group (B–D) rats. (A) Normal mitochondria (m) and mitochondrial cristae (arrow head) and normal myofilament in heart muscle (arrow) can be seen under TEM (TEM 25 000×) (B and C). Degeneration and fragmentation of myofibrils in heart muscle (arrow), perivascular and cytoplasmic vacancies (asterisk), mitochondrial swelling along with vacuolation and disappearance of mitochondrial cristae (arrow head). Sarcomeres (S). Nuclei (N) (B, TEM 10 000×; C, TEM 25 000×). (D) Electron dense droplets (arrow head), cytoplasmic and perivascular vacancies (asterisk) (TEM 12 000×).

30 min a day over 10 d. Meral et al. (2007) observed that 900 MHz EMF increased CAT activity in brain tissue. Martínez-Sámano et al. (2010) also reported a decrease in SOD activity associated with exposure to EMF. In addition, Tayefi et al. (2010) reported that EMF they applied throughout pregnancy and until the 21st postnatal day (4 h/d; 3 mT) produced a decrease in SOD activity. The literature contains only one study examining the effects of EMF on newborn rat heart tissue following prenatal exposure to EMF (Tayefi et al., 2010). However, that study differed from ours in several respects. For example, Tayefi et al. (2010) made no gender distinction between their rat pups, and pups were also exposed to EMF in the postnatal period. In addition, their daily length of EMF exposure and the effect of the EMF applied were different to those in our study. In that respect, our study is the first of its kind in the literature. It also differs from that of Tayefi et al. (2010) in terms of antioxidant enzymes. When the two studies are assessed together, they show that the results may vary depending on the study design, the length of EMF administration and degree of exposure.

GSH functions as a scavenger and a co-factor in the metabolic detoxification of ROS against oxidative damage. Yurekli et al. (2006) stated that EMF leads to oxidative damage, and that there is an accompanying decrease in GSH activity. In our study, we observed a significant decrease in GSH in heart tissue of male rat pups exposed to 900 MHz EMF. This may be attributed to oxidized GSH in tissue damaged due to EMF being removed faster than new GSH is produced (Meral et al., 2007; Reilly et al., 1991).

Iron is of vital importance for processes such as respiration electron transfer and oxygen transport. However, when iron enters into reaction with H_2O_2, it produces hydroxy radicals. These have a very powerful oxidizing effect. Ferritin is the protein responsible for iron storage. Excessive iron uptake from the circulation causes a rise in ferritin in organ parenchyma. Cytotoxic reactions are not seen when iron is bound to ferritin. When iron detaches from ferritin, however, it damages the cell and the cell membrane (Yoon et al., 2010). Studies show that increased iron content in the cells and tissue can increase the risk of cancer. Lai and Singh (2004) hypothesized that acute exposure to magnetic field initiates an iron-mediated process that increases free radical formation in cells (such as brain cells), leading to DNA strand breaks and cell death. In our study, too, we determined no significant difference in terms of hemosiderin granule density in the heart tissue of rats exposed to the effect of 900 MHz EMF in the prenatal period.

Apoptosis, also known as programmed cell death, establishes homeostasis in tissues and plays an important role in normal development. Generally, apoptotic cell death is

defined by morphological criteria and characterized by nuclear condensation and DNA fragmentation with no major ultrastructural changes in cytoplasmic organelles. It is seen in specific cell types in normal development. Oxidative stress can activate apoptotic signal pathways. Apoptosis also plays a role in a series of pathophysiological events, such as abnormal myocardial function and function loss (Goraca et al., 2009; Stanely Mainzen Prince and Roy, 2013). Several studies have stated that EMF exposure triggers the apoptotic process (Chavdoula et al., 2010; Francisco et al., 2013; Hancı et al., 2013). Hancı et al. (2013) reported that 900 MHz EMF applied on days 13–21 of the prenatal period caused widespread apoptosis in spermatogenic cells in the rat testis. Köktürk et al. (2013) reported that 900 MHz EMF applied in the prenatal and postnatal periods caused apoptosis in cerebellar Purkinje and granular cells in 80-d-old rats. In our study, too, 900 MHz EMF applied in the prenatal period caused widespread apoptosis in the heart tissue of male rat pups. In agreement with this finding of ours, Kiray et al. (2013) reported that exposure to 50-Hz EMF caused apoptosis in myocardial tissue in adult rats. In addition, Tayefi et al. (2010) also reported that exposure to EMF triggered apoptosis. Considering our study finding together with the results of other research, we think that the increased apoptosis in heart tissue caused by EMF may be correlated with a significant rise in MDA levels. Our findings also suggest that degree of oxidative stress and severity of myocardial injury may be associated with increased ROS production and the compromise of the balance between antioxidant defense systems (Goraca et al., 2009).

Light microscopy in our study revealed irregularity, degeneration and vacuolization in heart muscle fibers in the experimental group. Electron microscopy revealed swelling and crista loss in mitochondria, degeneration in myofibrils and structural impairments in Z bands. Our histopathological findings are supported by other studies. Tayefi et al. (2010) and Kiray et al. (2013) reported similar findings in their studies. The first response of a cell exposed to stress is swelling (Dianzani et al., 1969). We therefore think that the histopathological changes seen in our experimental group are due to stress in heart tissue caused by EMF.

Whether or not exposure to the effect of EMF in the prenatal period affects organogenesis by altering materno-fetal metabolism is still the subject of debate. However, our findings show that exposure to the effect of EMF in the prenatal period causes apoptosis and morphological damage in rat heart tissue. In addition, our results show that exposure to EMF leads to oxidative stress as a result of an increase in LPO and impairment of antioxidant defense systems. In conclusion, our findings may be said to show that the effects of exposure to EMF in the prenatal period also cause damage in rat heart tissue in the postnatal period.

Declaration of interest

The authors report no conflicts of interest.

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