Volume 23, No 1 | International Journal of Radiation Research, January 2025

Cardiac oxidative stress induced by cell phone electromagnetic radiation and the cardioprotective effect of aerobic exercise in rats

H.A. Akbari, A.A. Gaeini*, M.R. Kordi, A.A. Ravasi

Department of Exercise Physiology, Faculty of Sports Sciences and Health, University of Tehran, Iran

► ***Original article***

***Corresponding author:**
Abbas Ali Gaeini, Ph.D.,
E-mail: *aagaeini@ut.ac.ir*

Received: November 2023
Final revised: May 2024
Accepted: June 2024

Int. J. Radiat. Res., January 2025; 23(1): 45-52

DOI: 10.61186/ijrr.23.1.45

Keywords: *Antioxidants, reactive oxygen species, electromagnetic radiation, heart, oxidative stress.*

ABSTRACT

Background: Oxidative stress-induced tissue damage is a recognized concern linked to electromagnetic radiation (EMR) waves. ***Materials and Methods:*** In the present study, we explored the effect of moderate aerobic exercise (AE) on oxidative stress (OS) and cardiomyocyte damage resulting from cell phone EMR in rats. A total of 32 male Wistar rats were randomly allocated into the following groups: control (C), EMR, AE, and EMR+AE. Both EMR+AE and EMR groups were subjected to three hours of daily cell phone EMR (0.9-1.8 GHz) exposure for six weeks, while the AE and EMR+AE groups completed a treadmill AE five days per week. The heart was collected 48 hours after the last AE session to assess OS, histopathological damage, and left ventricular structure. ***Results:*** Tissue sections revealed that the EMR group had more significant heart tissue damage (increased apoptotic cells and collagen deposition) than the C group, and adding AE mitigated the damage ($P<0.05$). In the EMR group, in comparison to the EMR+AE, C, and AE groups, the activity of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) significantly decreased, and the concentration of malondialdehyde (MDA) elevated notably ($P < 0.05$). Exercise training in both the AE and EMR+AE groups resulted in a notable rise in SOD, CAT, and GPX activity, alongside a notable reduction in MDA concentration in comparison to the EMR group ($P < 0.05$). ***Conclusions:*** AE is advantageous in mitigating the adverse impacts of EMR emitted from mobile phones on the heart.

INTRODUCTION

Modern society's increasing reliance on mobile devices has sparked apprehension regarding the potential health impacts of electromagnetic radiation (EMR) emitted by cell phones. While offering advantages in everyday activities, the individuals exposed to cell phone EMRs face an elevated risk of experiencing tissue damage in various organs [1, 2]. Furthermore, the International Agency for Research on Cancer (IARC) has classified radiofrequency (RF) radiation as "potentially carcinogenic to humans," highlighting the increasing acknowledgment of possible health hazards linked to exposure to the EMR from mobile phones [3]. Several studies showed that cell phone EMR can increase the production of reactive oxygen species (ROS), reduce the activity of antioxidant enzymes in different organs, and ultimately lead to oxidative damage and dysfunction in those organs [4-8]. Being one of the tissues susceptible to oxidative stress (OS), the heart is crucial for maintaining circulatory function and overall physiological balance. Recent research indicates that exposure to cell phone EMR elevates ROS production, subsequently initiating OS and causing tissue damage within the heart [2, 9, 10]. At low to moderate levels, ROS perform crucial functions in regulating normal physiological processes related to development, including cell cycle progression, differentiation, proliferation, migration, and cell death, immune system function, maintenance of redox balance, and the activation of various cellular signaling pathways. However, damage to lipids, membranes, proteins, nucleic acids, and organelles can result from elevated levels of ROS, which may initiate cell death mechanisms like apoptosis [11]. Consequently, the variations in ROS levels were associated with or implicated in the onset of multiple diseases, encompassing cancer, neurodegenerative, cardiovascular, digestive, and respiratory diseases [12].

The OS arises from a disparity between the generation of ROS and the body's antioxidant defense mechanisms. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) are examples of antioxidant enzyme systems that serve to shield the body from the excessive generation of ROS [13]. Free radicals are ROS that continually circulate via the body as the byproducts of various physiological reactions. As a protective measure against oxidative damage induced by free radicals, the human body has evolved an antioxidant defense

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

system that employs processes such as metal chelation, enzymatic activities, and free radical scavenging to promptly neutralize these reactive species upon their formation [14]. Elevated generation of free radicals and heightened OS can affect the morphology and biochemistry of the heart [9]. Assessing the apoptosis of cardiac myocytes and collagen deposition stands out as a method for evaluating heart health, given that the occurrence of apoptosis in these cells and collagen deposition is implicated in various disease states, such as myocardial infarction and heart failure [15, 16]. Scientists and experts consistently endorse regular exercise as a crucial element of a healthy lifestyle. Most people would agree that engaging in regular physical exercise may improve general health, avoid certain illnesses, and increase overall fitness [17]. Regular aerobic exercise (AE) was shown to improve antioxidant defense mechanisms and reduce ROS production, thus attenuating OS and subsequent cell damage [18]. For instance, certain studies have demonstrated the beneficial effect of exercise training in reducing OS in the heart [19, 20].

Despite extensive research into the influence of AE on heart function and structure, there is still a lack of understanding regarding its impact on OS and damage to heart cells caused by EMR emission. In this regard, we speculate that by reducing OS, which may support heart health, engaging in AE may lessen cardiomyocyte damage and the adverse effects of EMR. As far as we know, no previous study has demonstrated the effect of moderate AE training in mitigating the adverse effects of EMR emitted from a cell phone on the hearts of male Wistar rats. Hence, this present study investigates the influence of AE on OS and cardiac tissue modifications in male Wistar rats subjected to EMR emitted from a cell phone.

MATERIAL AND METHODS

Animals

Thirty-two male Wistar rats weighing 200-250g were purchased from the Pasteur Institute of Iran (Tehran) and randomly divided into Control (C), EMR, AE, and EMR+AE groups. The rats were kept in standard transparent polycarbonate cages (four rats in each cage) and were allowed to acclimatize to laboratory conditions (a 12 h light/dark cycle, 50 ± 5% humidity, 22 ± 2 °C temperature) for two weeks. The cages were 43 cm × 29 cm × 20 cm, and the rats had unrestricted food and tap water access. Approval for the research was granted by the Research Ethics Committee at the University of Tehran (IR.UT.SPORT.REC.1401.047), and all methodologies followed the protocols outlined in the National Research Council's Guide for the Care and Use of Laboratory Animals.

AE protocol

In preparation for the primary protocol, rats assigned to AE and EMR+AE groups underwent a five -session adaptation phase on a treadmill in which the speed and duration of the AE progressively increased from 5 to 15 m/min and 5 to 15 minutes per session, respectively. Afterward, the animals participated in a six-week AE program, maintaining a pace of 15 meters per minute for 30 minutes daily, with five weekly sessions [4, 21]. The duration of the AE protocol was increased by 10 min each week, resulting in an 80-minute AE session by the last week. Furthermore, each exercise session was divided into two equal parts, with a 5-minute active rest period at 5 m/min between them.

EMR exposure

The male Wistar rats of the present study were exposed to a cell phone emitting EMR within the frequency range of 0.9-1.8 GHz for three hours daily over six weeks. During EMR exposure, the rats were placed in smaller cages to restrict their movement. The cell phone (Huawei Honor 7, China), with a specific absorption rate of 0.52 watts per kilogram, was positioned at a 15 cm distance from the rat cages to minimize the potential thermal effect of the device. A phone call was initiated using two mobile phones throughout the EMR process [4]. Animals in the AE group were subjected to the presence of a cell phone without a battery for the same duration.

Tissue processing and homogenate preparation

On the second day following the final exercise session, all rats were anesthetized via injection of ketamine and xylazine at a dosage of 88/10 mg/kg. The heart tissue of the rats was surgically extracted under sterile conditions and promptly rinsed with saline solution (0.9%). Subsequently, the tissue was frozen in liquid nitrogen and preserved at a deep freeze temperature of -80°C for later biochemical analysis. Furthermore, a portion of the heart tissue was fixed in 10% formalin for 48 hours, followed by a dehydration process with alcohol series, clearing with xylene (Sigma-1330-20-7), and infiltrating with paraffin wax to support the tissue for thin sectioning. Then, tissues were embedded into a paraffin wax block using a tissue-embedding center (LEICA EG 1160, WETZLAR, Germany), and sectioning (3-5 sections per specimen) with a fully automatic Leica 2155 rotary microtome (Germany) into 5 mm slices for subsequent Histopathological evaluation.

To assess antioxidant activities, about 100 mg of heart samples underwent homogenization in 1 mL phosphate-buffered saline (PBS [Sigma-p4417]). Next, the samples were subjected to centrifugation (Hettich, Germany) at a speed of 4000 revolutions per minute for 20 minutes at 4°C. The resulting liquid above the sediment, known as the supernatant, was

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

then preserved at a temperature of -20°C. This procedure was conducted to facilitate subsequent analysis of the levels of malondialdehyde (MDA), and the activity levels of the enzymes SOD, CAT, and GPX [22].

Determination of MDA concentrations

MDA levels in the samples were assessed using a ZellBio GmbH MDA assay kit (ZB-MDA-96A, Germany). The tubes were prepared by adding 0.1 mL of absolute ethanol to the 1.5 mL EP tubes for the blank tube, while for the standard tube, 0.1 mL of 10 nmol/mL standard solution was added. In the sample tube, 0.1 mL of the sample was added. Subsequently, 1 mL of the working solution was added to each tube, followed by thorough mixing with a vortex mixer (Qiagene, Iran). The tubes were tightly sealed with preservative film, with a small hole made in each film. Afterward, the tubes were incubated in a 100°C water bath for 40 minutes. Once the incubation was completed, the tubes were cooled to room temperature using running water and then centrifuged at 1078 g for 10 minutes. Following centrifugation, 250 µL of the supernatant from each tube was carefully transferred to a microplate using a micropipette. Finally, optical density (OD) values of each well were measured at 532 nm using a microplate reader (ELISA, BioTek, U.S.A), and the results were reported as µM/ml [23].

Determination of CAT activity

The CAT activity of the samples was assessed utilizing the ZellBio GmbH CAT Assay Kit (ZB-CAT-96A, Germany). In the plate, duplicate wells were filled with 25 µL of either samples or standards. The Zero standard was prepared by pipetting 25 µL of Assay Buffer. Subsequently, 25 µL of the Hydrogen Peroxide Reagent was added to each well using a multichannel/repeater pipette. The plate was then incubated at room temperature for 30 minutes. Following this incubation period, 25 µL of the Substrate was added to each well using a multichannel/repeater pipette, followed by the addition of 25 µL of the HRP Reagent to each well using the same pipetting method. The plate underwent another incubation at room temperature for 15 minutes. Finally, the OD at 560 nm was measured using a microplate reader, and the findings were expressed in U/mL [24].

Determination of GPX activity

A ZellBio GmbH GPX assay kit (ZB-GPX-96A, Germany) was used to measure the activity of GPX in the samples. During the enzymatic reaction phase, 20 µL of 1 mmol/L glutathione standard was placed in the non-enzyme tube, while the enzyme tube received 20 µL of 1 mmol/L glutathione standard and 20 µL of sample, followed by thorough mixing. After preheating the tubes and stock application solution at 37°C for 5 minutes, 10 µL was added to each tube and incubated at 37°C for 5 minutes. Then, the enzyme tube received just 200 µL of acid reagent, whereas the non-enzyme tube received 200 µL of acid reagent and 20 µL of the sample. Following a 10-minute centrifugation at 3100 g, 100 µL of the supernatant was utilized for the chromogenic reaction. In this phase, 100 µL of supernatant from non-enzyme and enzyme tubes, along with glutathione standard solution, was added to wells. Phosphate (100 µL) and DTNB solution (50 µL) were added to each well, followed by oscillation for 10 seconds and a 5-minute standing period before measuring OD values at 412 nm using a microplate reader, and the findings were expressed in U/mL [24].

Determination of SOD activity

SOD activity in the samples was assessed with the ZellBio GmbH SOD assay kit (ZB-SOD-96A, Germany). In duplicate wells of the plate, 10 µL of samples or standards were pipetted. Subsequently, 10 µL of Assay Buffer was pipetted into duplicate wells to serve as the Zero standard. Following this, 50 µL of the substrate working solution was added to each well using a multichannel pipette. Next, 25 µL of the xanthine oxidase solution was added to each well using a multichannel pipette. The plate was then incubated at room temperature for 20 minutes. Finally, the OD at 450 nm was read, and the results were reported as U/mL [24].

Histopathological evaluation

Heart tissue slices were stained with standard staining like Sirius Red to observe collagen deposition and Haematoxylin and Eosin (H&E) to observe overall cellular structure. For Sirius Red staining, tissue sections were deparaffinized and hydrated [25]. Nuclei were stained with the hematoxylin for 8 minutes, followed by a 10-minute rinse. Next, the sections underwent staining using Picro-Sirius red solution, comprising 0.5 grams of Sirius Red F3B (C.I. 35782) dissolved in 500 ml of saturated aqueous picric acid solution, for a duration of one hour. Subsequent to staining, the sections were washed with acidified water (5 mL of glacial acetic acid applied to 1 liter of water). Three changes of 100% ethanol were utilized to dehydrate the specimens, after which the transparencies were dried with xylene and adhered to a resinous medium. The H&E staining protocol, similar to the Fischer et al. protocol [26], began with dewaxing tissue sections in a Vinteb oven at 90°C for 20 minutes to remove paraffin. The samples were then subjected to paraffin removal in two different xylene solutions for 15 minutes. Tissue hydration started with 100% ethanol for 5 minutes, gradually transitioning to distilled water. The sections were then submerged in hematoxylin (Sigma-H9627) for seven seconds and then rinsed for one minute in distilled water. Following a two-second treatment with saturated

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

lithium carbonate (Sigma -1.05680), the specimens underwent rinsing with water and alcohol before being stained for three minutes with eosin (Sigma - HT110116). Dehydration was achieved by sequential placement in absolute alcohol. Then, the tissue sections were placed in two different xylene solutions for 15 minutes each to achieve transparency. Then, mounting was done with Entellan glue (Sigma- 1.07961), facilitating observation under a light microscope.

Finally, histological parameters were evaluated by two independent judges in a blind condition using a photo-light microscope (DM4000B Image Analyze System, Leica, Microsystems, Heidelberg GmbH, Heidelberg, Germany) equipped with a Leica DFC280 plus camera [27].

Statistical analysis

Data analysis was conducted using SPSS software (version 25.0, Chicago, IL, USA). Before conducting one-way ANOVA, normal data distribution was confirmed, variance homogeneity was assessed, and it was ensured that there were no significant outliers. Subsequently, mean variables across different groups were compared using Tukey's post hoc test. Significance was established for p-values less than 0.05, and Means ± SD was used to present the results.

RESULTS

Antioxidant

Figure 1 displays the changes in MDA concentration and the activity of antioxidant enzymes CAT, GPX, and SOD. Compared to the C group, a notable increase was observed in the levels of MDA in the EMR group (p<0.05). AE significantly reduced the levels of this indicator for lipid peroxidation compared to the control rats (P<0.05). The group subjected to both EMR and aerobic exercise (EMR+AE) showed a significant decrease in MDA levels when compared to the group exposed only to EMR (P<0.05). Nevertheless, the MDA levels in the EMR+AE group remained notably elevated compared to the C group (P<0.05). The activity of SOD, CAT, and GPX, which act as an indicator of antioxidant activity, significantly reduced in the EMR-exposed rats compared to the control rats (P < 0.05) (table 1).

Conversely, moderate AE of the present study increased the activity of antioxidant enzymes (SOD, CAT, GPX) in comparison to the C group (P <005). Performing AE in the rats exposed to the EMR (EMR+AE group) led to a notable rise in the activity of all three antioxidant enzymes in comparison to the EMR group (P<0.05). Nevertheless, these levels remained notably lower than those in the C group (table 1).

Histopathological considerations

The heart sections stained with H&E and Sirius Red revealed that in comparison to the C group, the proportion of apoptotic cells and myocardial collagen was considerably more significant in the EMR group (p<0.05) (figure 2). AE substantially reduced the percentage of these parameters compared to the C group (P<0.05). There was a notable reduction in the percentage of apoptotic cells and myocardial collagen of the EMR+AE group in comparison to the EMR group (table 1). Nevertheless, they remained significantly higher than the percentages observed in the control rats (P<0.05) (figure 3).

Left ventricular structure

The results showed no differences in left ventricular thickness (LVT) and left ventricle internal caliber (LVIC) among the groups (figure 3, table 1).

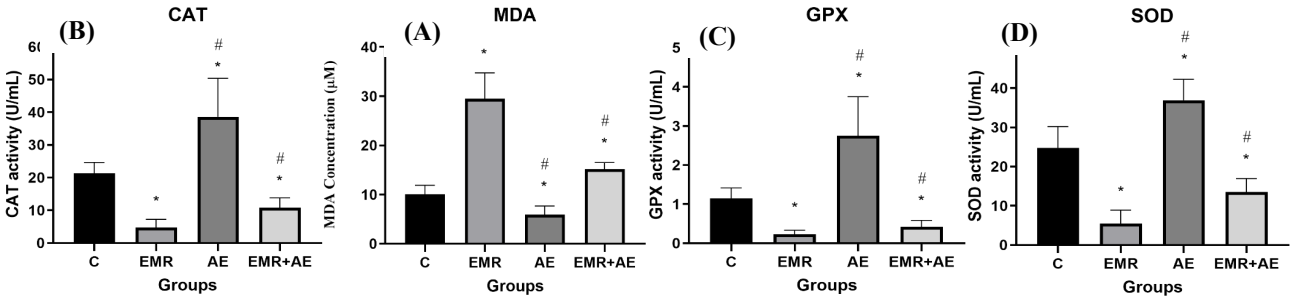


Figure 1. Levels of MDA, as well as the activities of CAT, GPX, and SOD enzymes in the heart tissue of different groups. **(A)** cardiac tissue MDA levels (µM/ml). **(B)** cardiac tissue CAT activity (U/ml). **(C)** cardiac tissue GPX activity (U/ml). **(D)** cardiac tissue SOD activity (U/ml). The values represent means ± SD; n = 8. Four groups: control (C), electromagnetic radiation (EMR), aerobic exercise (AE), electromagnetic radiation + aerobic exercise (EMR+AE). MDA, malondialdehyde; GPX, glutathione peroxidase; CAT, catalase; SOD, superoxide dismutase. * indicates significant differences from the C group (P<0.05); # indicates significant differences from the EMR group (P<0.05).

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

Table 1. Effects of EMR, AE, and EMR+AE on the oxidative and histological parameters of the heart.

Parameter	C (n = 8)	EMR (n = 8)	EMR + AE (n = 8)	AE (n = 8)
Body weight (g)	205.9 ± 15.3	197.9 ± 10.3	200.7 ± 8.7	197.5 ± 6.6
MDA (µM)	10.06 ± 1.84	29.46 ± 5.28*	15.21 ± 1.3*#	5.89 ± 1.80*#
CAT (U/mL)	21.26 ± 3.39	4.76 ± 2.47*	10.81 ± 2.98*#	38.56 ± 11.84*#
GPX (U/mL)	1.14 ± 0.27	0.23 ± 0.11*	0.42 ± 0.16*#	2.75 ± 0.10*#
SOD (U/mL)	24.75 ± 5.45	5.48 ± 3.39*	13.52 ± 3.40*#	36.87 ± 5.36*#
apoptotic cells (%)	11.57 ± 5.61	39.55 ± 4.78*	22.22 ± 4.99*#	2.13 ± 0.75*#
collagen deposition (% in mm)	11.39 ± 4.62	46.16 ± 7.38*	26.59 ± 6.30*#	2.48 ± 1.02*#
left ventricle thickness (mm)	3.32 ± 0.57	3.43 ± 0.98	3.44 ± 1.22	3.33 ± 0.80
left ventricle internal caliber (mm)	2.22 ± 0.34	2.28 ± 0.87	2.40 ± 0.47	2.39 ± 0.68

Data are expressed as means ± SD. Four groups: control (C), electromagnetic radiation (EMR), aerobic exercise (AE), electromagnetic radiation + aerobic exercise (EMR+AE). MDA, malondialdehyde; SOD, superoxide dismutase; GPX, glutathione peroxidase; CAT, catalase. * indicates significant differences from the C group (P<0.05); # indicates significant differences from the EMR group (P<0.05).

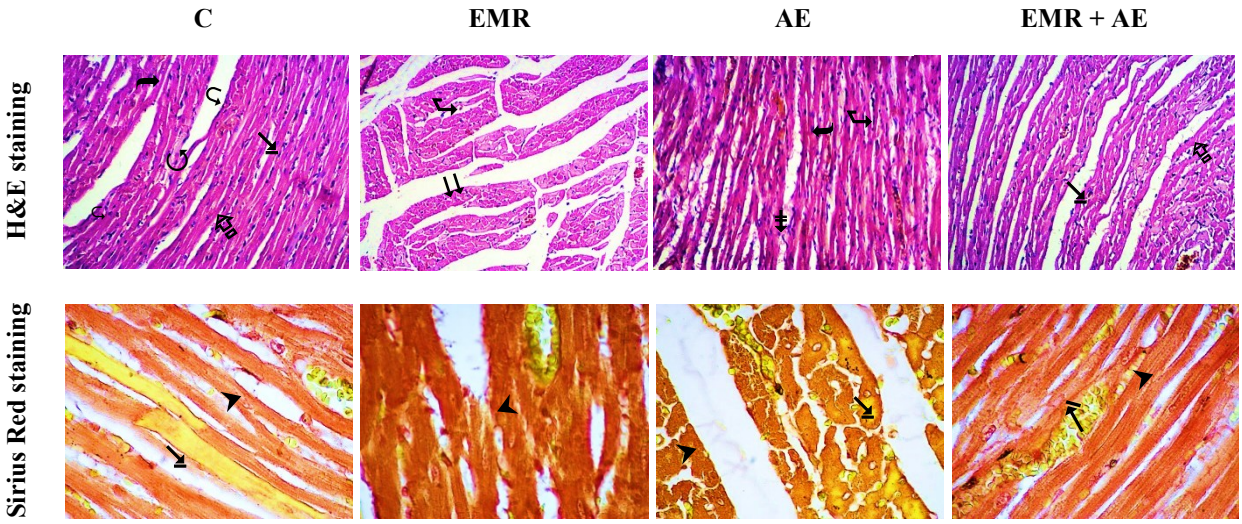


Figure 2. Sections of heart tissues stained with Hematoxylin-Eosin (H&E) and Sirius Red from different groups. Heart muscle cells (↘), intercalated disc (⇳), nucleus (↺), cross striations (↪), collateral branches (↟), disorganized myofibrils (➦), connective tissue among cardiomyocytes (↻), and apoptotic cardiomyocytes (⇊) were seen in all groups (H&E at a magnification of 400x). Cardiomyocytes (↘) and collagen (➤) were observed in all four groups (Sirius Red at a magnification of 400x). Four groups: control **(C)**, electromagnetic radiation **(EMR)**, aerobic exercise **(AE)**, electromagnetic radiation + aerobic exercise **(EMR+AE)**.

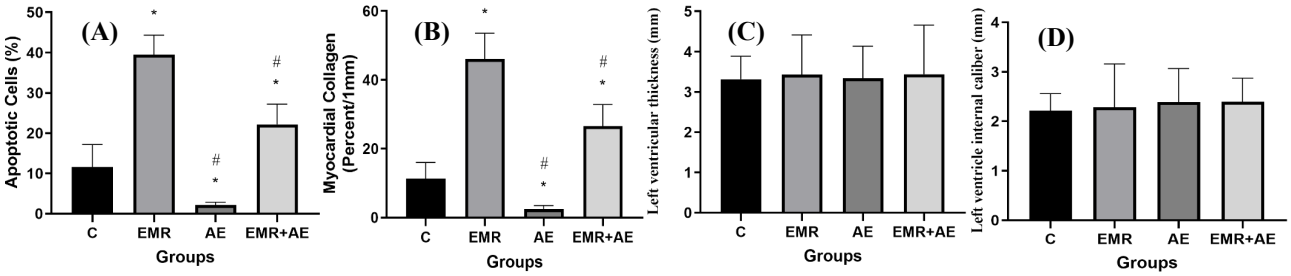


Figure 3. The percentage of apoptotic cells, myocardial collagen deposition, left ventricular thickness and left ventricle internal caliber of the heart. **(A)** apoptotic cells (percent), **(B)** myocardial collagen deposition (percent/mm), **(C)** left ventricular thickness (mm), and **(D)** left ventricle internal caliber (mm). The values represent means ± SD; n = 8. Four groups: control **(C)**, electromagnetic radiation (EMR), aerobic exercise (AE), electromagnetic radiation + aerobic exercise (EMR+AE). * indicates significant differences from the C group (P<0.05); # indicates significant differences from the EMR group (P<0.05).

DISCUSSION

The aim of the present study was to examine the impact of 6 weeks of AE and exposure to 0.9-1.8 GHz EMR on heart OS parameters, histological considerations, and left ventricular structure of the adult male Wistar rats. According to the results of the present study, exposure to the cell phone EMR for six weeks resulted in increased lipid peroxidation and diminished antioxidant enzyme activity, thereby exacerbating OS in the heart. Multiple studies have documented the impact of EMR in elevating OS in various body organs and blood [4, 7, 28]. For instance, Ozguner et al. examined the impact of cell phone EMR at a frequency of 0.9 GHz for 30 minutes daily over a period of 10 days on OS levels in the heart. They found that EMR could increase tissue MDA and nitric oxide (NO, a marker of OS) levels and decrease SOD,

[Downloaded from ijrr.com on 2026-11-05]
[DOI: 10.61186/ijrr.23.1.45]

CAT, and GPX activities [29]. Kerimoğlu *et al*. observed an elevation in MDA concentration and a decline in total antioxidant capacity in heart tissue following exposure to cell phone EMR at a frequency of 0.9 GHz (1 hour/day, for 39 days) [9]. Furthermore, Toossi *et al*. investigated the impact of mobile phone EMR operating at 0.9-1.8 GHz for 2 hours per day over a period of 20 days on some vital organs, including the liver, heart, kidney, cerebellum, and hippocampal, in pregnant mice. In all the examined tissues of the dams and the young mice, they discovered a rise in MDA concentration and a fall in SOD, CAT activity, and total thiol groups (TTG), a measure of antioxidant activity [10]. Esmekaya et al. showed that 0.9 GHz pulse-modulated radiofrequency (RF) radiation induces oxidative damage in the testis, lung, liver, and heart tissues of male Wistar albino rats. This damage is facilitated by lipid peroxidation, suppression of antioxidant enzymes, and elevated levels of NO [2]. Furthermore, Kuzay *et al.* studied the possible effects of eight weeks of RF Radiation exposure at a frequency of 2100 MHz for 60 minutes per day, conducted over 5 days per week, on the hypertensive and non-hypertensive rat's blood, aorta, and heart tissue. They revealed that cardiac and plasma MDA, total NO, and total sulphydryl groups (RSH)/glutathione (GSH) levels were significantly elevated after RF exposure [30]. Conversely, Devrim et al. reported a decrease in lipid peroxidation and xanthine oxidase (XO, a marker of OS) and adenosine deaminase (ADA) enzyme activities of the heart tissue following exposure to the EMR at a frequency of 0.9 GHz (4 times a day for 10 min, during 4 weeks) [1], which might be in terms of the intermittent nature of their EMR protocol.

Derived from the outcomes of the present study and present research background, cell phone EMR may lead to OS in the heart tissue by increasing lipid peroxidation and weakening the antioxidant defense mechanisms. The exact mechanism by which cell phone EMR induces OS is still uncertain, as electromagnetic fields induce diverse biological responses in heart cells and influence metabolic processes throughout the body by various mechanisms. Nonetheless, it is known that the plasma membrane is an EMR target. According to one theory, EMR causes OS by acting on the plasma membrane via non-thermal means, which might boost ROS production and plasma membrane NADH oxidase activity. Increased concentrations of free radicals can impact diverse cellular and physiological functions, including but not limited to calcium release from intracellular storage sites, gene expression, cell growth, and apoptosis [31, 32].

On the other hand, our findings demonstrated that AE had the potential to diminish OS in the heart by reducing lipid peroxidation and increasing antioxidant enzymes. Many studies were conducted to validate the impact of exercise in enhancing both antioxidant capacity and functional markers in the heart [19, 33, 34]. For instance, in a study by Kanter *et al.,* it was found that the concentrations of MDA and the activity of antioxidant enzymes (CAT, SOD, and GPX) in the heart tissue of streptozotocin-induced diabetic rats decreased and increased, respectively, following 4 weeks of low-intensity AE on a treadmill to a speed of 10 m/min [19]. Ranjbar *et al*. also discovered that 10 weeks of AE at a speed of 17 m/min for 50 min/day (5 days/week) led to a reduction in OS and an enhancement of the antioxidant defense system in the heart tissue of Post-ischemic Failing Rats [20].

A baseline of ROS is essential for cellular function, yet excessive ROS can damage cellular components like DNA, lipids, and proteins, ultimately causing necrosis and apoptosis [35]. In this regard, several studies documented the impact of EMR on heart morphology [9, 36, 37]. For example, a study in hypertensive rats demonstrated that 8 weeks of exposure to 2100 MHz RF radiation for 60 min/day and 5 days/week significantly increased left ventricular fibrosis and had aortic degenerative effects [30]. Kerimoğlu *et al*. also reported that 1 hour/day exposure to cell phone EMR for 39 days significantly increased the percentage of apoptotic myocardial cells due to increased OS [9]. Furthermore, our research revealed that exposure to EMR substantially increased the percentage of apoptotic cells and deposition of myocardial collagen, both of which are indicators of cardiac injury. Although exact mechanisms are not fully understood, it is hypothesized that EMR-induced myocardial cell damage may be associated with increased Cytochrome C, the activation of cleaved caspase-3 and cleaved caspase-9, increased expression of Bax, and potentially decreased levels of Bcl-2 protein. Additionally, EMR exposure might notably elevate phosphorylated ERK and JNK levels through OS, which could induce pro-apoptotic impacts on myocardiocytes [37].

The present study showed that AE reduced the pathologic changes in the heart tissue and ameliorated cardiac damage induced by EMR. The results of the other studies are consistent with our findings, indicating that AE decreases OS, consequently resulting in diminished damage to heart cells [19, 38, 39]. For instance, Wang *et al.* showed a decrease in myocardial fibrosis in type 2 diabetic rats after 8 weeks of moderate-intensity treadmill AE (15.2 m/min, 60 min/day, 5 sessions/weeks) via the inhibitions of OS and TGF-β1/Smad pathway [40]. After four weeks of treadmill interval exercise, five days per week, Chen et al. observed an enhancement in cardiac function in rats that had suffered myocardial infarction (MI). This improvement was attributed to the reduction in apoptosis, OS, and inflammation, which may have been induced by the activation of the AMPK/SIRT1/PGC-1alpha signaling pathway [41].

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

CONCLUSION

The findings of this study demonstrated that AE has the potential to reduce OS and mitigate myocardial damage in rats subjected to the cell phone EMR while also enhancing the activity of antioxidant enzymes in their hearts. Considering inevitable exposure to EMR waves in today's environment, engaging in regular AE can serve as a preventive measure against the harmful impacts of these waves on vital organs like the heart.

ACKNOWLEDGMENT

There is no individual to mention in this section specifically.

***Funding*:** This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

***Conflict of Interests*:** The authors declare that there are no conflicts of interest.

***Ethical consideration*:** This research was approved by the Research Ethics Committee of the University of Tehran, and all protocols were conducted according to the National Research Council's Guide for the Care and Use of Laboratory Animals (Ethical number: IR.UT.SPORT.REC.1401.047).

***Author contribution*:** H.A.A, A.A.G, and M.R.K conceived and designed the analysis. H.A.A and A.A.G conducted the experiment and collected the data. H.A.A and A.A.R performed the analysis. H.A.A wrote the original paper, and all three other authors reviewed the results and approved the final version of the manuscript.

REFERENCES

1. Devrim E, Ergüder IB, Kılıçoğlu B, *et al.* (2008) Effects of electromagnetic radiation use on oxidant/antioxidant status and DNA turn-over enzyme activities in erythrocytes and heart, kidney, liver, and ovary tissues from rats: Possible protective role of vitamin c. *Toxicol Mech Methods,* ***18****(9): 679-83.*
2. Esmekaya MA, Ozer C, Seyhan N (2011) 900 mhz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues. *Gen Physiol Biophys,* ***30****(1): 84-9.*
3. (2013) Non-ionizing radiation, part 2: Radiofrequency electromagnetic fields. *IARC Monogr Eval Carcinog Risks Hum,* ***102****(Pt 2): 1-460.*
4. Rasouli Mojez M, Ali Gaeini A, Choobineh S, Sheykhlouvand M (2021) Hippocampal oxidative stress induced by radiofrequency electromagnetic radiation and the neuroprotective effects of aerobic exercise in rats: A randomized control trial. *J Phys Act Health,* ***18****(12): 1532-38.*
5. Ismaiil LA, Joumaa WH, Moustafa ME (2019) The impact of exposure of diabetic rats to 900 mhz electromagnetic radiation emitted from mobile phone antenna on hepatic oxidative stress. *Electromagn Biol Med,* ***38****(4): 287-96.*
6. Oktem F, Ozguner F, Mollaoglu H, *et al.* (2005) Oxidative damage in the kidney induced by 900-mhz-emitted mobile phone: Protection by melatonin. *Arch Med Res,* ***36****(4): 350-5.*
7. Amiri H, Shabkhiz F, Pournemati P, *et al.* (2023) Swimming exercise reduces oxidative stress and liver damage indices of male rats exposed to electromagnetic radiation. *Life Sci,* ***317****: 121461.*
8. Tawfik MS, Saif-Elnasr M, Elkady AA, *et al.* (2018) Protective role of ferulic acid against the damaging effect induced by electromagnetic waves on rat liver and intestine tissues. *International Journal of Radiation Research,* ***16****(4): 421-30.*
9. Kerimoğlu G, Mercantepe T, Erol HS, *et al.* (2016) Effects of long-term exposure to 900 megahertz electromagnetic field on heart morphology and biochemistry of male adolescent rats. *Biotech Histochem,* ***91****(7): 445-54.*
10. Bahreyni Toossi MH, Sadeghnia HR, Mohammad Mahdizadeh Feyzabadi M, *et al.* (2018) Exposure to mobile phone (900-1800 mhz) during pregnancy: Tissue oxidative stress after childbirth. *J Matern Fetal Neonatal Med,* ***31****(10): 1298-303.*
11. Redza-Dutordoir M, Averill-Bates DA (2016) Activation of apoptosis signalling pathways by reactive oxygen species. *Biochim Biophys Acta,* ***1863****(12): 2977-92.*
12. Liu Z, Ren Z, Zhang J, *et al.* (2018) Role of ros and nutritional antioxidants in human diseases. *Front Physiol,* ***9****: 477.*
13. Rajlic S, Treede H, Münzel T, *et al.* (2023) Early detection is the best prevention-characterization of oxidative stress in diabetes mellitus and its consequences on the cardiovascular system. *Cells,* ***12****(4).*
14. Chaudhary P, Janmeda P, Docea AO, *et al.* (2023) Oxidative stress, free radicals and antioxidants: Potential crosstalk in the pathophysiology of human diseases. *Front Chem,* ***11****: 1158198.*
15. Cowling RT, Kupsky D, Kahn AM, *et al.* (2019) Mechanisms of cardiac collagen deposition in experimental models and human disease. *Transl Res,* ***209****: 138-55.*
16. Bennett MR (2002) Apoptosis in the cardiovascular system. *Heart,* ***87****(5): 480-7.*
17. Bull FC, Al-Ansari SS, Biddle S, *et al.* (2020) World health organization 2020 guidelines on physical activity and sedentary behaviour. *Br J Sports Med,* ***54****(24): 1451-62.*
18. He F, Li J, Liu Z, *et al.* (2016) Redox mechanism of reactive oxygen species in exercise. *Front Physiol,* ***7****: 486.*
19. Kanter M, Aksu F, Takir M, *et al.* (2017) Effects of low intensity exercise against apoptosis and oxidative stress in streptozotocin-induced diabetic rat heart. *Exp Clin Endocrinol Diabetes,* ***125****(9): 583-91.*
20. Ranjbar K, Nazem F, Nazari A (2016) Effect of exercise training and l-arginine on oxidative stress and left ventricular function in the post-ischemic failing rat heart. *Cardiovasc Toxicol,* ***16****(2): 122-9.*
21. Akbari H, Gaeini A (2021) Moderate exercise training as an effective strategy to reduce the harmful effects of cell phone radiation on wistar rat's semen quality. *International Journal of Radiation Research,* ***19****(2): 317-23.*
22. Naderi R, Mohaddes G, Mohammadi M, *et al.* (2015) Voluntary exercise protects heart from oxidative stress in diabetic rats. *Adv Pharm Bull,* ***5****(2): 231-6.*
23. Javanmard F, Bidmeshkipour A, Ghasemian Yadegari J (2022) Ceratonia siliqua l improved cryodamage of asthenozoospermic specimens: An experimental study. *Int J Reprod Biomed,* ***20****(12): 1029-38.*
24. Karami E, Goodarzi Z, Ghanbari A, *et al.* (2022) Dataset on biochemical markers and histological alterations in rat kidney intoxicated with cadmium chloride and treated with antioxidant. *Data Brief,* ***43****: 108394.*
25. Rittié L (2017) Method for picrosirius red-polarization detection of collagen fibers in tissue sections. *Methods Mol Biol,* ***1627****: 395-407.*
26. Fischer AH, Jacobson KA, Rose J, Zeller R (2008) Hematoxylin and eosin staining of tissue and cell sections. *CSH Protoc,* ***2008****: pdb.prot4986.*
27. Pourmousavi L, Hashemkandi Asadi R, Zehsaz F, Jadidi RP (2023) Effect of crocin and treadmill exercise on oxidative stress and heart damage in diabetic rats. *PLoS One,* ***18****(12): e0281692.*
28. Moghadasi N, Alimohammadi I, Safari Variani A, Ashtarinezhad A (2021) The effect of mobile radiation on the oxidative stress biomarkers in pregnant mice. *J Family Reprod Health,* ***15****(3): 172-78.*
29. Ozguner F, Altinbas A, Ozaydin M, *et al.* (2005) Mobile phone-induced myocardial oxidative stress: Protection by a novel antioxidant agent caffeic acid phenethyl ester. *Toxicol Ind Health,* ***21****(9): 223-30.*
30. Kuzay D, Ozer C, Goktas T, *et al.* (2018) Effects of 2100 mhz radio frequency radiation on the viscosity of blood and oxidative stress parameters in hypertensive and normal rats. *International Journal of Radiation Research,* ***16****(4): 431-42.*
31. Kıvrak EG, Yurt KK, Kaplan AA, *et al.* (2017) Effects of electromagnetic fields exposure on the antioxidant defense system. *J Microsc Ultrastruct,* ***5****(4): 167-76.*
32. Phaniendra A, Jestadi DB, Periyasamy L (2015) Free radicals: Properties, sources, targets, and their implication in various diseases. *Indian J Clin Biochem,* ***30****(1): 11-26.*
33. Chodari L, Smailnejad S, Fallahi M, *et al.* (2019) Oxidative stress is markedly reduced by combined voluntary exercise and testos-

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]

terone in the heart of diabetic rats. *Acta Endocrinol (Buchar),* ***15*** *(2): 173-81.*
34. Zarei S, Taghian F, Sharifi G, Abedi H (2022) Novel prevention insights into depletion of oxidative stress status through regular exercise and grape seed effective substance in heart ischemia rat model. *Food Sci Nutr,* ***10****(3): 833-45.*
35. Senoner T, Dichtl W (2019) Oxidative stress in cardiovascular diseases: Still a therapeutic target? *Nutrients,* ***11****(9).*
36. Martinelli I, Cinato M, Keita S*, et al.* (2022) Cardiac cell exposure to electromagnetic fields: Focus on oxdative stress and apoptosis. *Biomedicines,* ***10****(5).*
37. Zhu W, Cui Y, Feng X*, et al.* (2016) The apoptotic effect and the plausible mechanism of microwave radiation on rat myocardial cells. *Can J Physiol Pharmacol,* ***94****(8): 849-57.*
38. Kwak HB, Kim JH, Joshi K*, et al.* (2011) Exercise training reduces fibrosis and matrix metalloproteinase dysregulation in the aging rat heart. *Faseb J,* ***25****(3): 1106-17.*
39. Wang T, Zhang L, Cai M, Tian Z (2023) Effects of different exercise modalities on inhibiting left ventricular pathological remodeling in patients with heart failure with reduced ejection fraction: A systematic review and network meta-analysis. *Life Sci,* ***319****: 121511.*
40. Wang SQ, Li D, Yuan Y (2019) Long-term moderate intensity exercise alleviates myocardial fibrosis in type 2 diabetic rats via inhibitions of oxidative stress and tgf-β1/smad pathway. *J Physiol Sci,* ***69*** *(6): 861-73.*
41. Chen Y, Huang Q, Feng Y (2023) Exercise improves cardiac function in the aged rats with myocardial infarction. *Physiol Res,* ***72****(1): 27-35.*

[Downloaded from ijrr.com on 2026-11-05]

[DOI: 10.61186/ijrr.23.1.45]